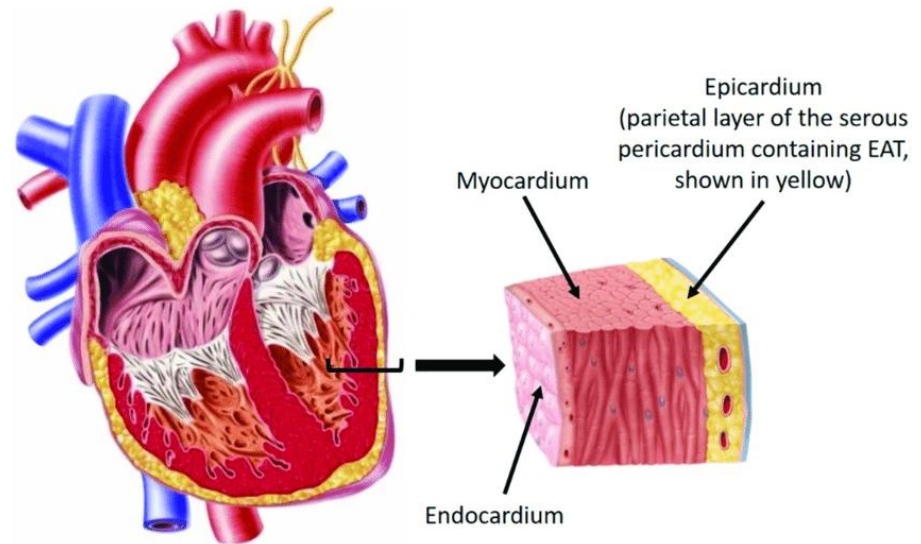


# PATHOPHYSIOLOGY OF CARDIOVASCULAR SYSTEM PART 2

# CONTENT

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- Endocardial diseases
- Myocardial diseases
- Pericardial diseases
- Arterial diseases
- Vein diseases
- Disorders of lymphatic vessels



# Endocardial diseases

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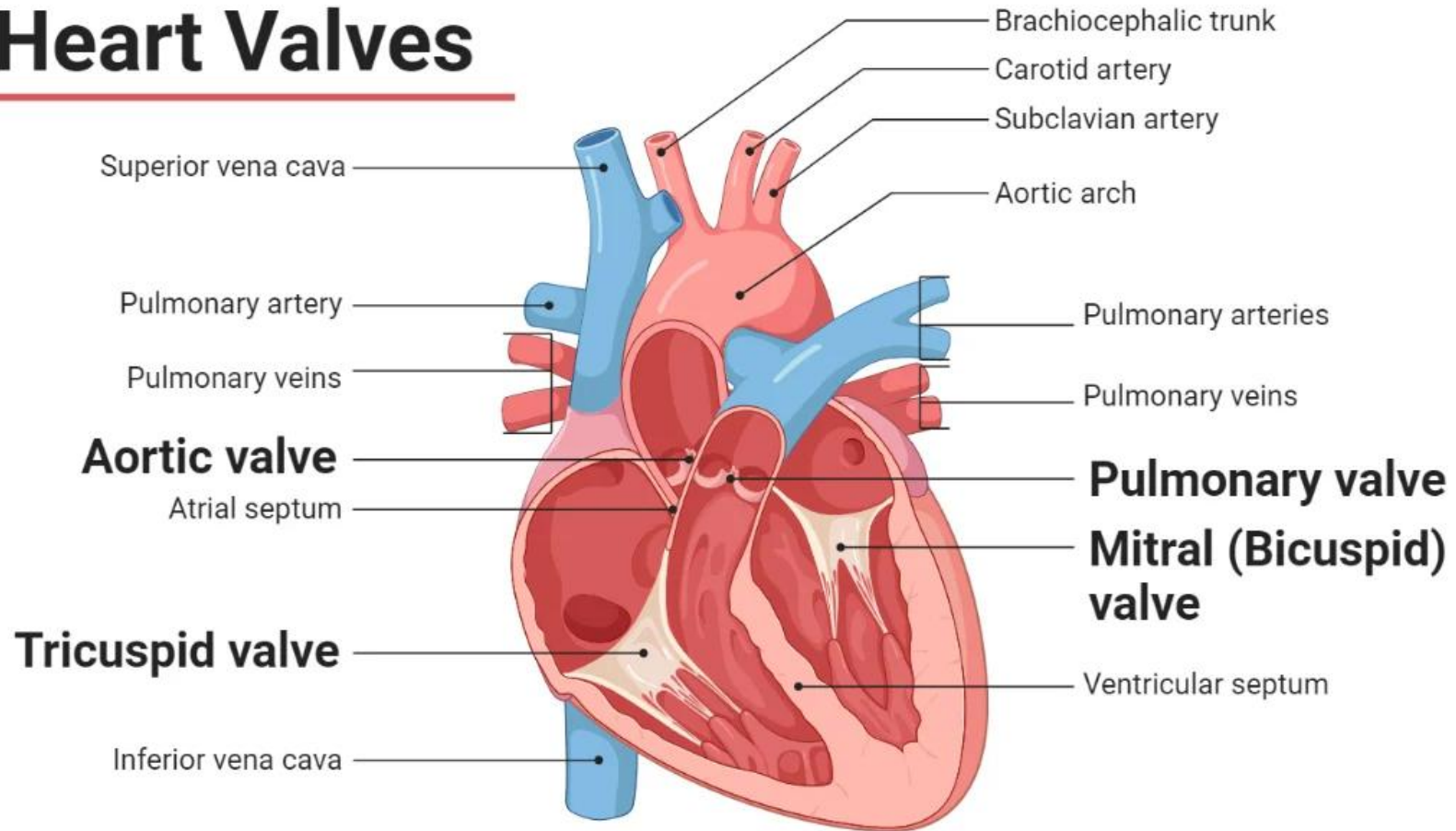
1. Valvular defects (valve disorders)
  - Stenosis (smaller valve area compared to normal)
  - Insufficiency (insufficient closure of the valve with regurgitation of blood)
2. Rheumatic fever
3. Infective endocarditis

# Valvular defects (valve disorders)

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## Heart Valves

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# Valvular defects (valve disorders)

- Destruction of valves that prevent normal closure of valve (**insufficiency**)
- Reduction in the valve area with mechanical obstruction to blood flow (**stenosis**)
- As a result, hemodynamic changes occurs (CVS - closed system of connected vessels)

# Hemodynamic changes in CVD

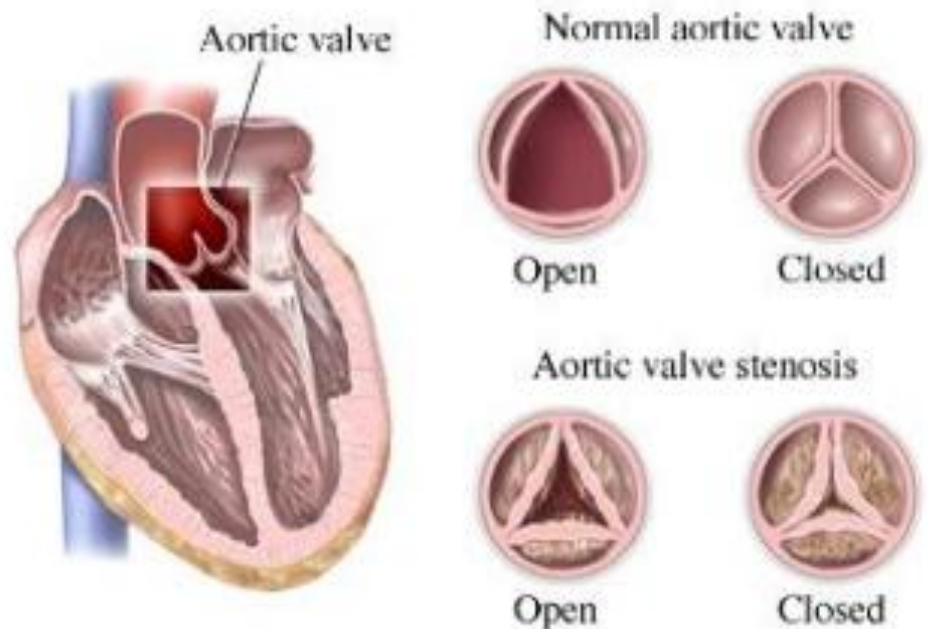
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- In order to understand hemodynamic processes in the blood vessels and the heart, you need to understand Pascal's law.
- **Pascal's law** (the **principle of transmission of pressure in fluid**) - states that a **pressure change** at any point in a closed filled system of vessels **is transmitted throughout the fluid** in such a way **that the same change occurs everywhere**.

# Aortic stenosis

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- Obstruction in the passage of blood from the left ventricle to the aorta during systole, as a consequence of reduction in the aortic orifice
- Depending on the location:
  - On the valve itself
  - Subvalvular
  - Supravalvular

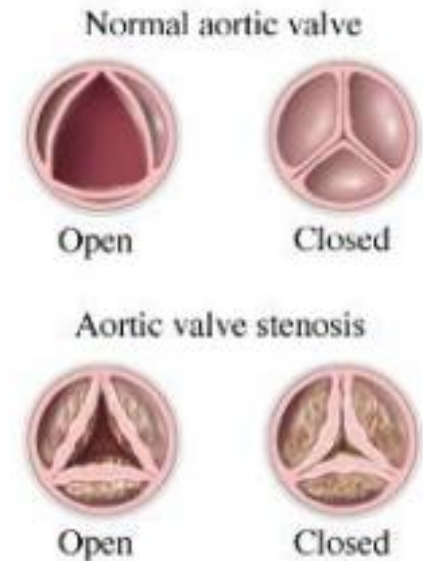


# Aortic stenosis

---

## ETIOLOGY:

1. Congenital
  2. Bicuspidal
  3. Rheumatic  
(in combination with defect in other orifice)
  4. Senile
  5. Other: after the infective endocarditis
- The basic pathophysiological disorder is **decrease in the area of the aortic orifice** (2-3 cm<sup>2</sup>), which leads to reduced ejection volume (during systole) and increased pressure in the left ventricle

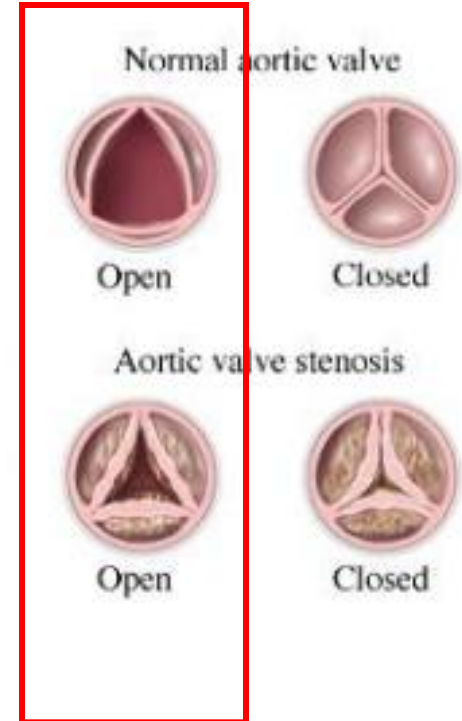




# Aortic stenosis

## ETIOLOGY:

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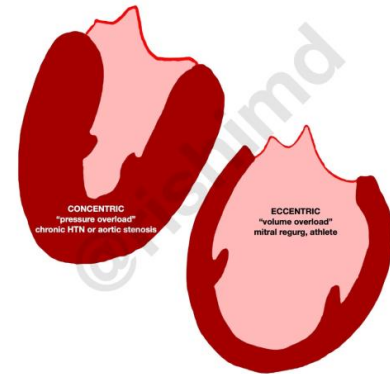


# Aortic stenosis

- PATHOPHYSIOLOGICAL CONSEQUENCE of AS:

1. increased ejection resistance for blood
2. increased systolic pressure in the LV
3. concentric hypertrophy of the LV
4. **LV diastolic dysfunction**
5. increase of end-diastolic pressure in LV with retrograde increase in systolic pressure in LA (by **Pascal's law**)
6. pulmonary vein congestion and pulmonary edema
7. dilatation and reduced contractility of the LV (**systolic dysfunction**)
8. decrease in ejective fraction during systola (MV in rest is normal but MV during effort is reduced)

## LV HYPERTROPHY



# Aortic stenosis

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- Clinical manifestations:
  1. Dyspnea
  2. Consciousness disorder (Syncopa)
  3. Angina pectoris
  4. First insufficiency of the left heart and then the right heart (later)
  5. Changes in the strength of the pulse wave (**pulsus parvus et tardus - delayed weaker than normal**)
  6. **Convergent blood type pressure - the difference between Sys and Dia BP is reduced**

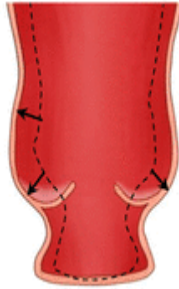

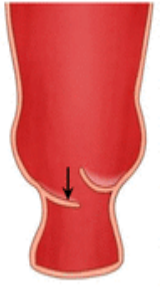
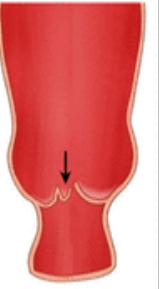
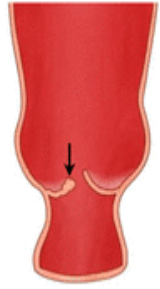
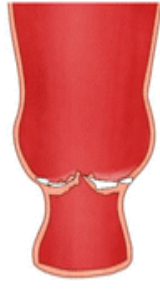
# Aortic insufficiency

- Returning of blood from the aorta to the LV during diastola

## ETIOLOGY

- Valvular insufficiency:

1. Congenital; 2. Rheumatic fever; 3. Infective endocarditis

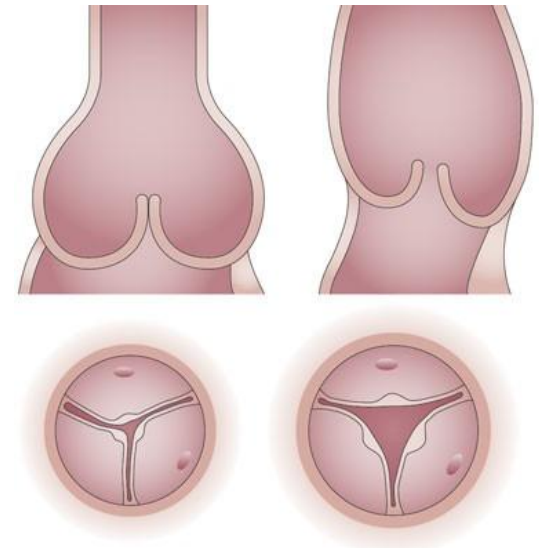
AR Types	Type 1		Type 2		Type 3	Others
	Functional aortic dilatation	Cusp perforation	Cusp prolapse		Cusp retraction	Calcified cusp
Mechanism						
Description	Aortic annular, sinus and/or root dilatation	Perforated cusp of any cause, no other abnormality	Total prolapse of cusp	Partial prolapse of cusp	Retraction with shortening of cusp	Severe calcified, or rheumatic valvular disease that may combined aortic stenosis
Surgical Options	Ascending aorta graft replacement, STJ remodeling, aortic annuloplasty	Patch repair, AVR	Plication, resuspension, AVR	Plication, resuspension, AVR	Shaving, AVR	Infective endocarditis

# Aortic insufficiency

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## ETIOLOGY

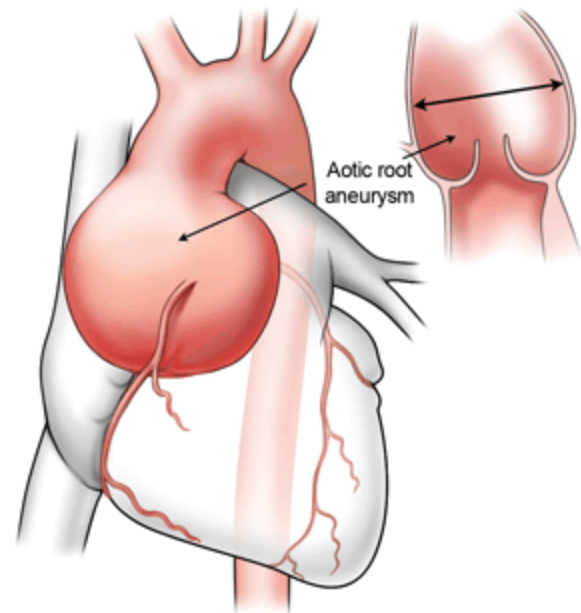
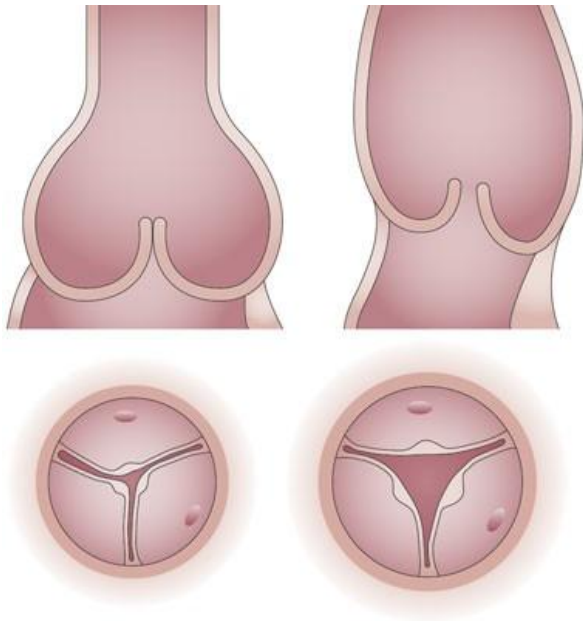
- in addition to disorders of the Aortic valve itself, AI also can occur as a consequence of disorders in the **aortic root dilatation**:
  1. Annuloectation of the aortic valve
  2. Dissecting aortic aneurysm
  3. Infectious diseases (syphilis)
  4. Severe hypertension
  5. Rheumatoid arthritis



# Aortic insufficiency

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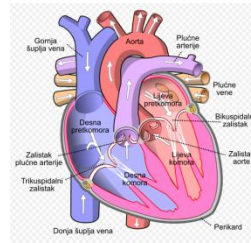
- The basic pathophysiological disorder is the inability of the valve to remain completely closed during diastole with blood returning to the LV from the aorta.



# Aortic insufficiency

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- The main **compensatory mechanisms** in AI are
  1. **Reduction of afterload** (to reduce the volume of blood that is returning to the heart in diastola)
  2. **Increase of preload** (to increase the volume of blood that is ejected in systola) **which leads to increased stress of the LV wall**
- **Consequences of AI:**
  1. **Increased minute volume of the heart in rest** (up to 2 times) with a only slight increase in minute volume during exertion (small effective systolic volume)
  2. **Eccentric hypertrophy and LV expansion**
  3. **Impairment of LV function with dilatation of the heart, reduction of contractile mass and ischemia**

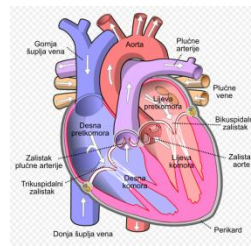


# Aortic insufficiency

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## Clinical manifestations:

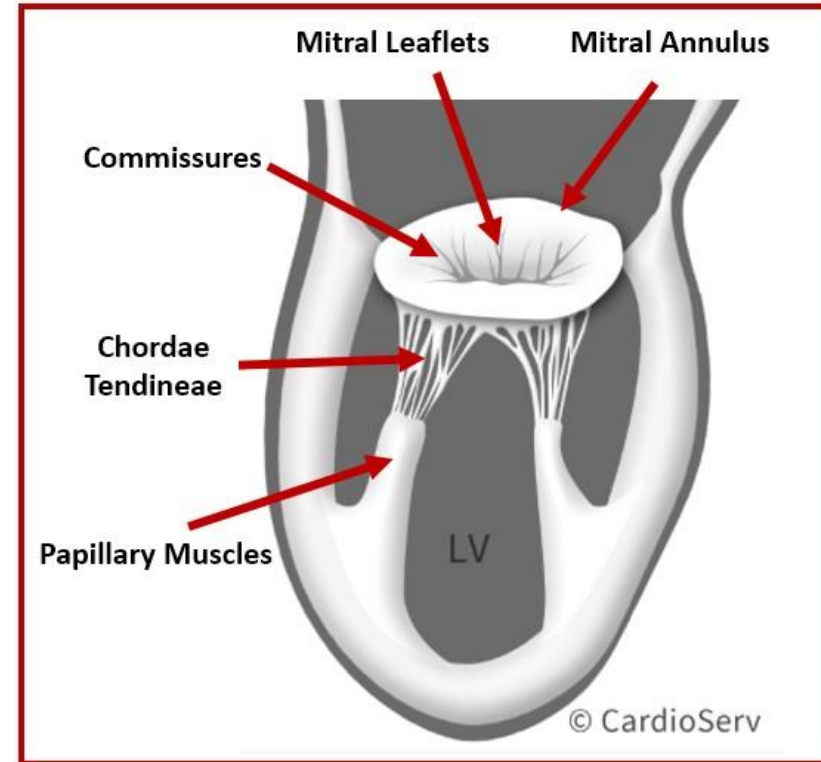
1. Dyspnea
2. Consciousness disorder (syncope)
3. Angina pectoris
4. Insufficiency of the left and then the right heart
5. Changes in the strength of the pulse wave (pulsus magnus et celer - initially strong, quickly loses strength pulse wave)
6. Divergent type of blood pressure (increasing the difference)





# Mitral orifice/device

- Mitral device (6 parts)
  1. Left Atrium
  2. Mitral annulus (ring)
  3. Mitral valves
  4. Chordae tendineae
  5. Papillary muscles
  6. Belonging part of the wall of LV



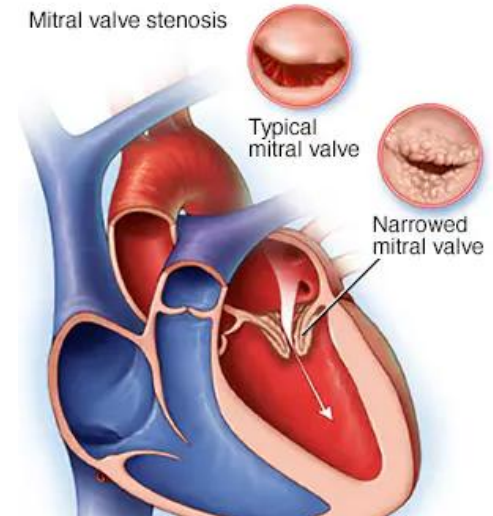
# Mitral stenosis

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- Narrowing of the mitral orifice that interferes with LV filling during diastole

## ETIOLOGY:

1. Rheumatic fever
2. Other non-rheumatic
  - Complex heart defects (Sy Lutembacher = ASD+MS)
  - Carcinoid (Neuroendocrine tumor)



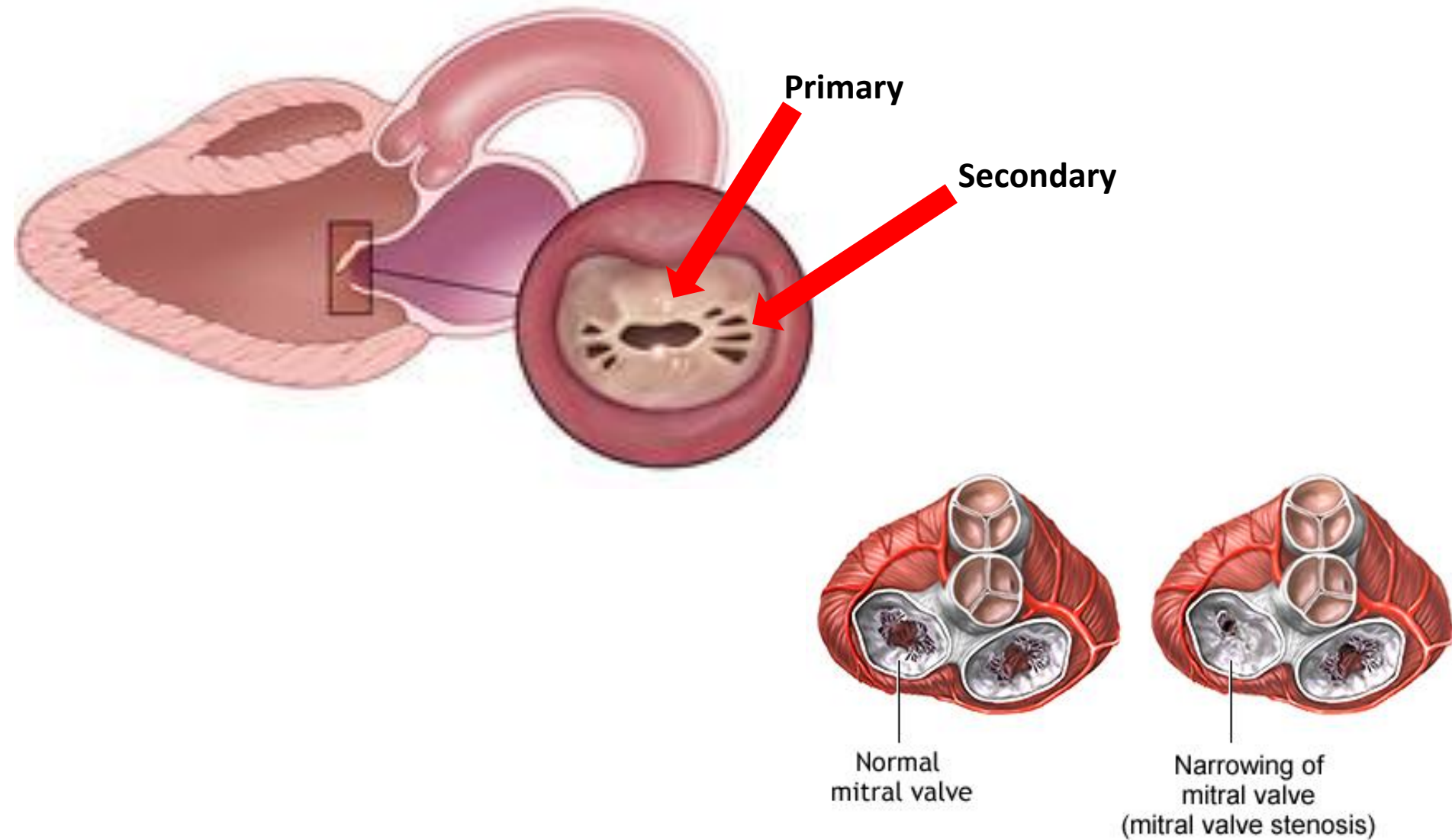
# Mitral stenosis

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- Physiological surface 4-6cm<sup>2</sup>
- In addition to the primary orifice itself, blood also passes between the chords of the papillary muscles (**secondary orifice**).
- Changes to cusp:
  - fibrous thickening
  - fusion of commissures cusps and chordae
  - calcifications
- The border area occurs when the area is reduced to 50% (2.5 cm<sup>2</sup>) by narrowing the "primary" and blocking the "secondary" orifice (symptoms at rest occur when the orifice area is less than 25% - 1.5 cm<sup>2</sup>)

# Mitral stenosis

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# Mitral stenosis

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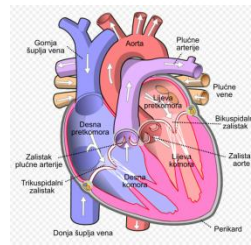
## PATHOGENESIS (sequence of events):

1. Narrowing of the mitral orifice
2. Increased pressure in the LA and its dilation
3. Pulmonary hypertension, 3 stages:
  1. passive pulmonary HTA (retrograde pressure increase in pulmonary veins, capillaries and arteries)
  2. active pulmonary HTA (when the orifice area is about 1-2cm (corresponds to a pressure of 25mmHg), there is an active constriction of the pulmonary arterioles)
  3. irreversible pulmonary HTA long term state (proliferation of medial muscle fibers and thickening of the basement membrane)

# Mitral stenosis

## PATHOGENESIS (continued):

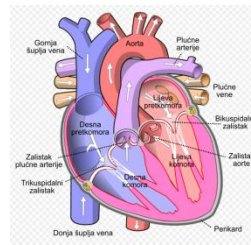
- increased resistance and pressure in pulmonary blood vessels – PHTA
- interstitial and alveolar edema
- relative insufficiency of the pulmonary valve (pulmonary HTA)
- hypertrophy of RV
- dilatation of RV
- relative insufficiency of the tricuspid valve
- right heart failure
  - pretibial edema, hepatosplenomegaly, swollen jugular veins



# Mitral stenosis

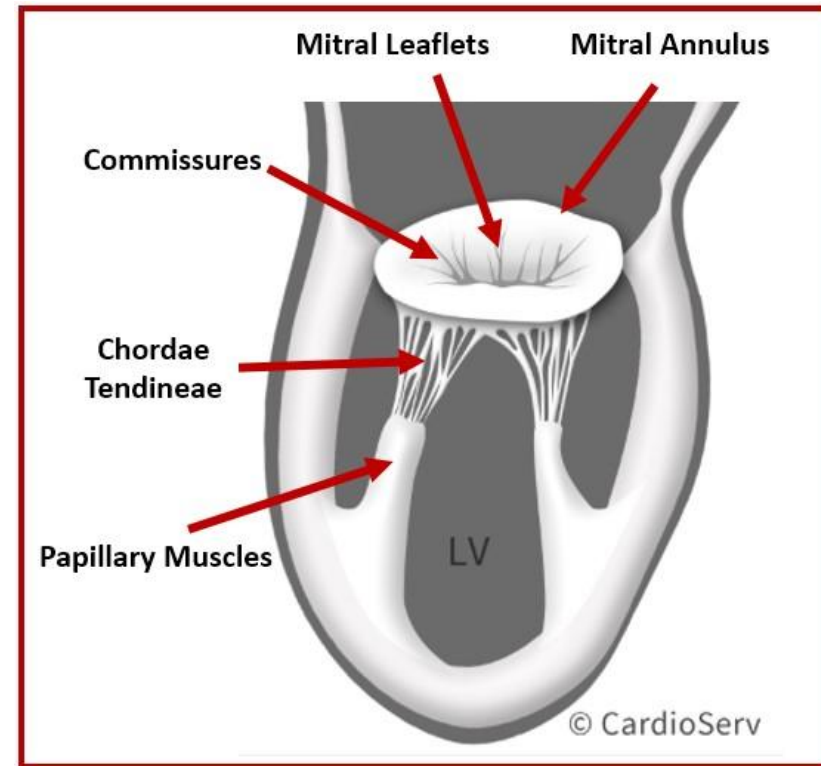
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- Clinical manifestations in summary:
  1. Reduced systolic volume and minute volume
  2. Insufficiency of the left heart
  3. Insufficiency of the right heart



# Mitral insufficiency

- By returning blood from the LV to the LA during systole
- Mitral device (6 parts)
  1. Left atrium
  2. Mitral annulus (ring)
  3. Mitral valves
  4. Chordae tendineae
  5. Papillary muscles
  6. Belonging part of the wall LV



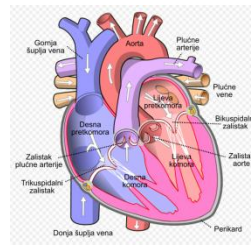


# Mitral insufficiency

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ETIOLOGY of **acute** mitral insufficiency:

- Ischemia during MI
  - papillary muscle necrosis
- Degenerative processes
  - Trauma or spontaneous rupture of the chords
- Inflammatory processes
  - Rheumatic fever
  - Infectious bacterial endocarditis

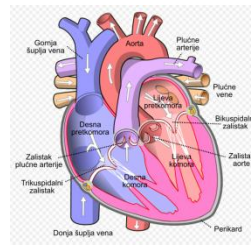


# Mitral insufficiency

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## ETIOLOGY of **chronic** mitral insufficiency:

- Infections
  - Bacterial endocarditis
- Structural changes
  - Papillary muscle rupture
- Degenerative processes
  - Calcification of the mitral annulus,
  - Marfan syndrome (connective tissue weakness)
- Inflammatory processes
  - Rheumatic fever
  - Systemic lupus
- Congenital disorders
  - Cuspis split
- Functional/relative Mitral Insufficiency –
  - Dilation of LV



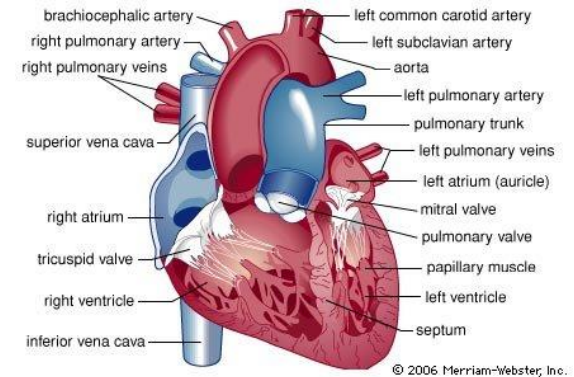
# Mitral insufficiency

## PATHOPHYSIOLOGY:

- regurgitation of blood in LA
- increased blood volume load LA
- hypertrophy and enlargement of LA
- increase in systolic pressure in LA

(but most of the energy during contraction is spent for shortening the myofibrils of the dilated cardiac structures, not for increasing the pressure)

- LA insufficiency
- pressure increase in LA
- increase in pressure in the pulmonary circulation



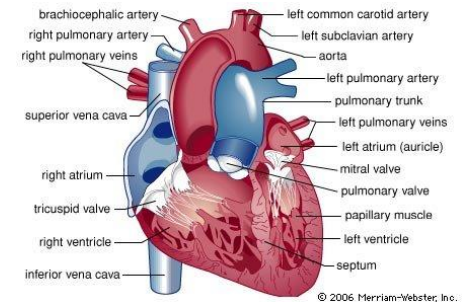
# Pulmonary stenosis

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- It is characterized by reduced blood flow from RV to Tru. Pulmonalis during systola

## PATHOPHYSIOLOGY:

- Increased pressure in RV
- Hypertrophy of RV
- RA hypertrophy
- Increased pressure in the vena cava superior and inferior and peripheral capillaries



# Pulmonary insufficiency

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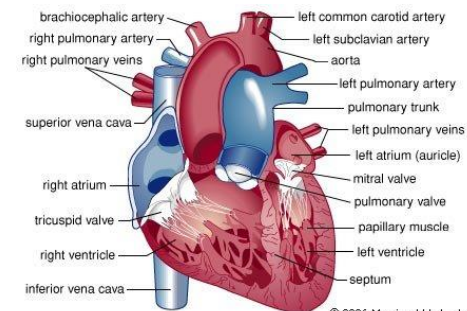
- By returning the blood from Truncus Pulmonalis in RV during diastola

## ETIOLOGY:

1. Relative/functional: pulmonary HTA
2. Organic: infectious endocarditis, rheumatic fever, carcinoid syndrome

## PATHOPHYSIOLOGY:

- Increased RV blood volume load
- Hypertrophy of RV
- RV and RA insufficiency



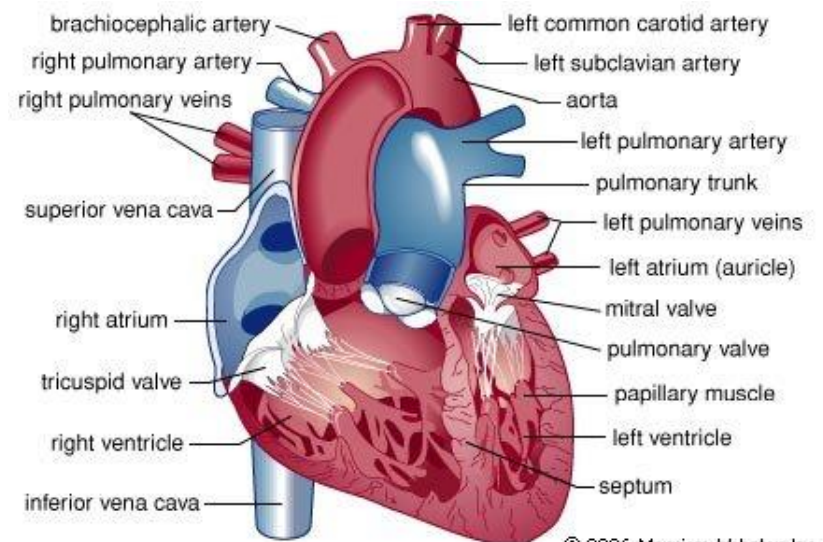
# Tricuspid stenosis

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- By reduced blood flow from RA to RV during diastole

## ETIOLOGY:

- Rheumatic fever (associated with mitral valve disease)
- Carcinoid syndrome (Neuroendocrine tu - amines)
- Systemic Lupus
- Right atrial factors:
  - Thrombus
  - Mixom
  - Metastatic Tu

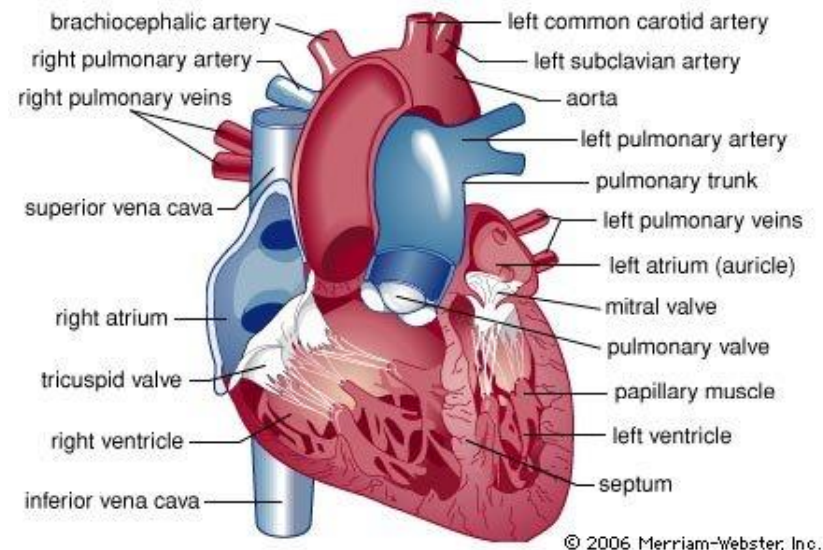


# Tricuspid stenosis

---

## PATHOPHYSIOLOGY:

- Normal area is  $7\text{cm}^2$  (critical narrowing is at  $1.5\text{cm}^2$ )
- Normal pressure gradient  $1\text{mmHg}$  ( $3\text{-}10\text{mmHg}$  is critical due to convergence of pressure in the vena cava systems)
- Two types:
  - Organic
  - Functional



# Tricuspid stenosis

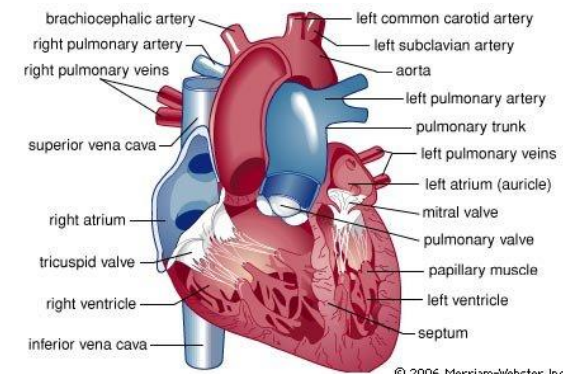
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## Hemodynamic changes:

- Increased pressure in the right atrium
- Increased pressure in the systemic venous circulation - vena cava superior and inferior
- Enlargement of the liver, spleen, pretibial edema

## Clinical manifestations:

- Malaise
- Signs of increased systemic venous pressure
- Hepatosplenomegaly
- Pretibial edema
- Swollen jugular veins of the neck
- Ascites





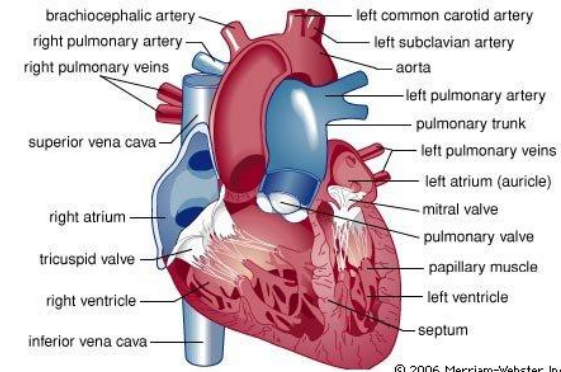
# Tricuspid insufficiency

---

- By returning blood from RV to RA during systola

## ETIOLOGY:

- Functional (extended RV)
  - Pulmonary hypertension
  - Left-right shunt
  - Mitral defect
- Organic (with organic valve damage)
  - Rheumatic fever
  - Bacterial endocarditis
  - Trauma



# Tricuspid insufficiency

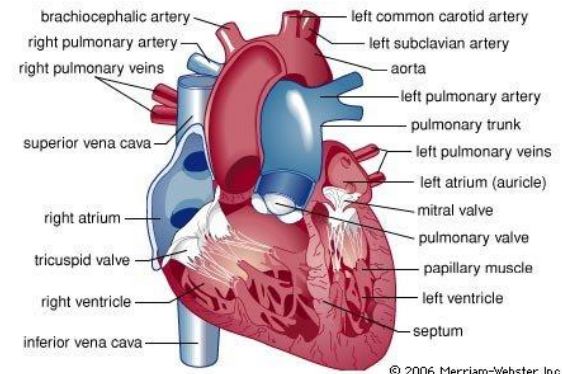
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## Pathophysiology:

- Blood regurgitation in RA
- Increased pressure on RA
- Elevated systemic venous pressure
- Pretibial edema, hepatosplenomegaly, ascites

## Clinical manifestations:

- Malaise (decreased MV)
- Signs of increased systemic venous pressure
  - Hepatosplenomegaly
  - Pretibial edema
  - Swollen jugular veins of the neck
  - Ascites



# Endocardial diseases

---

1. Valvular defects (valve disorders)
  - Stenosis (smaller valve area compared to normal)
  - Insufficiency (insufficient closure of the valve with regurgitation of blood)
2. Rheumatic fever
3. Infective endocarditis

# Rheumatoid fever

---

- Specific aseptic consequence/complication of earlier streptococcal infection (most often pharynx) followed by systemic damage to connective tissue and other organs (heart and brain) **2-3 weeks after the infection**

## ETIOLOGY:

1. Genetic predisposition
2. **Group A beta-hemolytic streptococcus** (strains 3, 5, 18, 19, 24 with **M-Ag capsule** that reduces phagocytosis)
  - Exposure to collectives
  - Poor hygienic conditions

# Rheumatoid fever

---

## PATHOGENESIS (2 theories)

### 1. Immune theory

- Cross reaction
  - membrane Ag streptococci - sarcolemma of heart muscle;
  - Ag streptococcus-cytoplasm of nerve cells of the hypothalamus;
  - Streptococcus M protein-keratin;
- M protein as superantigen (leads to hyper/autoimmune response)
- Impaired T helper response

### 2. Toxic

- Direct toxic effect of streptococci or their toxins Streptolysin O and S.

# Rheumatoid fever

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## CHANGES

### Heart

- Rheumatic pancarditis
  - Endocarditis (dominantly on the left heart; cusps and chordae)
  - Myocarditis (damage to the sarcolemma and cardiomyocytes)
  - Pericarditis (fibrinous exudate with pericardial thickening)

### Joints

- Serous arthritis
- Goat

### Encephalitis, chorea...

# Infective endocarditis

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- **Infectious process on the endocardium** that predominantly affects the valves (but can affect any endocardial structure)
- Types
  1. Acute
  2. Subacute
  3. Chronic

# Infective endocarditis

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## Acute form

### ETIOLOGY

Staphylococcus, Streptococcus, Gonococcus,  
Haemophilus influenzae

- **It predominantly occurs on normal valves**
- In the acute form, damage to the chordae, valves or the muscle occurs quickly and leads to hemodynamic disturbances



# Infective endocarditis

---

## Subacute form

ETIOLOGY (agents with the low virulence but high adherence power for endothelium)

Streptococcus viridans , Enterococcus,  
Pneumococcus, Candida albicans, Aspergillus

- **For previously damaged endothelium**

Tendency to thrombus formation

Altered hemostasis process

# Infective endocarditis

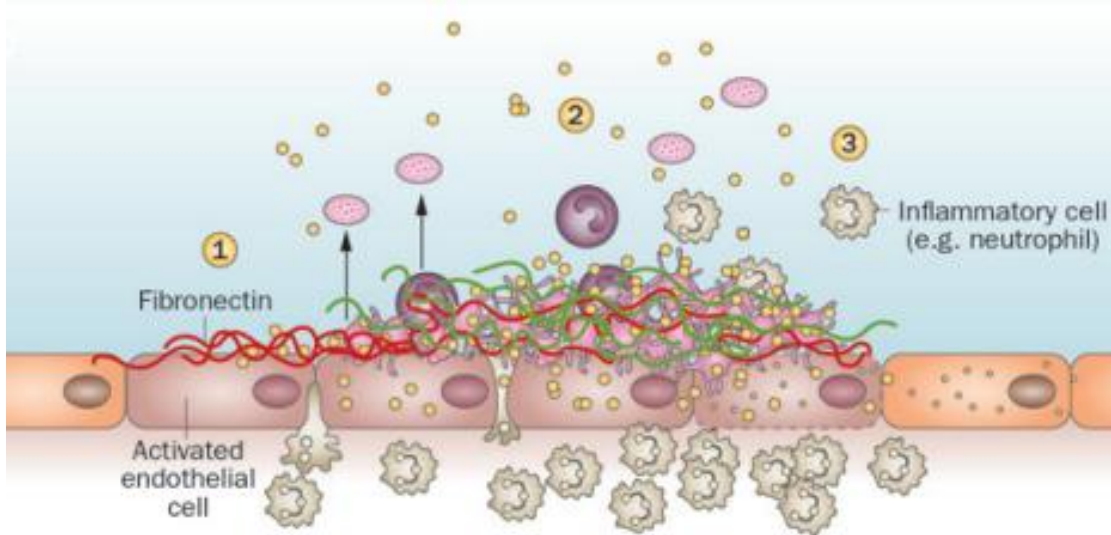
Colonization of valves as a consequence of an inflammatory lesion

Sequence of events

1. **Activated endothelial cells display integrins** that promote a local increase in **fibrinectin**. **Some bacteria like *S. aureus* can adhere to fibrinectin**

2. **Adhered bacteria** cause an endothelial cell response that **secretes cytokines and other proinflammatory factors, clot formation**, and subsequent **growth of vegetations**.

3. **Endothelial cells are damaged by bacteria** or cells of **the immune system** or changes in endothelial cells due to the presence of bacteria



Werdan, K., Dietz, S., Löffler, B. *et al.* Mechanisms of infective endocarditis: pathogen–host interaction and risk states. *Nat Rev Cardiol* **11**, 35–50 (2014).

# Infective endocarditis

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## CONSEQUENCES:

- On the heart
  - Rupture of papillary muscle, chorda, septum myocardium
  - Abscess of the valvular ring with spread to the pericardium and the occurrence of purulent pericarditis
  - Disturbances of the conduction system of the heart
  - Microabscesses and/or vasculitis
  - Stenosis and valve insufficiency
- On other organs - embolus (bacterial colonies)
  - infarcts of the CNS and other organs
  - mycotic aneurysms of arteries

# Infective endocarditis

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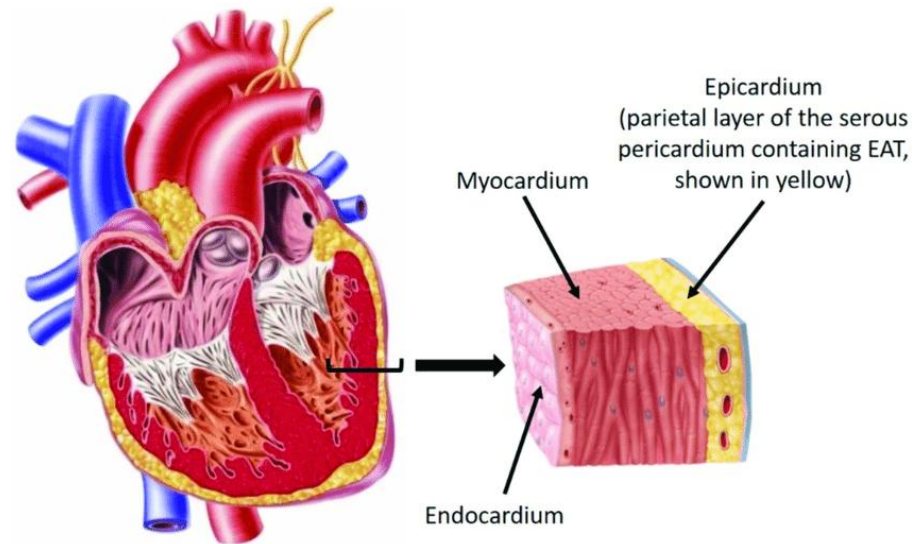
## Consequences

- Immune system
  - Constantly activation of the immune response (bacteremia)
  - Acute and chronic glomerulonephritis
  - Pericarditis

# CONTENT

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- Endocardial diseases
- Myocardial diseases
  - Myocarditis
- Pericardial diseases
- Arterial diseases
- Vein diseases
- Disorders of lymphatic vessels



# Myocarditis

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## DEFINITION

- Inflammatory process localized in the myocardium
- Occurs equally in all ages, but more often in younger men
- **"Disease with 10 faces"** - quite **variable clinical picture**

# Myocarditis

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## ETIOLOGY

- **Viruses**
  - Coxsackie A and B, Echovirus, Influenza, Poliovirus, Cytomegalovirus, HIV, SARS-Cov-2
- Bacteria
  - *Corynebacterium diphtheriae*, Meningococcus, *Borrelia burgdorferi*
- Rickettsia
  - R. Burnetti, R. Rickettsii
- Parasites
  - *Trichinella spiralis*, toxoplasma...
- **Autoimmune causes** – systemic lupus

# Myocarditis

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## PATHOGENESIS

- Myocardial damage occurs as a **result of activated effectors immune mechanisms** for destroying microorganisms

Clinical picture - depends on the **degree of anatomical damage to the myocardium**

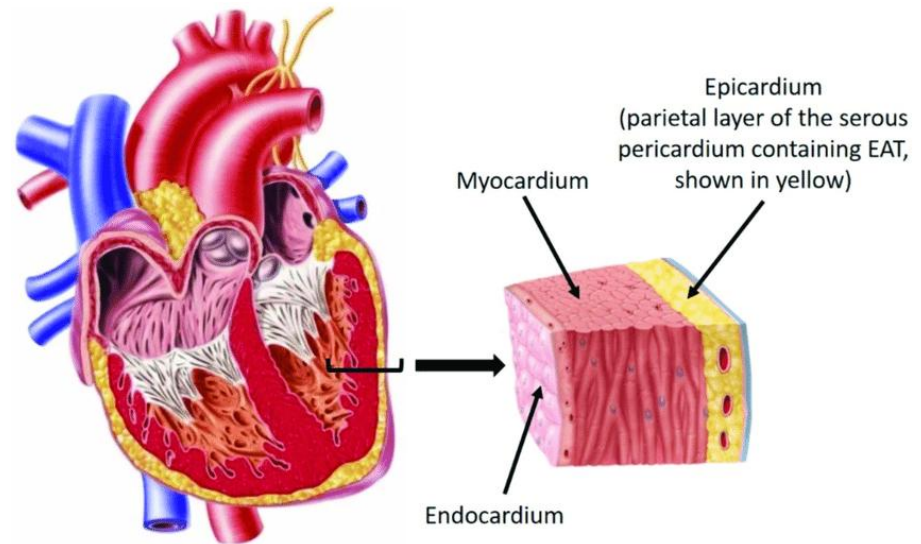
- Symptoms of a respiratory infection usually precede it
- Weakness, rapid fatigue, shortness of breath, cough, palpitations, chest pain and impaired consciousness.
- Rapid heart rate, heart rhythm disorders, weakened tones, decreased blood pressure, peripheral cyanosis.



# CONTENT

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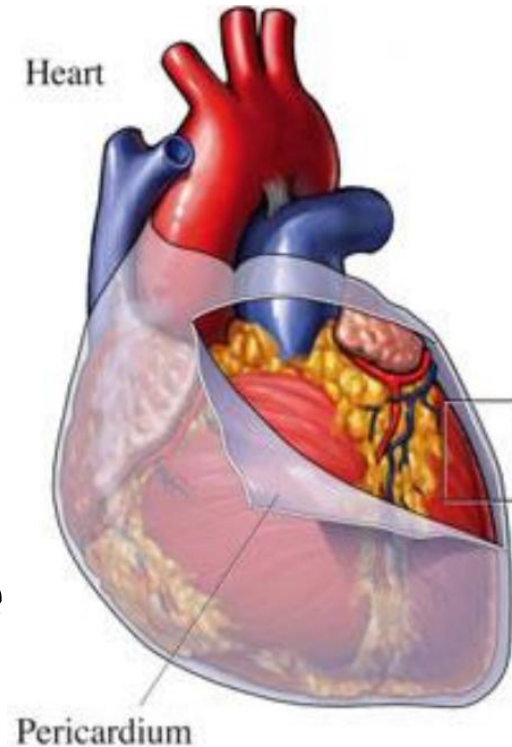
- Endocardial diseases
- Myocardial diseases
- Pericardial diseases
- Arterial diseases
- Vein diseases
- Disorders of lymphatic vessels



# Pericardium

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- Visceral and parietal layer
- The pericardial volume and pressure relationship is exponential type
- The relationship between the pressure in the pericardium and on the right ventricle is linear
- In summary, **even a small increase of volume in the pericardium, which occurs acutely, significantly increases the pressure on the right ventricle**



# Acute pericarditis

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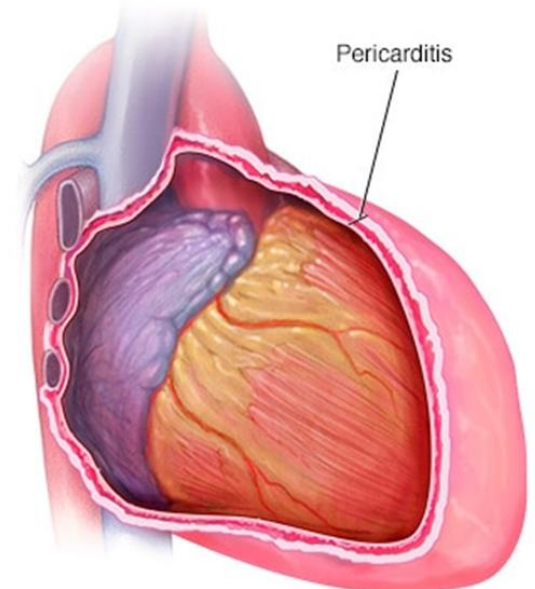
- **Acute inflammation of the pericardium** (accompanied by the formation of exudate and thickening of the pericardium layers)

## ETIOLOGY

1. Infections
  - Coxsackie viruses, Streptococcus and Staphylococcus
2. Autoimmune diseases
3. Radiation therapy

## Clinical manifestation:

- Chest pain
- Changes in the ECG



# Acute pericarditis

---

- Pericardial effusion (two types):
  - In form of **transudate**
    1. LV insufficiency
    2. Hypoproteinemia
  - In form of **exudate** (inflammation)
    1. Sanguinous (TB, neoplasm)
    2. Serosanguinous (trauma, disorders of coagulation)
    3. Chilosy (injury thoracic ductus)

# Acute pericarditis

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- Risk of cardiac tamponade (increase volume)
- Impaired diastolic function of the right heart due to effusion pressure
- The onset of tamponade depends on
  - Effusion volume
  - Effusion rates
  - Pericardial compliance
- Example: a sudden effusion even with a volume of 200 ml can sometimes cause tamponade

# Acute pericarditis

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- Clinical presentation of Cardiac tamponade:
  - Distended jugular veins (increased pressure in RV)
  - Hypotension
  - Weakened heart sounds
  - A paradoxical pulse
    - In inspiration, the reduction of systolic pressure by more than 10 mmHg
    - Reduction in left ventricular volume as a result of increased pressure in the right ventricle
    - Reduction of systolic volume of LV

# Constrictive pericarditis

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- Thickening, fibrosis and calcification of the pericardium
- Loss of elasticity/compliance

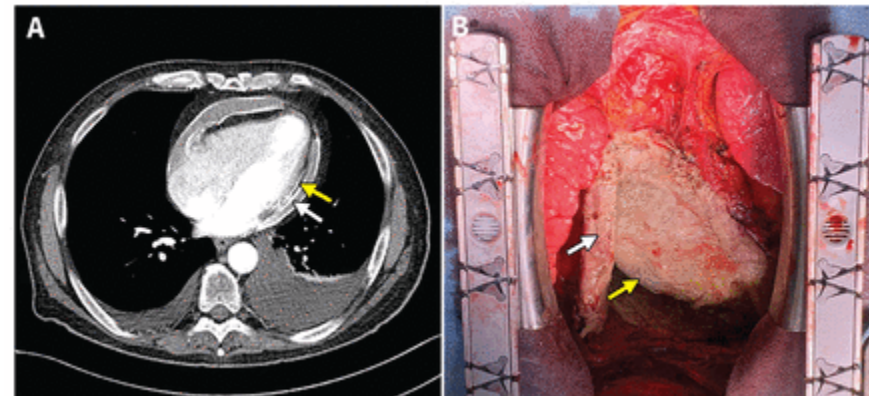
## ETIOLOGY:

- Radiation
- Connective tissue diseases
- Tuberculosis
- Idiopathic causes

# Constrictive pericarditis

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- **The heart is in a rigid shell** that increases their diastolic pressure of both ventriculi and reduces the diastolic blood filling
- **Filling takes place only in early phase of diastole**
  - the **volume of the ventricles quickly increases to the limit** of the pericardium's elasticity
  - in **late diastole filling is significantly reduced** or even absent
  - It is **manifested by right heart failure** with a positive Kussmal test sign (increased venous pressure in inspiration )





# CONTENT

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- Pericardial diseases
- Disorders of blood vessels
  - Aorta
  - Peripheral arteries
    - Occlusive
    - Functional
  - Veins
- Disorders of lymphatic vessels

# Arterial disorders

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- Due to histological differences, the disorders can be divided into:
  - Diseases of the aorta
  - Diseases of peripheral arteries
    - Occlusive diseases
    - Functional diseases

# Aorta diseases

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## 1. Aneurysms of aorta (thoracis and abdominal)

- Dissection of aorta

## 2. Occlusive syndromes:

- acute
- chronic (Sy of visceral branch of abdominal aorta, Sy Lerich ect...)

## 3. Aortitis

# Aorta aneurysms

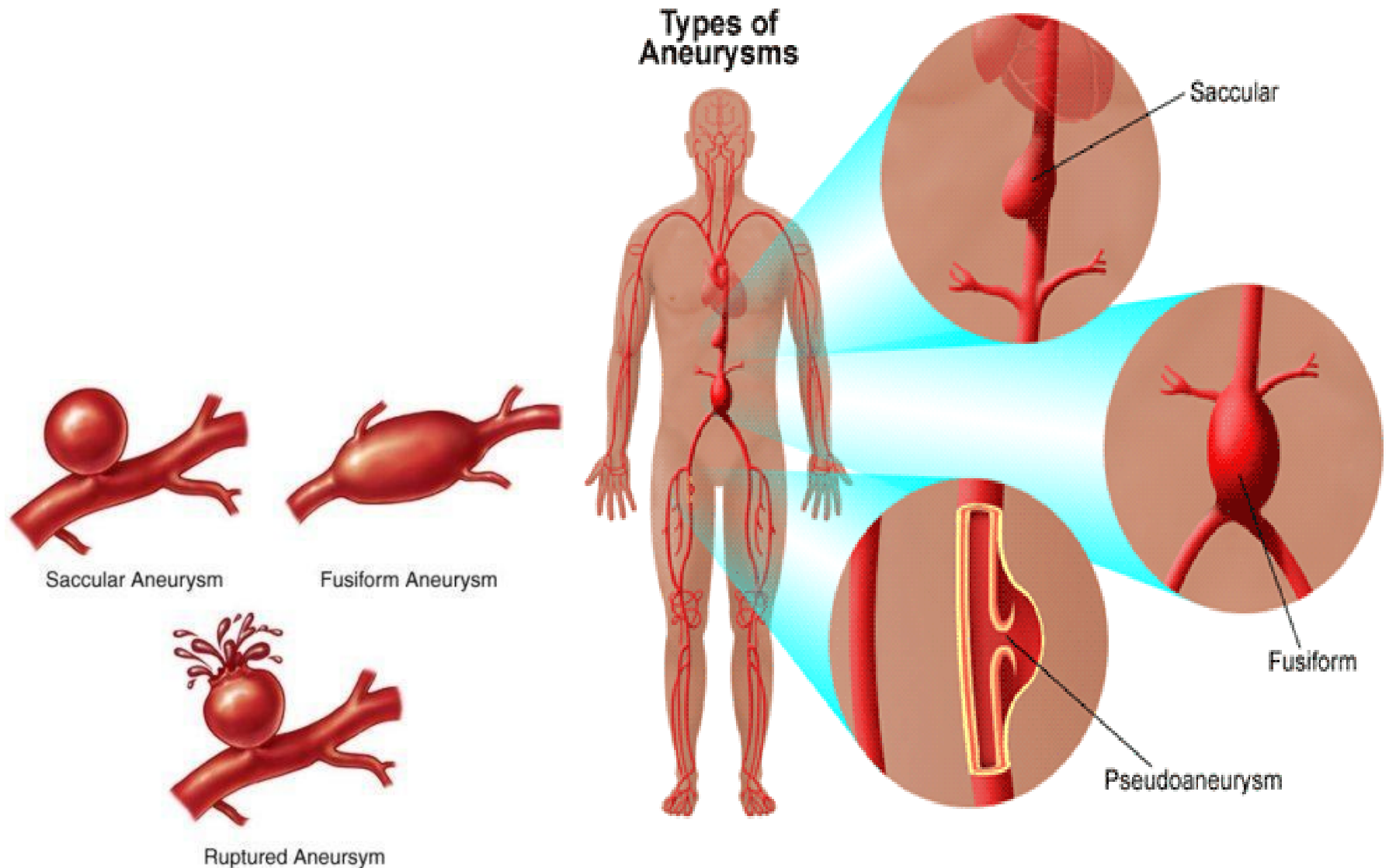
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## Localized or diffuse widening of the aortic wall

Types:

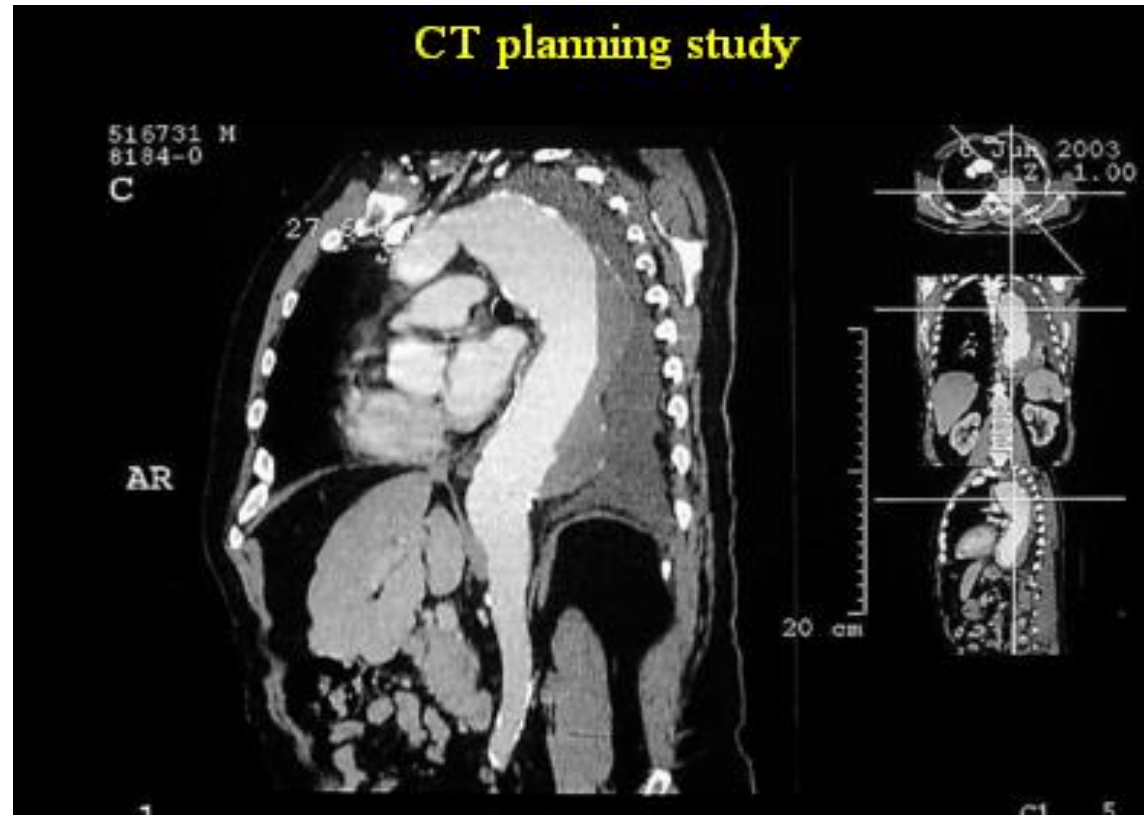
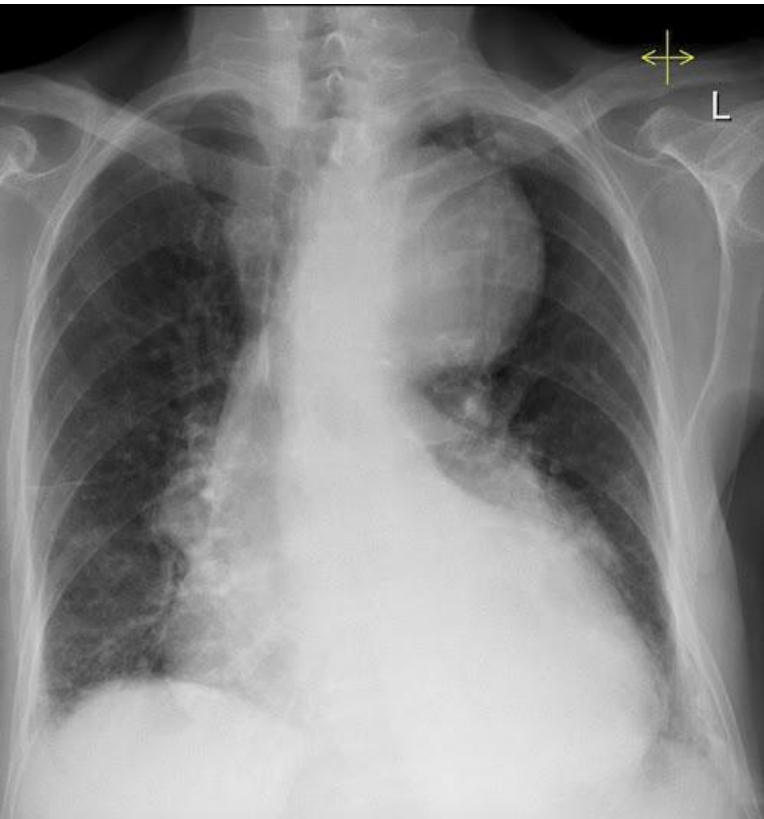
- According to the wall structure:
  - **true** (they have all 3 layers)
  - **fake** (rupture of intima and media)
- According to shape:
  - **baggy** (saccular),
  - **spindly** (fusiform),
  - **dissectant**
- Special forms:
  - **mycotic** (infectious)
  - **AV- aneurysms** ( pathological communication aorta-vein )

# AORTA ANEURYSMS



# AORTA ANEURYSMS

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# ANEURYSMS OF THE THORACIC AORTA

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- Can occur in ascending, decedent part or aortic arch  
**ETIOLOGY:** lues, atherosclerosis, arteritis, trauma, poststenotic
- **Clinical Manifestations :**
  - insufficiency of aortic valves (in ascending part)
  - compressive syndromes on the surrounding tissues:
    - esophagus (dysphagia)
    - trachea (cough, dyspnea)
    - N.recurrens (hoarseness)

# ANEURYSMS OF THE ABDOMINAL AORTA

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- Most common in the population (very often discovered accidentally)

## **ETIOLOGY:**

- atherosclerosis,
  - trauma,
  - sepsis,
  - idiopathic (TNF- $\alpha$  and IL-1 activate Mo / Mf , fibroblasts and endothelial cells, **synthesize metal proteases**).
- **Clinical manifestations**
    - Abdominal pain
    - Compression
    - Rupture



# DISSECANT ANEURYSM OF AORTA

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**Longitudinal split of wall layers of aorta with penetration of blood** between intima and adventitia

## **ETIOLOGY:**

- Cystic necrosis of tunica media
- Increased hemodynamical aortic wall stress (HTA, Coarctation of aorta)

## **PATHOPHYSIOLOGY: (2 mechanisms)**

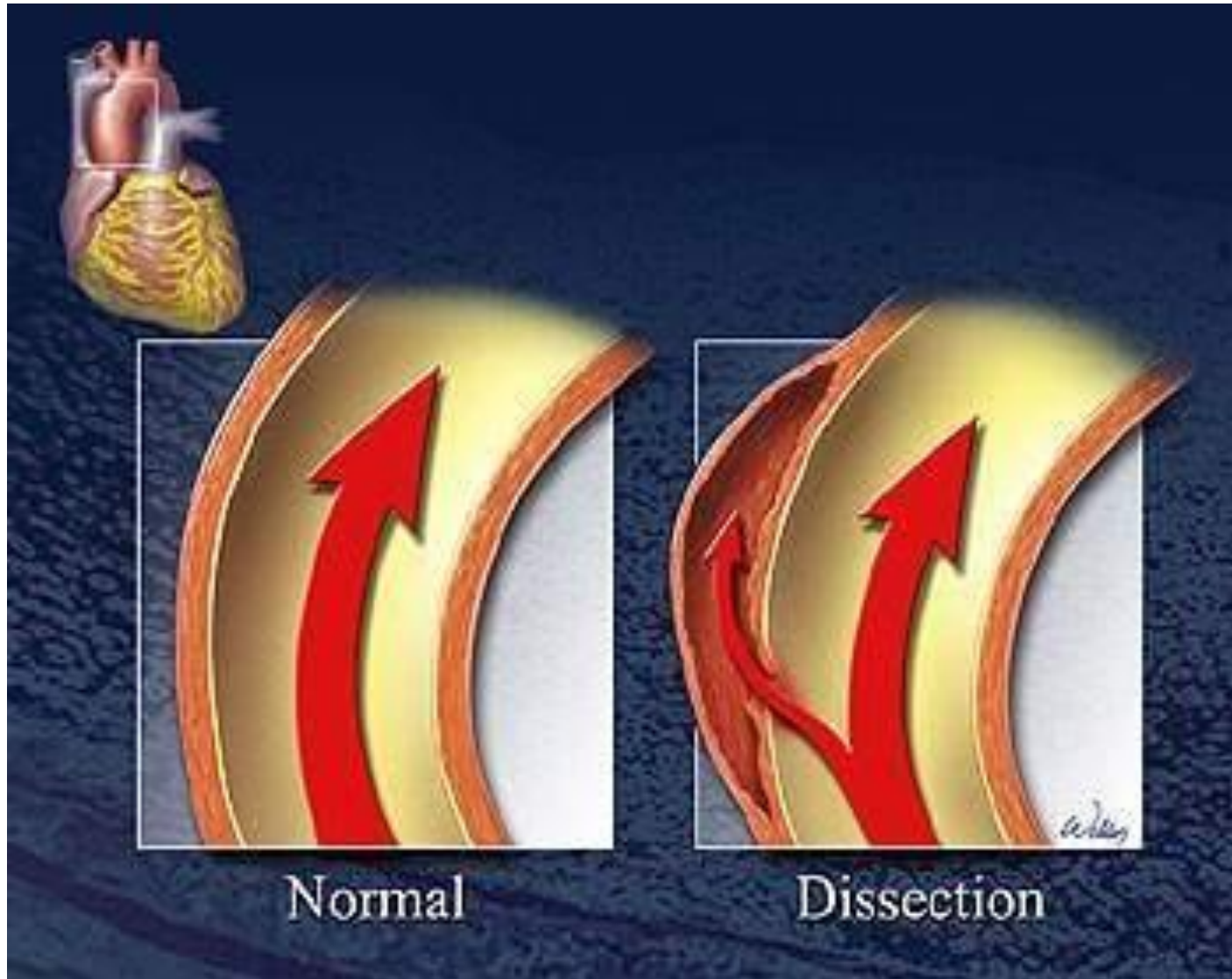
- 1. Degeneration of media**, bleeding from vasa vasorum
- 2. Spontaneous rupture of intima** and penetration of blood in the degenerate media

## **• Types:**

- Type 1 (ascending-descending),
- Type 2 (ascendant)
- Type 3 (descendant)

# AORTIC DISSECTANT ANEURYSM

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# OCCLUSIVE DISEASES OF THE AORTA

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**TYPES: ACUTE or CHRONIC**

- **Acute occlusive diseases of aorta:**
  - 1. Thrombus from left heart - LA and LV** (for ex. in dilatative cardiomyopathy, artificial heart valves, subacute bacterial endocarditis, myxoma LA, AF)
  - 2. Thrombus from the right heart - RA or RV** (paradoxal embolism)
  - 3. Thrombus from aorta** - create "riding thrombus" which is located in the bifurcation of the aorta

# OCCLUSIVE DISEASES OF THE AORTA

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- **Chronic occlusive diseases of aorta:**

## **ETIOLOGY:**

- Atherosclerosis
- Thrombosis
- Lues

- **Types:**

- **Syndrome of aortic arch** (narrowing or obstruction of one or more branches of aorta : carotid, vertebral, subclavian...)
- **Syndrome of visceral branch of aorta** (abdominal/intestinal angina, renovascular HTA...)
- **Sy Lerich** (the final part of the abdominal aorta and the bifurcation on the iliac branches - gradual obstruction with the appearance of gluteal pain and impotence)

# AORTITIS

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- **Inflammatory proces** of the aorta

## **ETIOLOGY:**

- Cardiovascular Lues
- Takayashu arteritis
- Ulcerative colitis
- Psoriatic arthritis
- M. Bechterev
- Sy Reither

# PERIPHERAL ARTERIES DISEASES

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## Occlusive diseases of peripheral arteries

### TYPES: **ACUTE** or **CHRONIC**

#### 1. **Acute occlusion ETIOLOGY:**

##### a) **Embolism:**

- thrombus localized in the heart (heart failure, AMI, LV aneurysm, atrial fibrillation)
- extracardiac thrombus (aorta and peripheral artery aneurysms)
- septic embolism (fibrin and fibrin strands containing bacteria during subacute bacterial endocarditis)
- calcium (valvular defects with calcification)
- tumors (myxoma LA)
- fatty tissue (fracture of long bones)
- air (air embolism)

# PERIPHERAL ARTERIES DISEASES

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## Occlusive diseases of peripheral arteries

### 2. Acute occlusion ETIOLOGY :

- b) **Thrombosis** on the complicated (unstable) atheromatous plaque and thrombosis on the intact blood vessel IBS - heart, CVB - brain ( due to dehydration, use of oral contraceptives ).

# PERIPHERAL ARTERIES DISEASES

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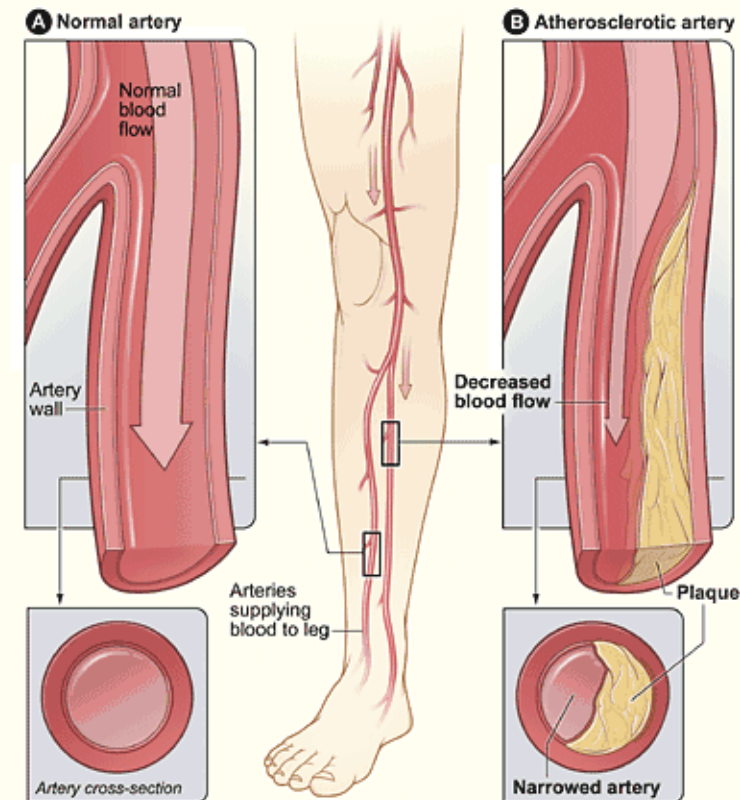
## Occlusive diseases of peripheral arteries

- **Chronic occlusion ETIOLOGY:**
  1. **Obliterating atherosclerosis** (Atherosclerosis obliterans) which is defined as **narrowing of the terminal aorta** and its branches **by atheromatous plaque**
    - Stages in the development of atheromatous plaque :
      - Endothelial dysfunction
      - Fatty stripes
      - Transitional lesion
      - Fibrous plaque
      - Complicated plaque
  2. **Thrombangitis obliterans** (M. Burger) on small and medium vessels
  3. **Non-specific arteritis** (Takayasu)
  4. **Arteritis the connective tissue diseases** (vasculitis)



# PERIPHERAL ARTERIES DISEASES

- Occlusive diseases of peripheral arteries
  - Chronic occlusion
    - Atherosclerosis obliterans



# PERIPHERAL ARTERIES DISEASES

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- **Thrombangitis obliterans (M.Burger)**
- **PATHOPHYSIOLOGY:**
  - segmental **arteritis** of small and medium arteries without calcifications **and thrombophlebitis** of superficial veins, which histologically correspond to panarteritis or panphlebitis (infiltration by lymphocytes, fibroblasts, and giant cells)
  - the **inflammatory process spreads** perivascularly and can affects artery, vein, and nerve
  - of the affected blood vessels **occurs thrombosis**

# PERIPHERAL ARTERIES DISEASES

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- M. Burger
- ## Distal damage

Diminished blood supply causes damage and death of tissue



# PERIPHERAL ARTERIES DISEASES

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## Functional diseases of arteries:

1. **Morbus and syndrome Raynaud**
2. **Acrocyanosis:** permanent and painless cyanosis of hands and feet because of vasoconstriction of arteriole in skin
3. **Livedo reticularis:** reticular cyanosis of hand , trunk and feet
  - primary ( idiopathic ) and secondary ( autoimmune diseases , hematological diseases )
4. **Erythromelalgia:** occasional vasodilation of the small arteries of the hands and feet. It is manifested by redness, local temperature increase, and pain.

# PERIPHERAL ARTERIES DISEASES

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- **Morbus and Syndrome Raynaud's**
- **Definition :** **Spasm of arteriolas of fists** with Intermittent **change of color and temperatures**
- **Types:**
  - **Primary** ( Morbus ) Raynaud: idiopathic, in younger women
  - **Secondary** (Syndrome) Raynaud: autoimmune diseases, intoxications, professional noxe, primary pulmonary hypertension
- **Pathogenesis - Some trigger:**
  - the cold
  - emotional stress etc.

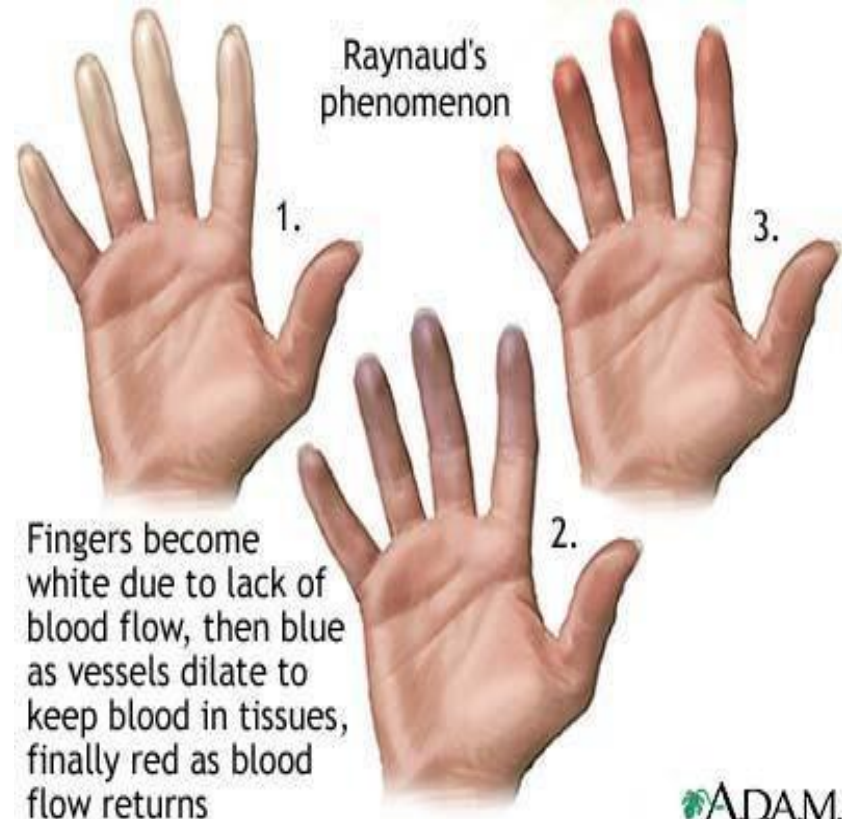
# PERIPHERAL ARTERIES DISEASES

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- **Morbus and Syndrome**  
**Raynaud's**

## Phases:

- Phase I: pallor
- II phase: cyanosis
- III phase: redness (hyperemia)



# VEIN DISEASES

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- *Varicosae Venae*
- *Thrombophlebitis superfitalis*
- *Thrombophlebitis profunda*
- Chronic vein insufficiency

# VEIN DISEASES

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***Varicose veins*** - Dilated, twisted and elongated superficial veins with dysfunctional valves

## **ETIOLOGY:**

- Primary:
  - Innate or constitutional weakness of connective tissues (shortage of collagen and elastin)
  - Bad muscular-venous pump
  - Reduced tone of veins walls
  - Elevated venous pressure
  - But **with with normal deep venus system**



# VEIN DISEASES

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## *Varicose veins*

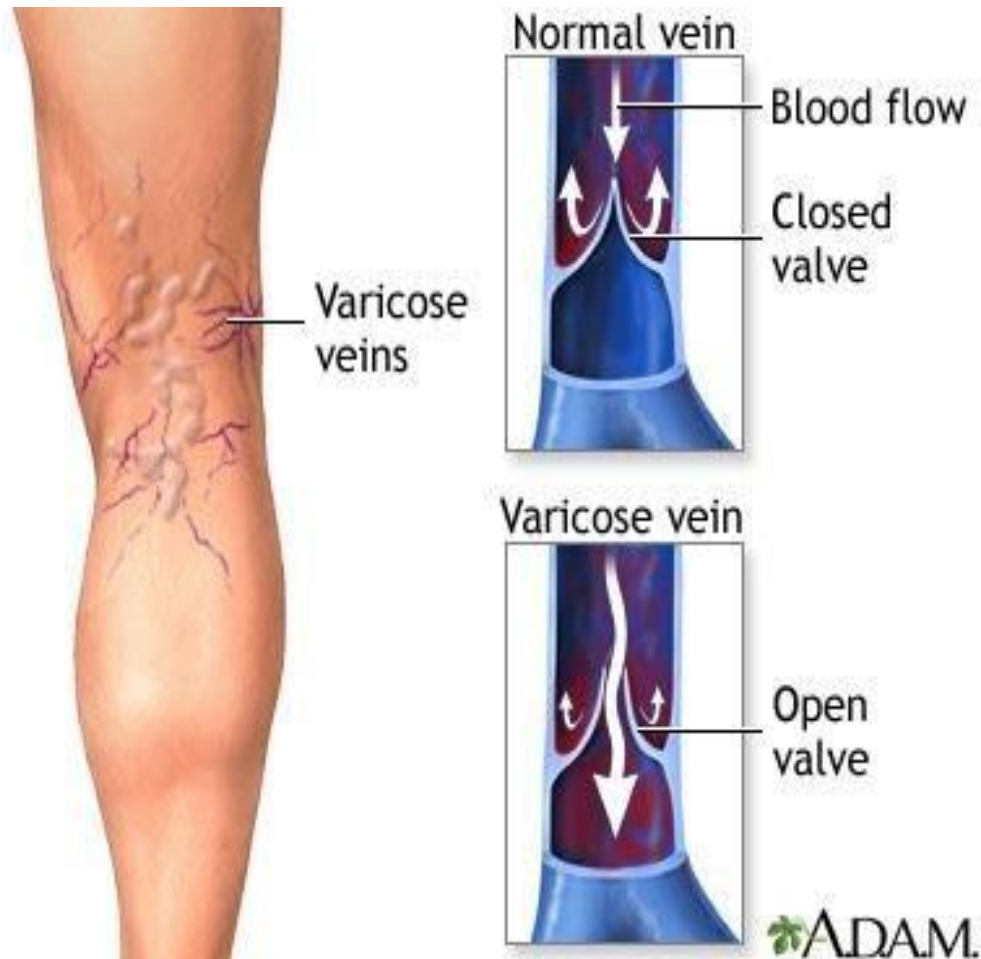
### ETIOLOGY:

- **Secondary:**
  - Superficial varicosities are **the result of proces in deep veins** (obstruction, compression, phlebotrombosis, A-V fistula)
  - **transfer of pressure to the superficial venous system** via venous anastomoses

# VEIN DISEASES

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## *Varicose veins*



# VEIN DISEASES

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- *Varicose veins*



# VEIN DISEASES

## *Thrombophlebitis superficialis*

- Inflammation and thrombosis of superficial vein
- **Etiology :**
  - trauma
  - malignancy
  - oral contraceptives
  - idiopathic (M.Burger)



# VEIN DISEASES

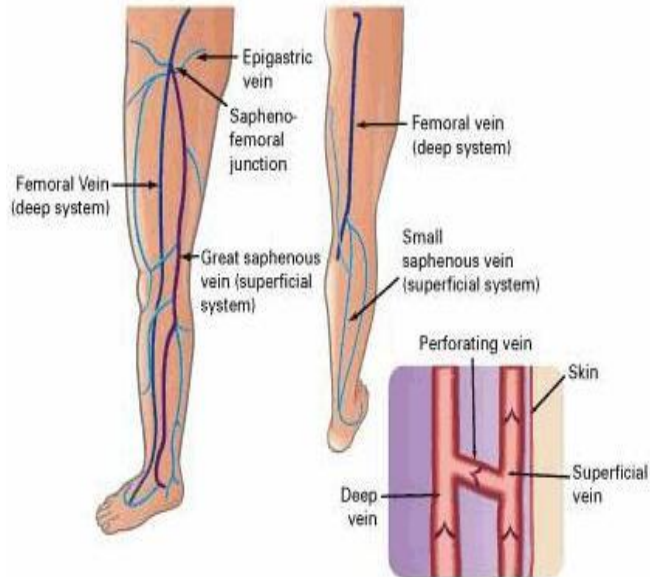
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## *Thrombophlebitis profunda*

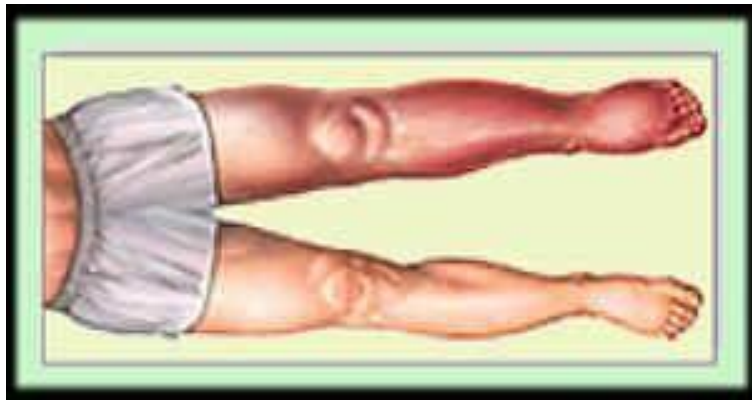
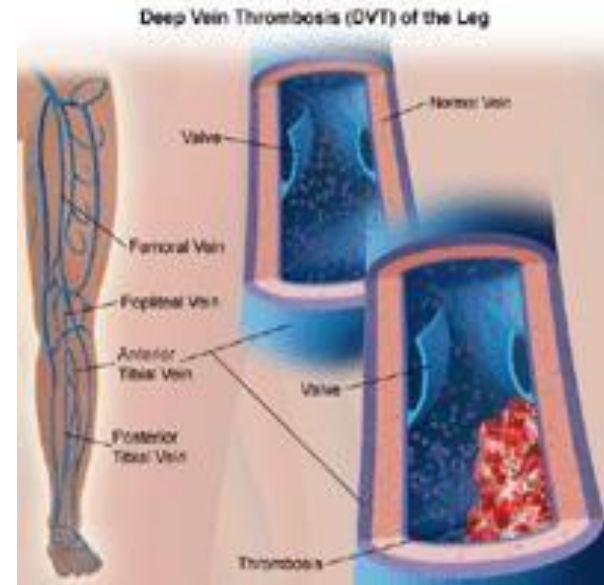
- Creation of thrombus in the deep veins and their consequent inflammation
- **Etiopathogenesis** : disturbed Virchow Triad
  - **Injury of endothelium** (mechanical, chemical, physical and biological factors)
  - **Stasis/turbulent flow** (immobilization, compression)
  - **Blood composition disorder**
    - **Hypercoagulability** (DIC, reduction of AT-III, proteins S and C, dysfibrinogenemia, liver diseases, estrogen) and
    - **hyperviscosity of blood** ( polyglobulia, dehydration, paraproteinemia)

# VEIN DISEASES

## *Thrombophlebitis profunda*



Perforating veins connect the deep system with the superficial system





# VEIN DISEASES

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## ***Thrombophlebitis profunda***

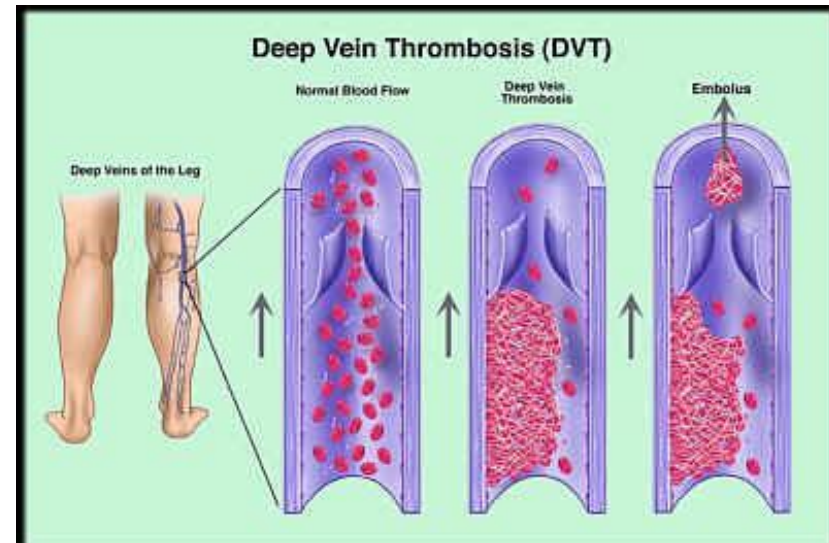
- ***sequence of events:***

1. Platelets aggregation on the veins valves

1. Formation of fibrin network and coagulum

2. Formation of a thrombus "tail" (thrombus growth)

3. Embolization



# VEIN DISEASES

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## **Chronic vein insufficiency (weakness)**

- **Permanent and irreversible disorder of local venous circulation**
- **Etiology :**
  - Repeated deep vein thrombosis
  - Compression of veins
  - AV fistulae
  - varicose veins



# VEIN DISEASES



# LYMPH VESSEL DISORDERS

## Lymph Nodes and Vessels

