HEART DISEASES
ARRHYTHMIA

February 27, 2014

Myocarditis

Inflammation of the heart muscle

Classification

- **specific and non-specific** (specific –when inflammation is granulomatous).
- **acute, subacute and chronic** – depending upon the duration of inflammatory response.
- **infectious and non-infectious** – depending on etiology.

Infectious causes

- **Viral** – coxsackie B virus, Epstein-Barr virus, cytomegalovirus, influenza A and B, herpes.
- **Bacterial** – diphtheria, tuberculosis, salmonella, tetanus, pyogenic bacteria.
- **Spirochetal** – syphilis, leptospirosis.
- **Fungal** – candidiasis, aspergillosis.
- **Rickettsial** – typhus.
- **Protozoal** – toxoplasmosis, malaria.
- **Helminthic** – trichomonosis, filariasis.

Non-infectious causes

- **Cardiotoxins** – catecholamines, cocaine, alcohol, carbon monoxide, arsenic, heavy metals (copper, lead, iron).
- **Hypersensitivity reactions** – antibiotics, diuretics, insect bites (bee, wasp, spider, scorpion), snake bites.
- **Systemic disorders** – collagen-vascular diseases, sarcoidosis, celiac disease, thyrotoxicosis, hypereosinophilia.
- **Idiopatic myocarditis (Fiedler's)**

Clinical manifestation

- excessive fatigue,
- chest pains,
- unexplained sinus tachycardia,
- congestive heart failure
- low voltage QRS complexes,
- ST elevation, or heart block.
- pulmonary edema and cardiomegaly.

Cardiac failure

A state in which impaired cardiac function is unable to maintain an adequate circulation for the metabolic needs of the body

- In most cases cardiac insufficiency is manifested by a decrease in cardiac output
- Cardiac output (CO) is the volume of blood ejected from the left ventricle each minute.

CO= Heart rate*Stroke Volume

Cardiac failure classification

- **Myocardial** – due to direct affection of myocardium
- **Overload** – due to heart overload.
- **Mixed** – due to combination of myocardium direct affection and its overload.

Heart overload

- **Increased pressure load (afterload)** is observed at systemic and pulmonary arterial hypertension, valvular stenosis (mitral, aortic, pulmonary), chronic lungs diseases.
- **Increased volume load (preload) -** valvular insufficiency, severe anemia, thyrotoxicosis.

Cardiac failure classifications

- **Acute cardiac failure** - sudden reduction in CO resulting in systemic hypotension
  - acute myocardial infarction
  - acute intoxications
  - ruptures of the ventricle walls or valves
- **Chronic or congestive cardiac failure** - compensatory mechanisms try to maintain the CO
  - ischemic heart disease
  - systemic arterial hypertension
  - chronic lungs diseases

Left ventricle failure, right ventricle failure, and mixed forms

Left ventricle failure

- **pulmonary congestion and lungs oedema**

High pulmonary venous pressure leads to extravasation of the fluid to lungs tissues.

- **low perfusion and decreased O_2 supply** of all the tissues due to decreased left ventricular output.

Consequences: kidney's ischemic necrosis, hypoxic encephalopathy, weakness and fatigue.

Right ventricle failure

- increased systemic venous pressure.
- edema (feet, ankles, abdominal viscera, especially liver).
  - impaired liver breaks down less aldosterone, further contributing to fluid accumulation.
  - GI - disoders (anorexia, malabsorption, chronic blood loss).
- ascites - fluid accumulation in the peritoneal cavity.

Cardiac failure classification

- **primary heart failure** (cardiogenic form) - IHD, AMI, myocarditis
- **secondary heart failure** (non-cardiogenic form) - acute profound blood loss, collapse; exudative pericarditis

Cardiac failure symptoms

- **Shortness of breath ("dyspnea") -** due to excess fluid in the lungs.
- **Fatigue -** due to low cardiac output.
- **Persistent coughing** – fluid accumulation in the lungs
- **Edema** swelling of the feet, ankles, legs, abdomen.
  - Kidneys retain NaCl and water → venous and the capillary pressure increases → loss of fluid into the interstitial fluid volume.

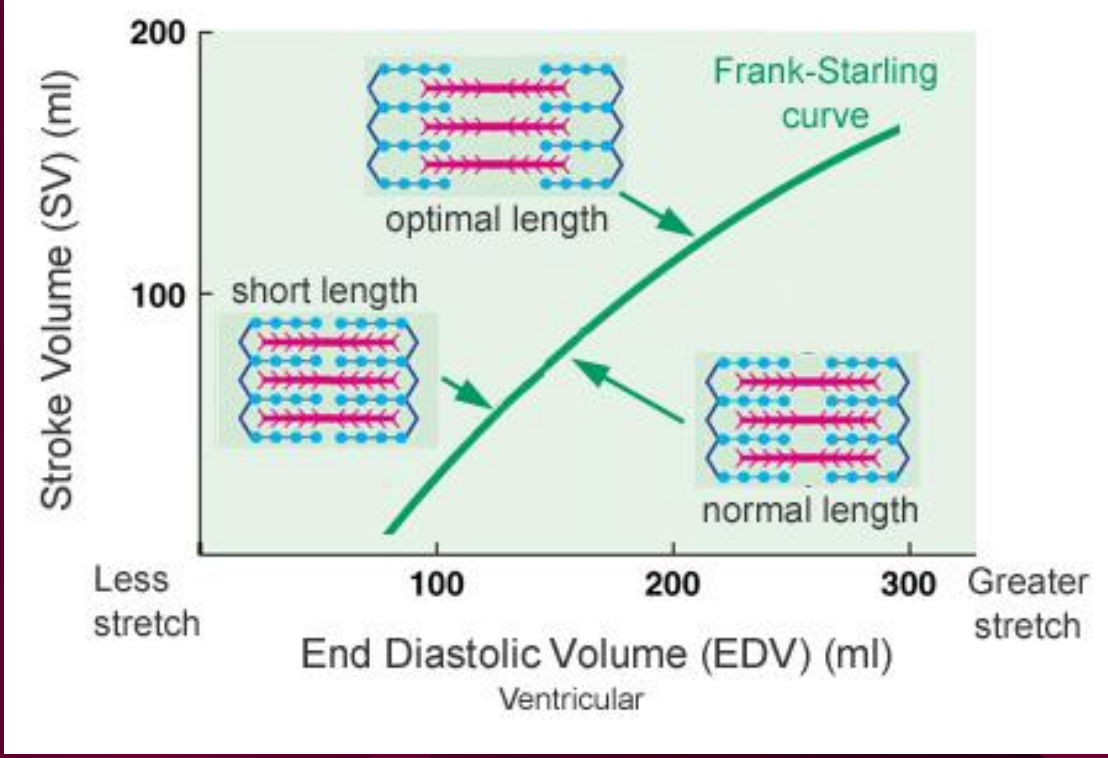
Urgent mechanisms of compensation

CO= Heart rate * Stroke Volume

Tonogenic dilatation of the heart - increased length of ventricular fibers results in increased stroke volume

Further dilatation weakens the work of the heart (myogenic dilatation)

Frank- Starling´s law of the heart



Urgent mechanisms of compensation

- **Increased sympathetic tone -** the constriction of blood vessels and tachycardia
- Constriction of the afferent renal arterioles → decreased glomerular filtration rate → activation of renin-angiotensin-aldosterone cascade → increased salt-and water-retention

Long-term mechanism of compensation

Myocardial hypertrophy

- **Physiological hypertrophy** - high stroke volume - develops in high muscular activity (sportsmen, dancers, workers).
- **Pathological hypertrophy -** low stroke volume - number of nervous fibers and blood vessels does not corresponds to increased mass of myocardium.

Reasons of pathological hyperthrophy

Heart diseases: Myocardial disorders, pericarditis, valvular disorders, congenital heart disease.

Vascular disorders: atherosclerosis, systemic hypertension.

Diseases of the lungs and pleura.

Acromegaly, anaemia, obesity, thyrotoxicosis, severe physical work and sports.

Ischemic heart disease

IHD or coronary artery disease - imbalance between the myocardial supply and its demands in oxygenated blood

- **The reasons of increased oxygen demand**:
  - Exercises,
  - Infectious diseases,
  - Pregnancy,
  - Increased BMR (basal metabolic rate) in hyperthyroidism,
  - Hypertrophy of cardiac muscle

Etiology of IHD

- **The reasons of low oxygen supply**:
  - Atherosclerosis,
  - Spasm of arteries,
  - Thrombus and Embolism,
  - Shock, Anemia, CO poisoning,
  - Lung diseases
- **Risk factors for IHD**
  - high blood cholesterol,
  - high blood pressure (hypertension),
  - physical inactivity, smoking, obesity

Angina pectoris

Angina pectoris is chest pain due to ischemia of the heart muscle.

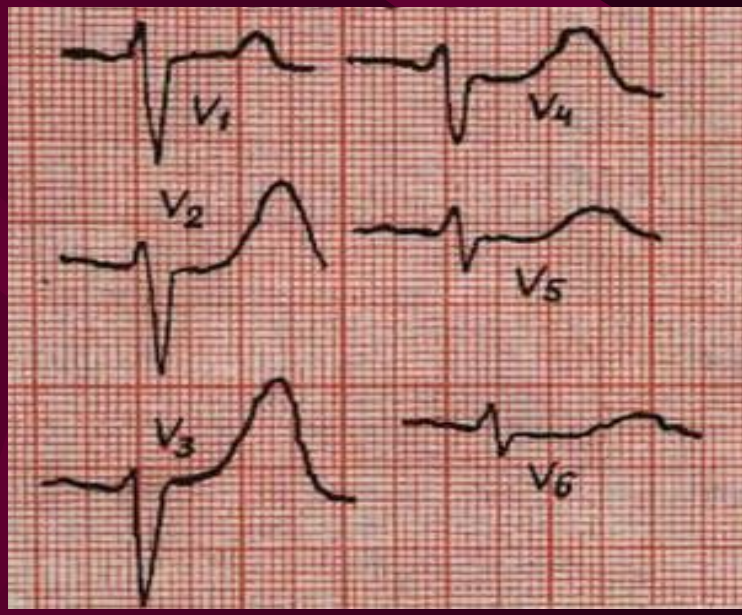
- Greek ankhon ("strangling") + Latin pectus ("chest")
- chest discomfort (pressure, heaviness, tightness, squeezing, burning, etc.)
- location - chest, epigastrium, back, neck, jaw, shoulders
- pain radiation - arms, shoulders, neck into the jaw.

Angina pectoris

Hypoxia ⟷ **Ischemia**

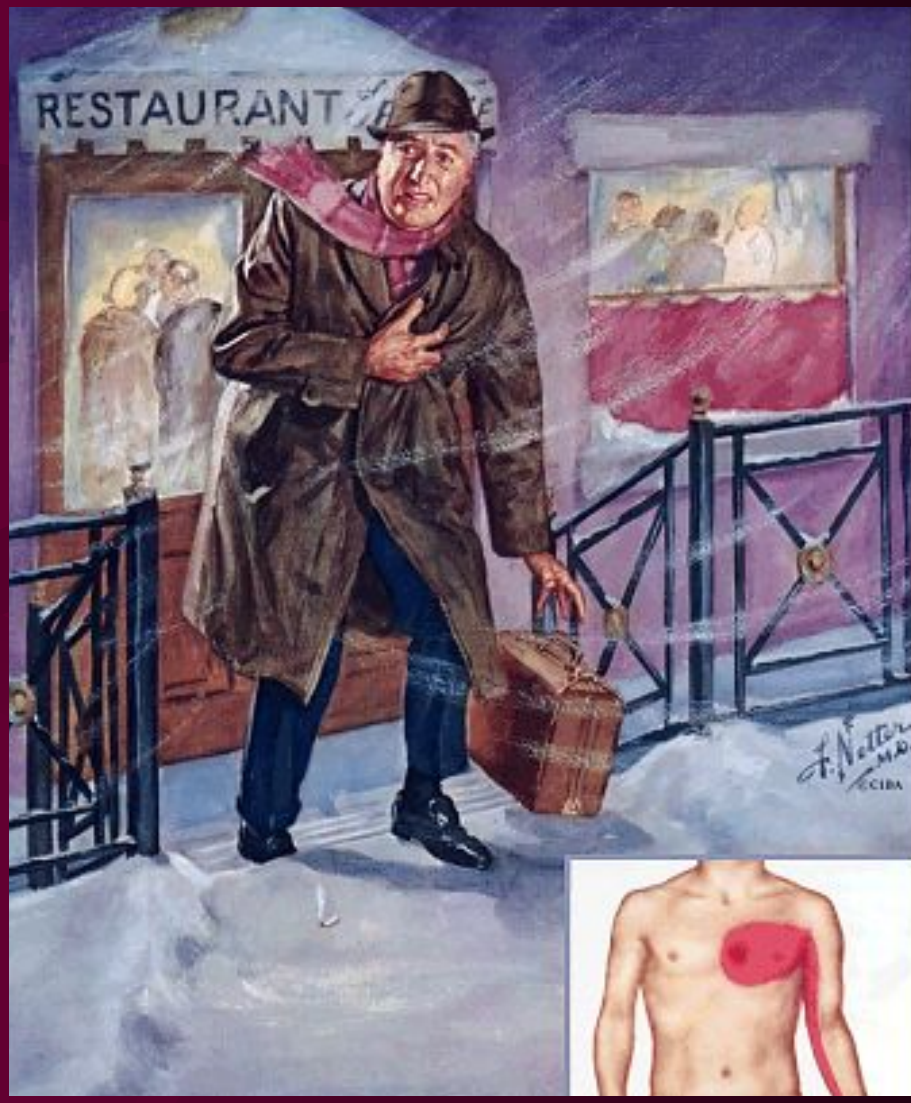
Acidosis

Myocardial cells injury



Angina pectoris

- **Triggers of angina:**
  - **physical exertion**
  - **emotional stress**
  - **heavy meals**
  - **extreme cold and heat,**
  - **excessive alcohol consumption**
  - **cigarette smoking**



Myocardial infarction

Death or necrosis of myocardial cells

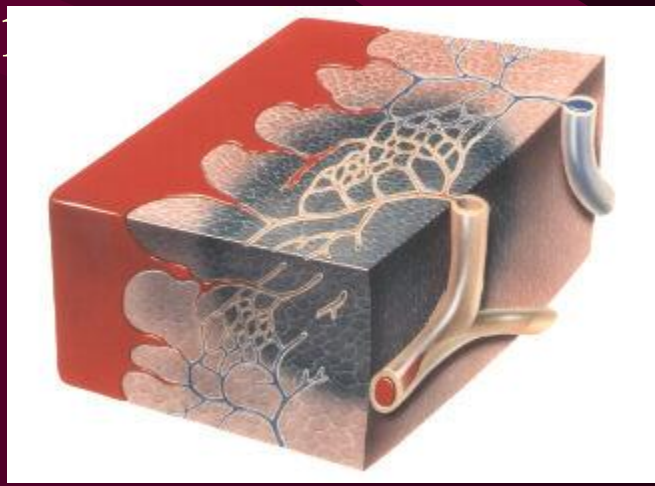
Etiology

- increased myocardial metabolic demand
  - physical exertion, severe hypertension, severe aortic valve stenosis
- decreased delivery of oxygen and nutrients to the myocardium via the coronary circulation
  - thrombus coronary occlusion,
  - fixed (atherosclerosis) or a dynamic coronary artery stenosis.

Myocardial infarction

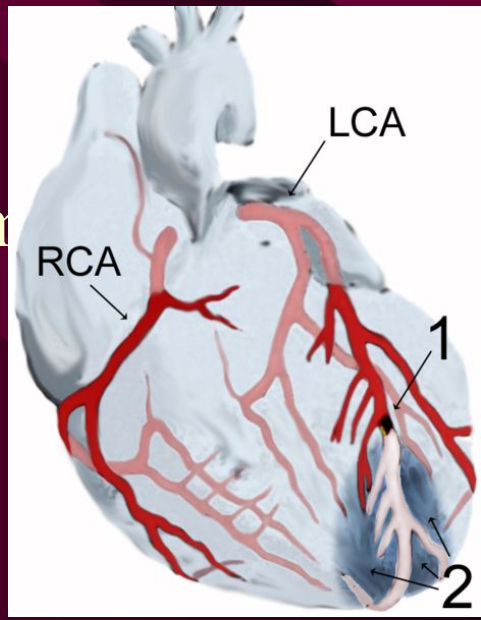
The severity of MI is dependent on:

- level of the occlusion in the coronary artery
- length of time of the occlusio
- presence or absence of collateral circulation.



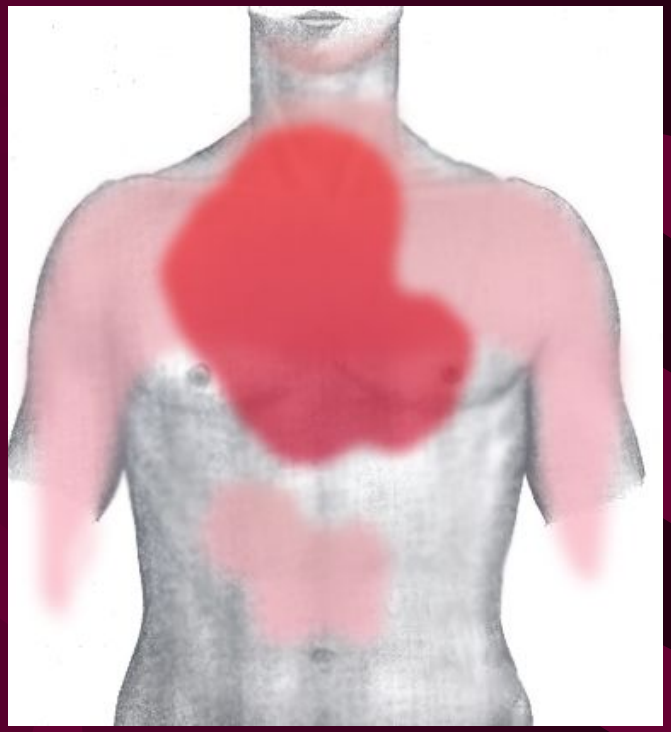
Myocardial infarction

- The death of myocardial cells first occurs in the endocardium, than it is spread to the myocardium and epicardium.
- After a 6- to 8-hour period of coronary occlusion, most of the distal myocardium has died.
- The extent of myocardial cell death defines the magnitude of the AMI.



Signs and symptoms of MI

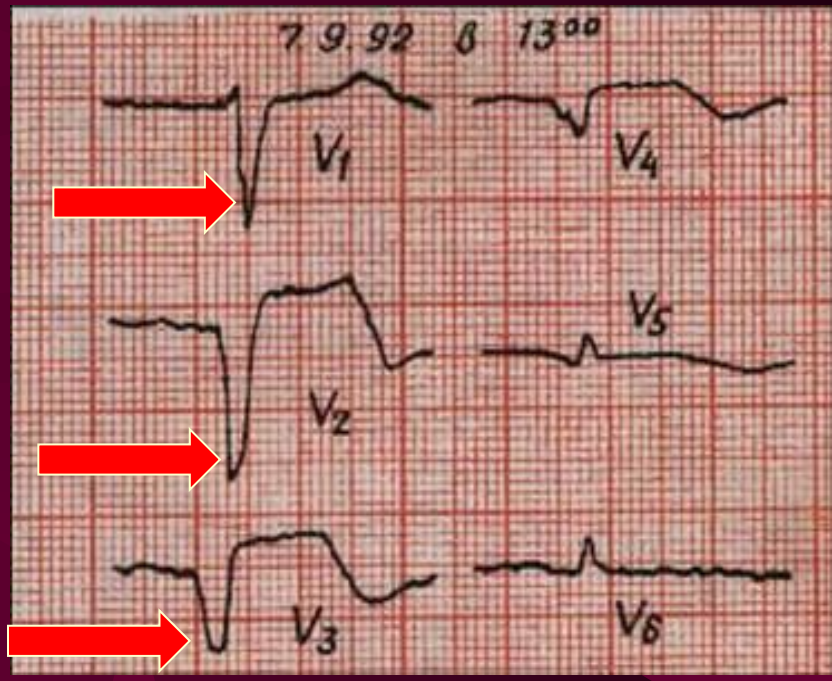
- Chest pain
- Radiation of chest pain into the jaw/teeth, shoulder, arm, and/or back
- Associated dyspnea or shortness of breath
- Associated epigastric discomfort with or without nausea and vomiting
- Associated diaphoresis or sweating
- Impairment of cognitive function without other cause



pain location in MI

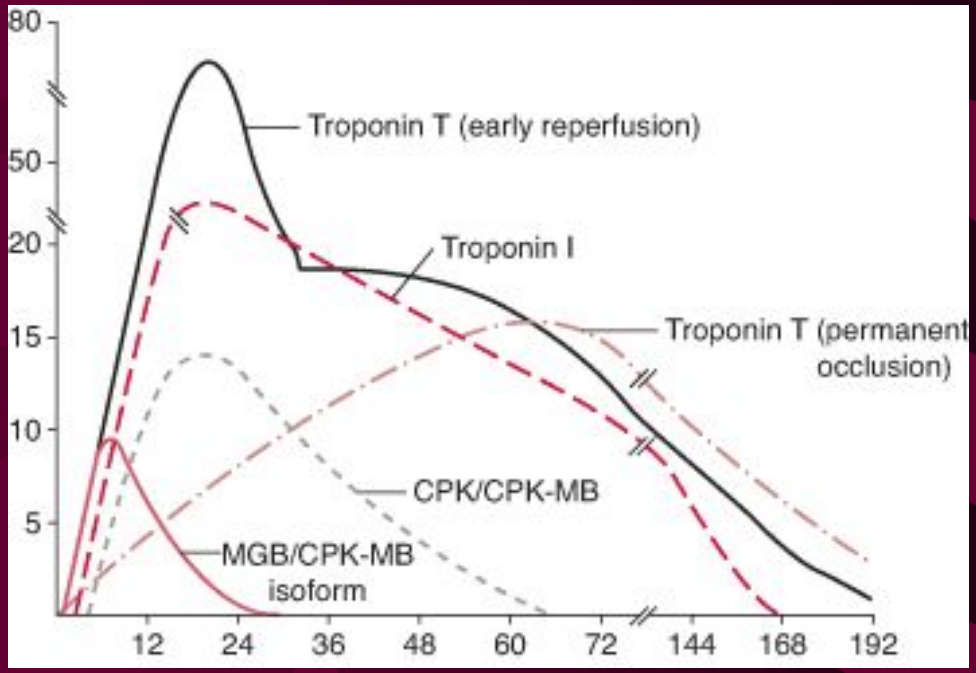
Signs and symptoms of MI

- A wide and deep Q wave in the ECG is a lesion wave, and the sign of transmural MI.
- When only part of the wall is necrotic there are deeply inverted, symmetrical T-waves (*coronary T- waves*) and mostly ST depression are observed in the ECG.



Signs and symptoms of MI

- Enzymes and proteins concentration in a blood correlates with the amount of heart muscle necrosis.
  - creatin phosphokinase (CPK)
  - troponin
  - myglobin



Reperfusion of MI

- circulation brings neutrophils to re-perfused tissues that release toxic oxygen radicals and cytokines (inflammation with additional injury).
- reperfusion brings a massive influx of Ca++ which leads to activation of enzymes  progressive destruction of all cell structures.

Cardiogenic shock

- Cardiogenic shock is a severe reduction of cardiac output
- The pulmonary capillary wedge pressure is normal or elevated in contrast to other types of shock (blood loss or vasodilatation).
- The cardiac pump do not get rid of the blood volume received and it is therefore accumulated in venous system
- The lower part of a body is filled with blood in distensible vessels, and the upper part of the body is pale.

Cardiogenic shock symptoms

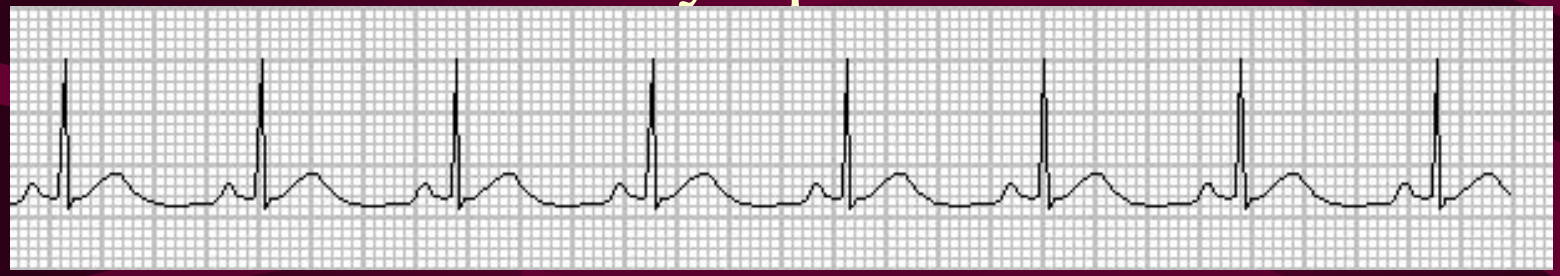
- Anxiety, restlessness, altered mental state
- Hypotension
- A rapid, weak, thready pulse
- Cool, clammy, and mottled skin (cutis marmorata)
- Distended jugular veins
- Oliguria (low urine output)
- Rapid and deep respirations (hyperventilation)
- Fatigue

Arrhythmia classification

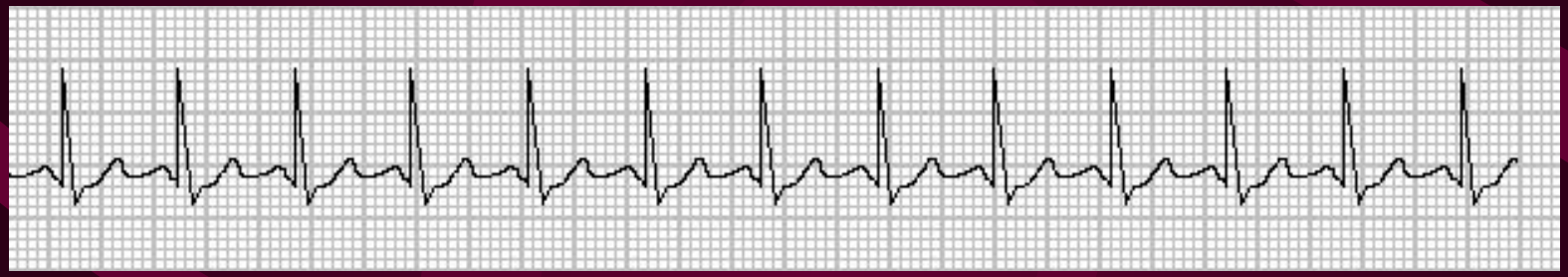
Function disturbed	**Arrhythmia type**	**Examples**
automatism	chronotropic	tachycardia bradycardia
conductivity	dromotropic	blocks
excitability	bathmotropic	extrasystoles
contractility	inotropic	pulse alternans

Pathology of automatism

- Sinus tachycardia – heart rate above 100 bpm - due to increased sympathetic tone



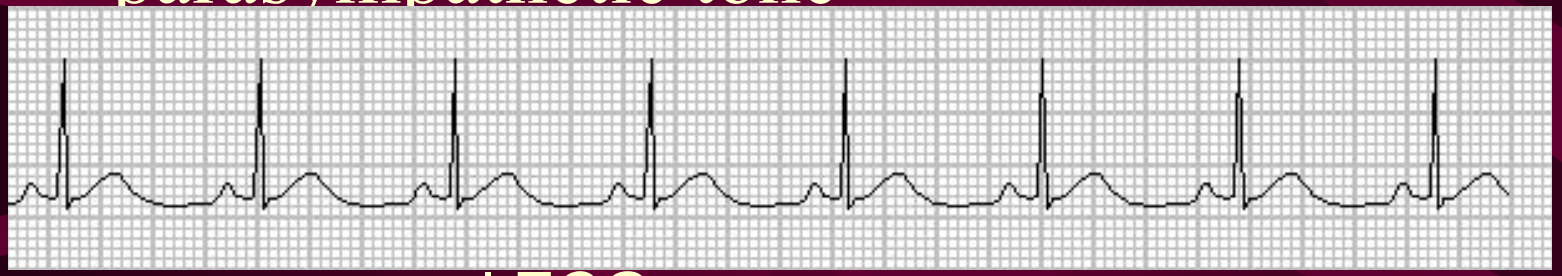
normal ECG



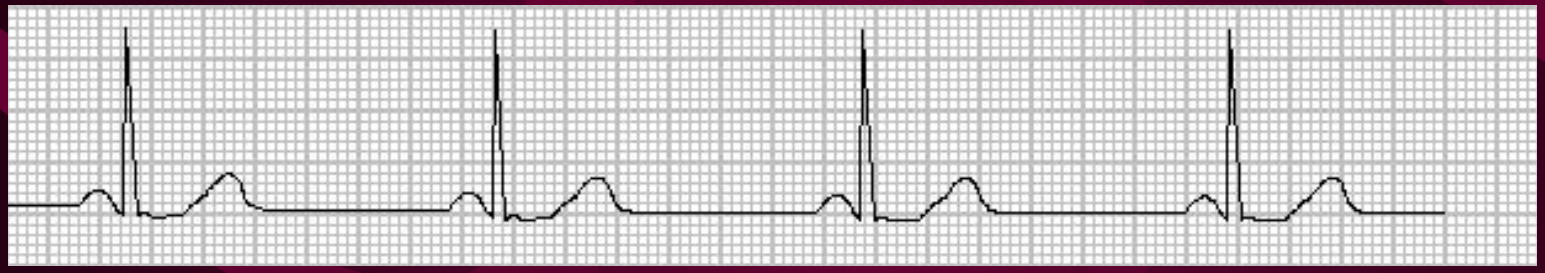
sinus tachycardia (shortened RR or TP interval)

Pathology of automatism

- Sinus bradycardia – less than 60 bpm due to decreased sympathetic and increased parasympathetic tone



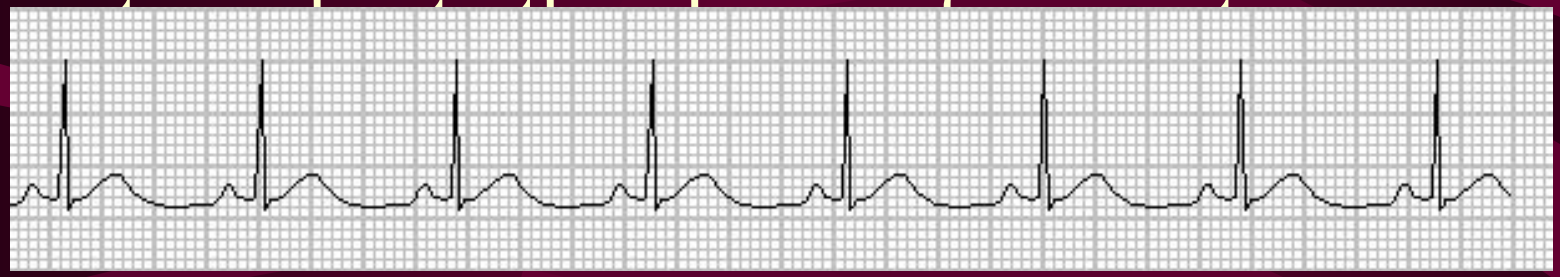
normal ECG



sinus bradycardia (increased RR or TP interval)

Pathology of automatism

- Sinus arrhythmia fluctuation of the vagal



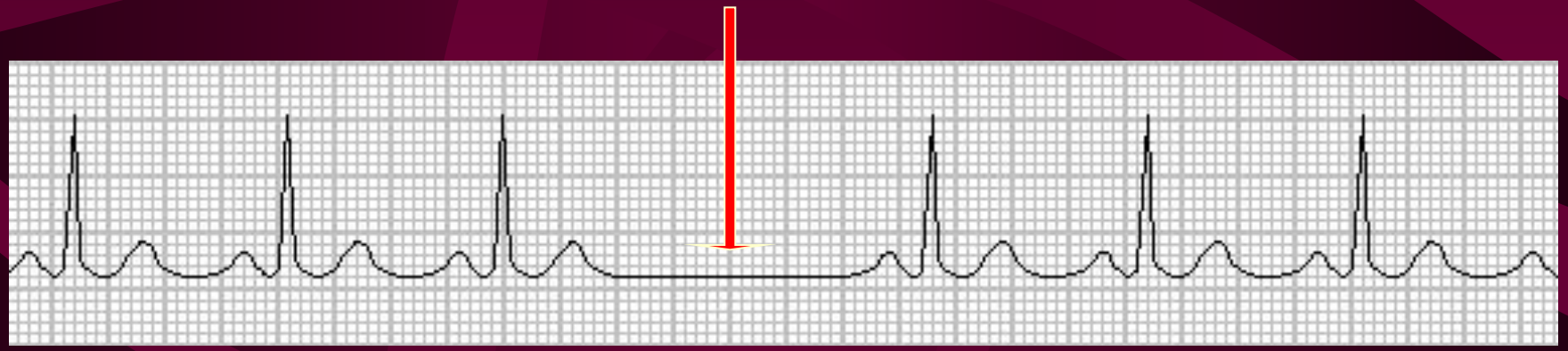
normal ECG

Expiration

Inspiration

Conduction abnormalities

- Sino-atrial block is characterized by long intervals between consecutive P-waves.
- Reason - ischemia or infarction of the SA node.



Atrioventricular block

- Atrioventricular block is the blockage of the conduction from the atria to the AV-node. Three degrees of AV block are known.
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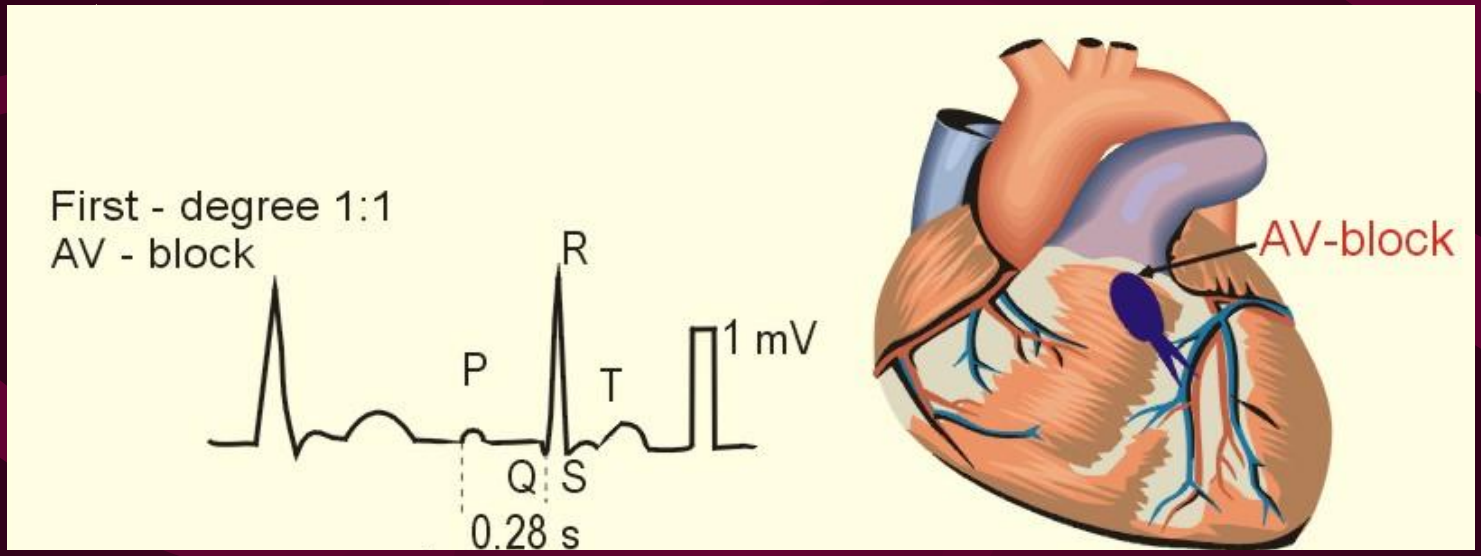
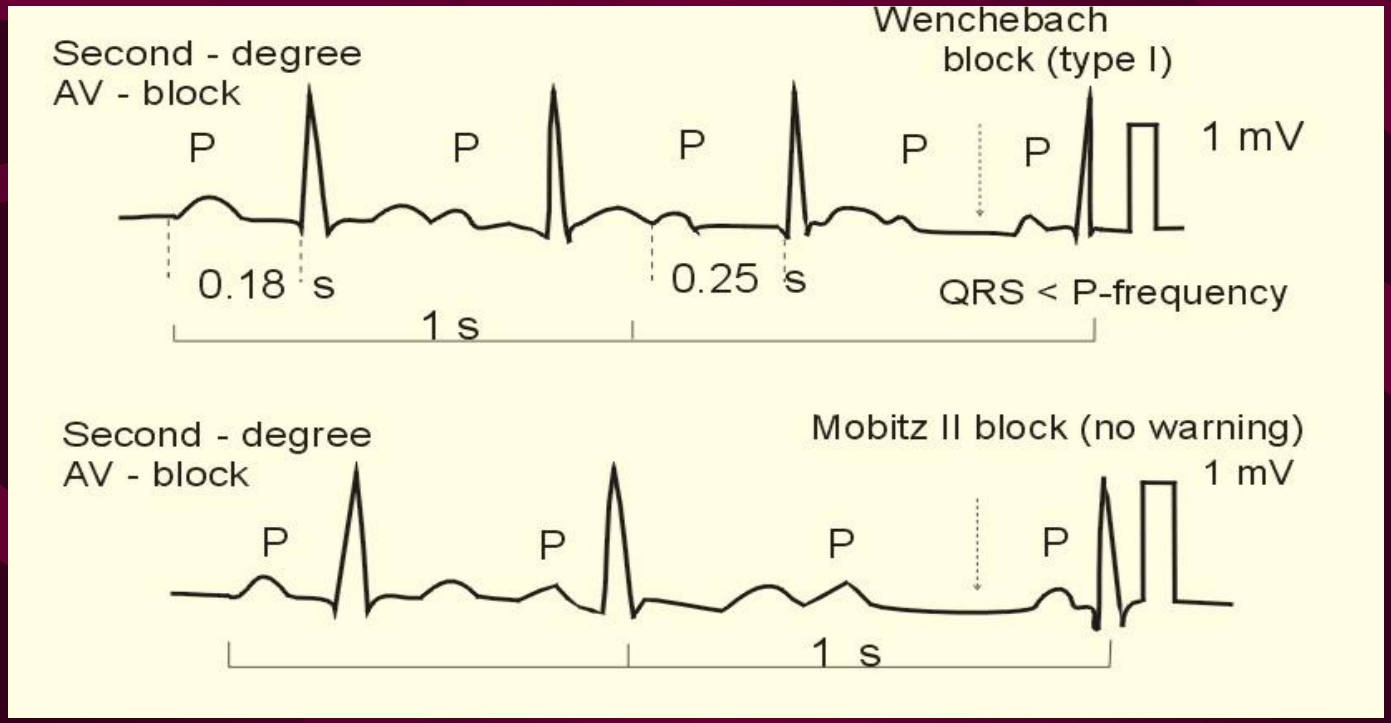


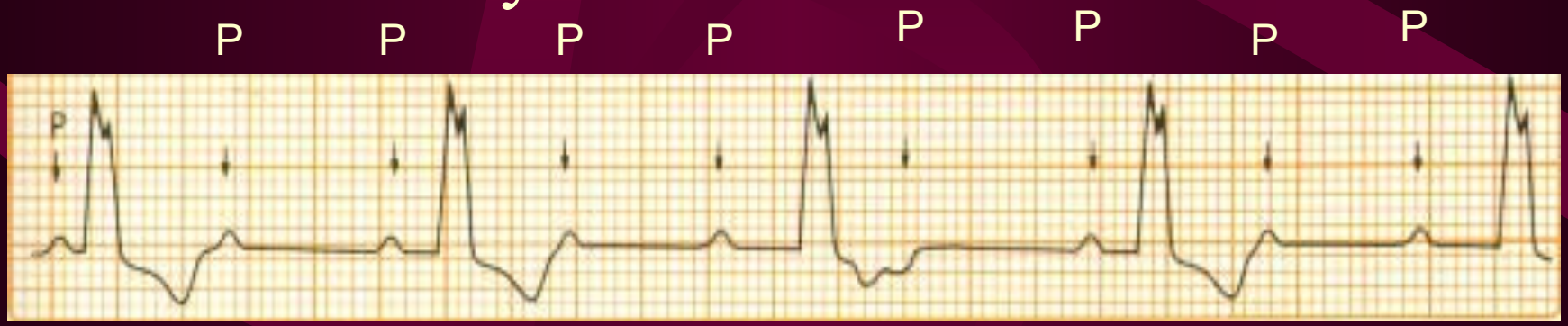
Atrioventricular block

- 2nd degree AV block- some of the P-waves are not followed by QRS-complexes
- Mobitz type I - PQ-interval is increased progressively until a P-wave is not followed by a QRS-complex. (Wenchebach block).
- Mobitz type II block - the ventricles drop some beats



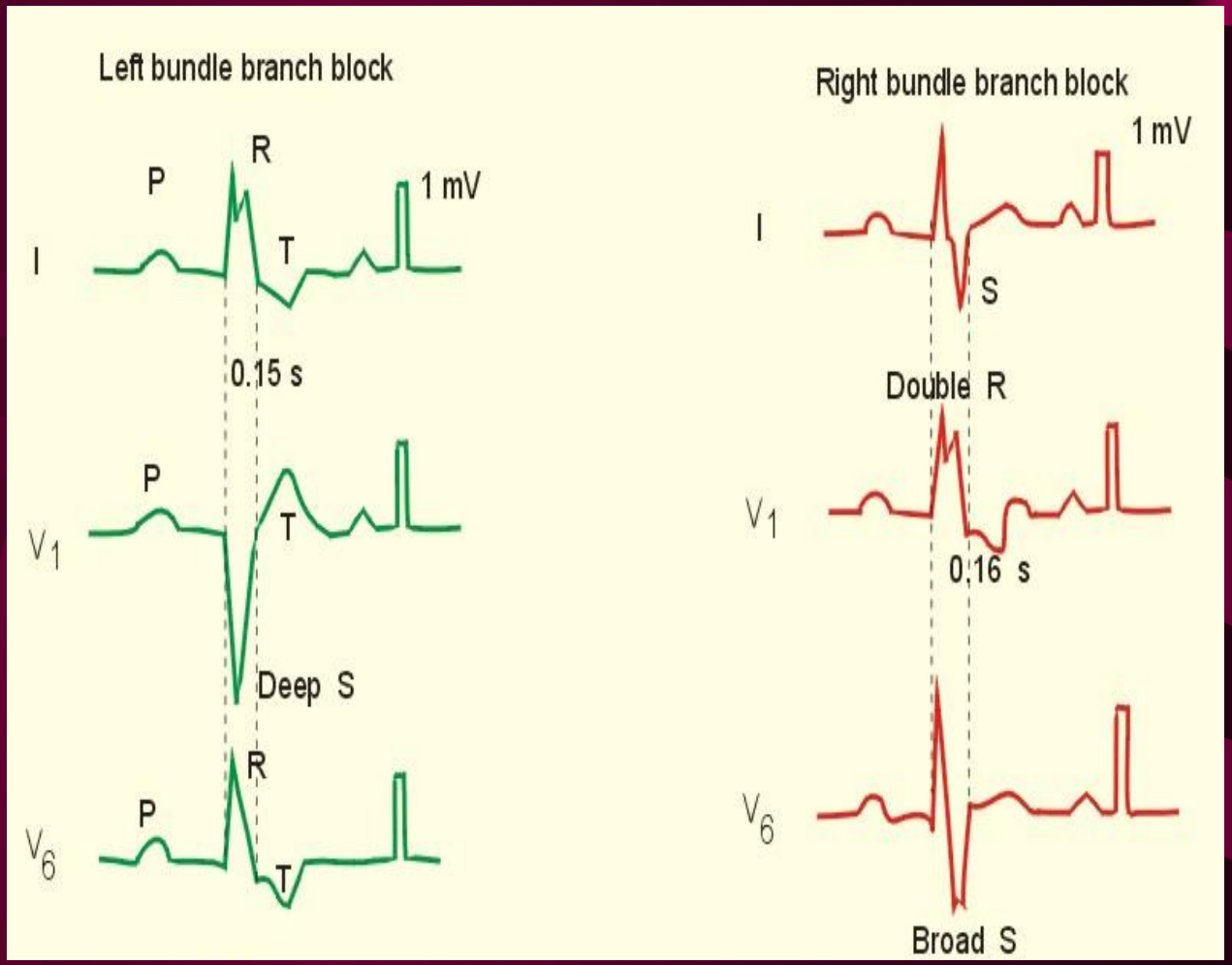
Atrioventricular block

- 3rd degree AV block (complete AV-block) is a total block of the conduction between the SN and the ventricles.
- Atriums are regulated by SA node, ventricles by AV node



Bundle branch block

- Bundle branch block is a block of the right or the left His bundle branches
- QRS-complex becomes wider than normal (more than 0.12 s).
- The signal is conducted first through the healthy branch and then it is distributed to the damaged side.

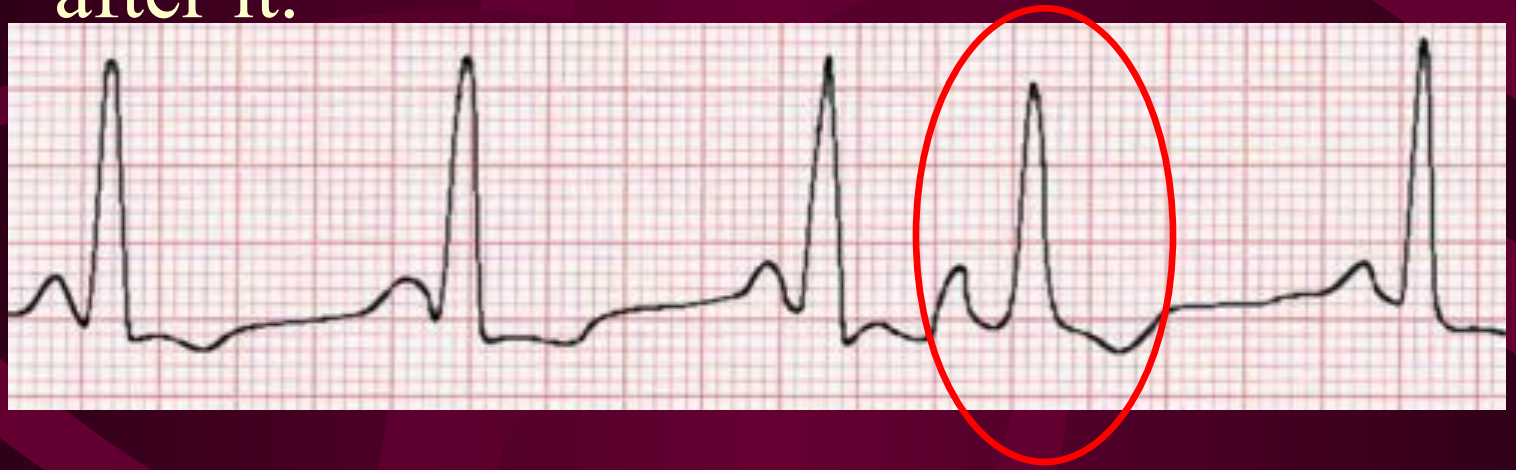


Pathology of excitability

- Pathology of excitability is usually manifested with ectopic beats (outside the sinus node).
  - extrasystole (premature contraction, ectopic beat)
  - paroxysmal tachycardia
  - fibrillation.
- Reasons: ischaemia, mechanical or chemical stimuli, metabolic disturbances..

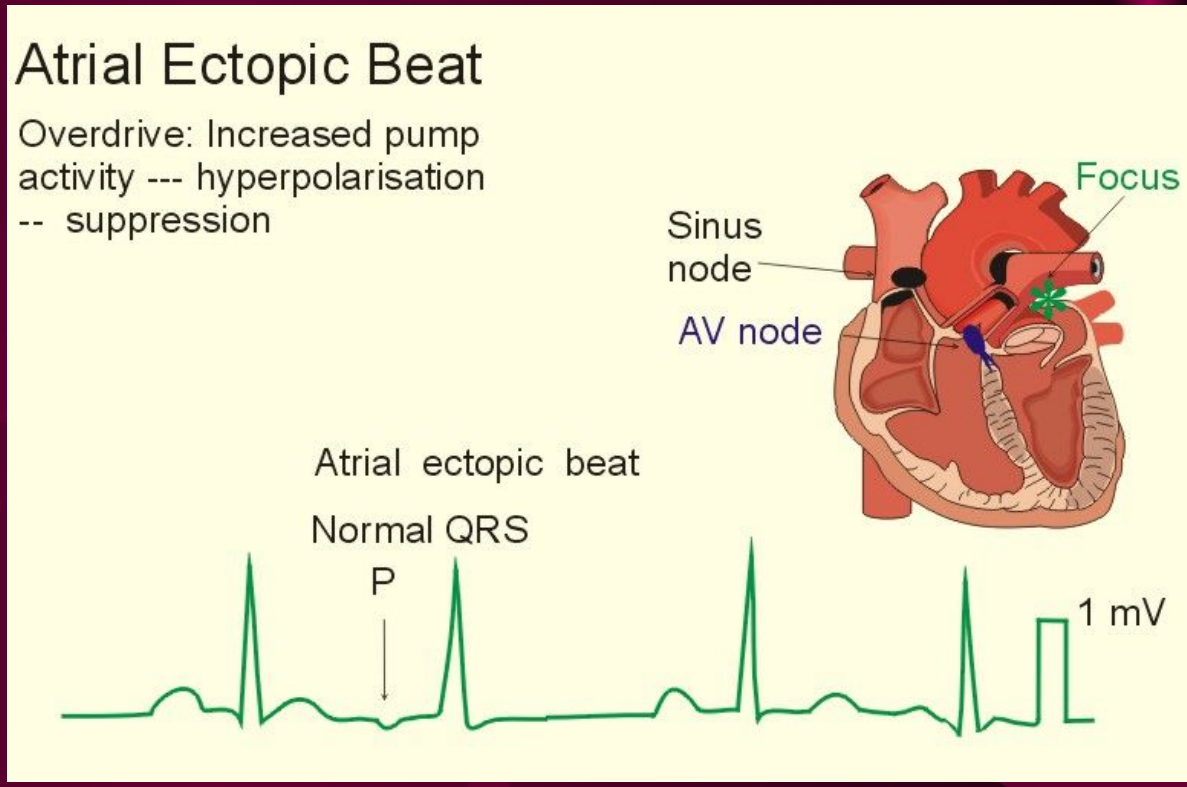
Sinus extrasystole

- **Sinus extrasystole** originates in the normal pacemaker – SA node. ECG picture is normal, there is no compensatory interval after it.



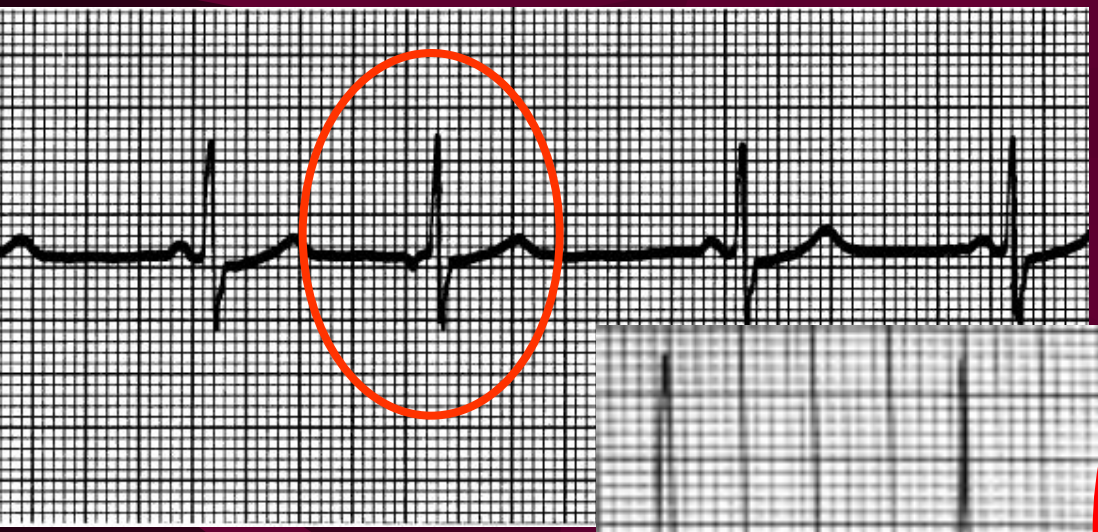
Atrial ectopic beat

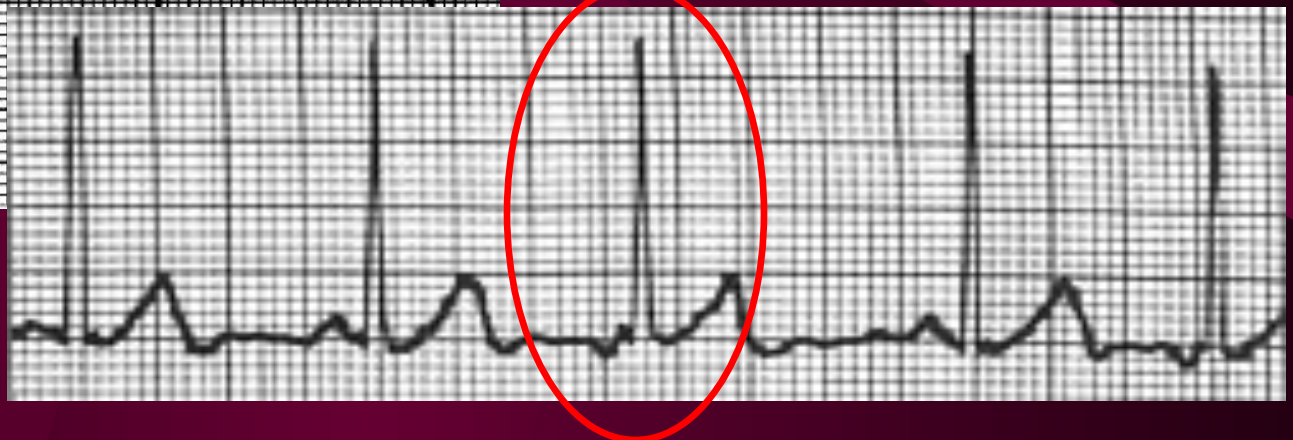
- Atrial ectopic beats have abnormal P-waves and are usually followed by normal QRS-complexes.
- Short compensatory interval is following the premature beat.
- Ectopic beat is weak
- Post-extrasystolic contraction is strong.



Premature junctional contractions

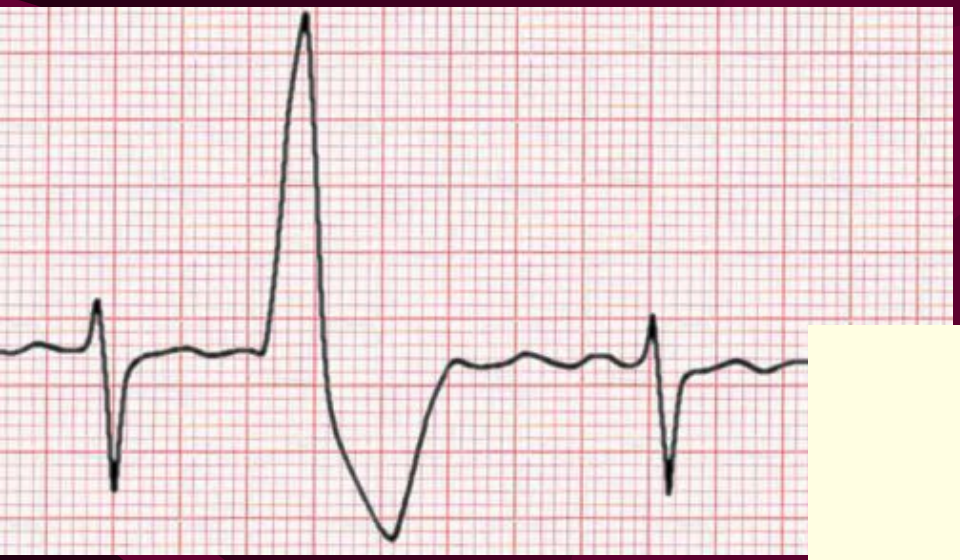
- Ectopic beat originate in the atrio-ventricular node.
- P-wave is negative
- Compensatory interval a less longer than after premature atrial contraction

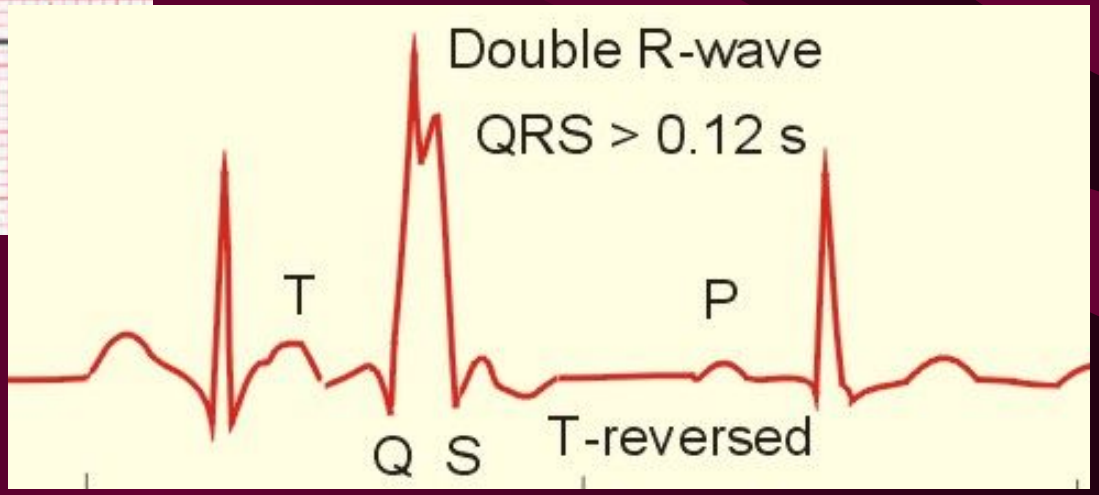




Ventricular ectopic beat

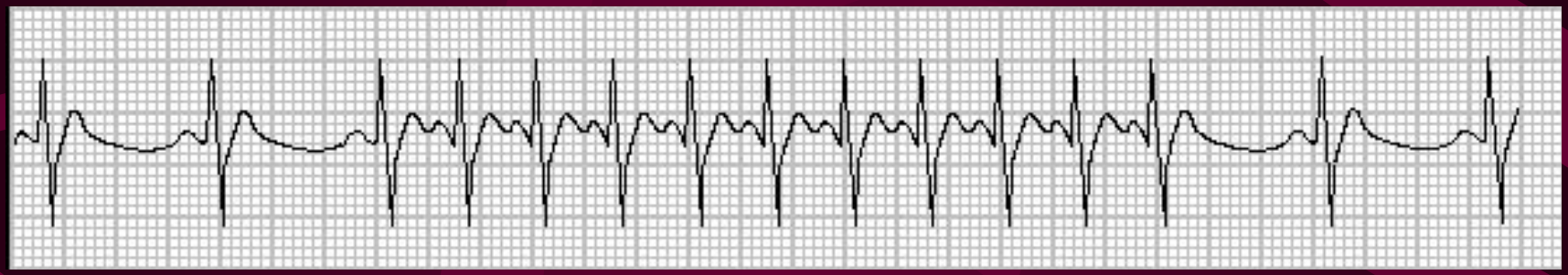
- wide QRS-complex (above 0.12 s),
- long compensatory interval (2RR)





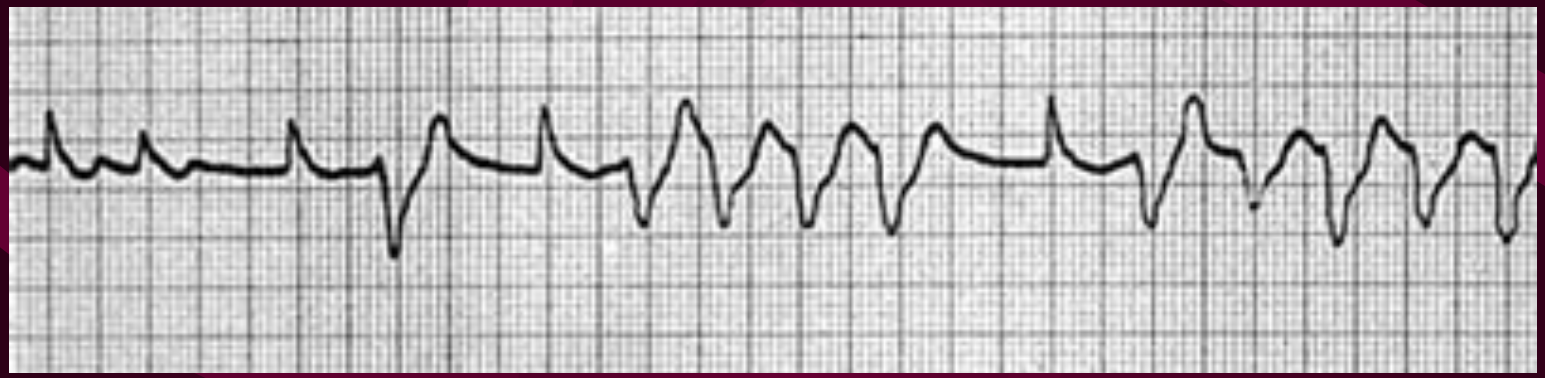
Paroxysmal ectopic tachycardia

- **Paroxysmal atrial tachycardia** is elicited in the atrial tissue outside the SA node as an atrial frequency around 200 bpm.



Paroxysmal ectopic tachycardia

- **Paroxysmal ventricular tachycardia** ≤ 120 bpm
- P-waves are absent
- QRS-complexes are wide and irregular.

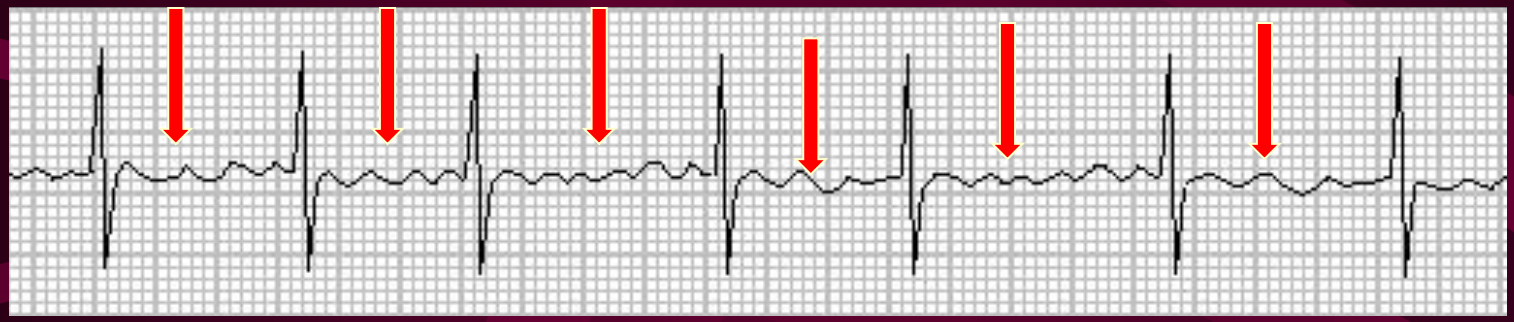


Disorders of hemodynamic in the pathology of excitability

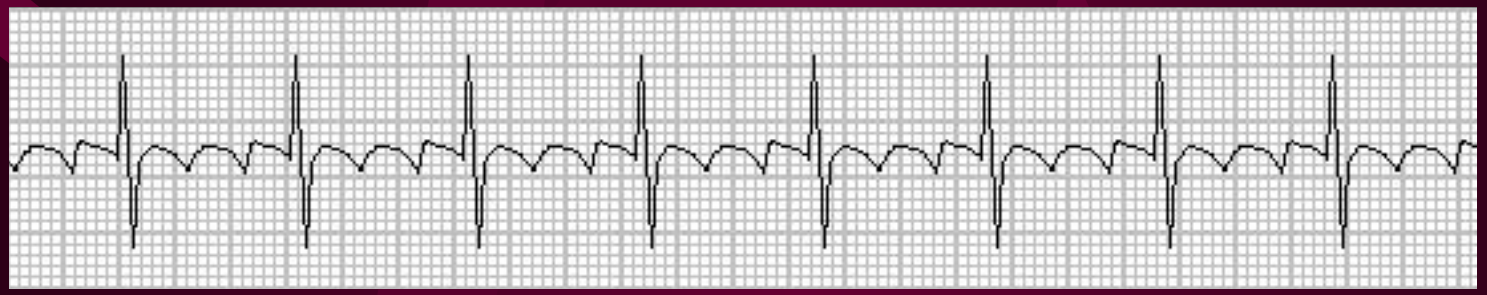
- Single extrasystole clinically manifests in the feeling of «interruption» of cardiac activity.
- Plural extrasystoles can seriously violate the hemodynamic:
  - extrasystoles appear in different phases of cardiac cycle - so they are ineffective in hemodynamic
  - Myocardium can't react to the normal impulse during compensatory pause following extrasystole

Atrial fibrillation and flutter

- Atrial fibrillation - more than 400 P-waves per min , QRS-frequency of 150-180 bpm, f-waves

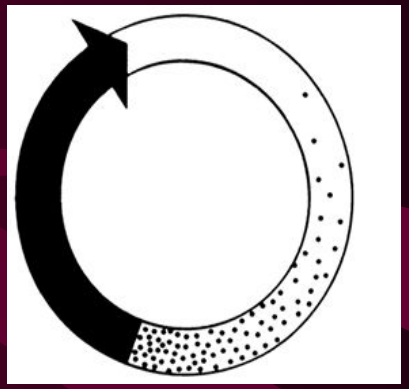


- Atrial flutter atrial frequency is about 300 bpm, sawtooth-like P-waves



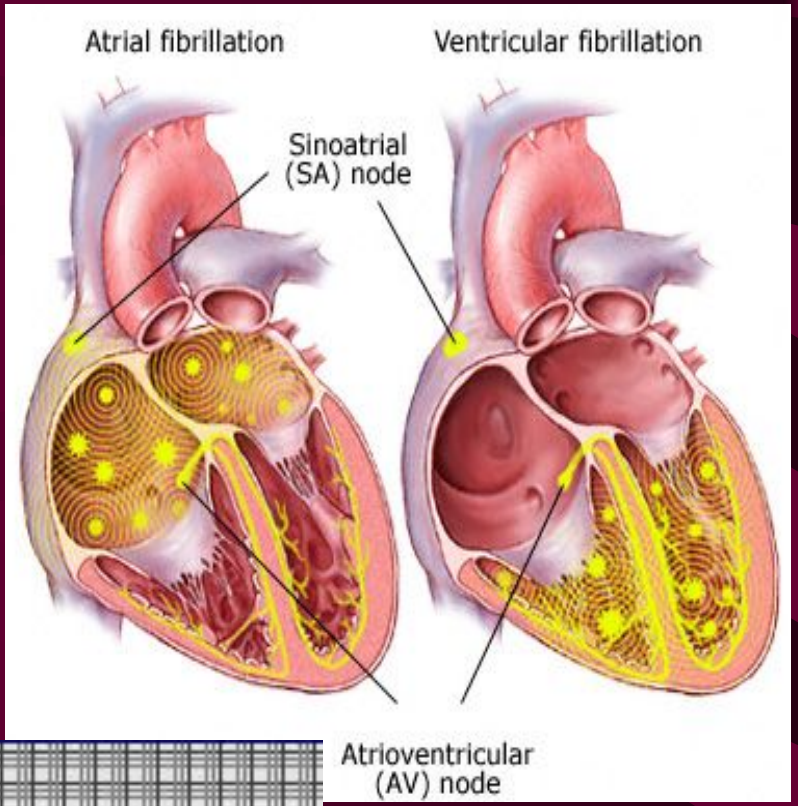
Reasons of atrial fibrillation

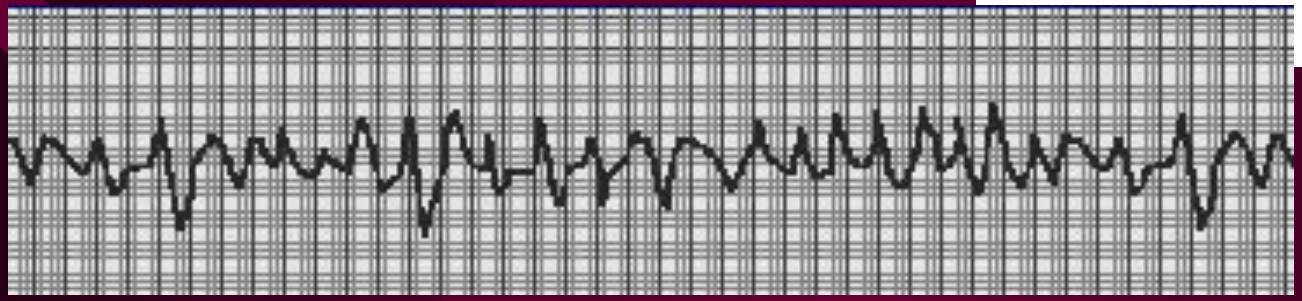
- **Re-entry phenomenon** - cardiac impulse travel around in cardiac muscle without stopping .
  - Dilatation of the heart - long impulse pathway in cardiac muscle.
  - Decreased velocity of impulse conduction (ischemia, high blood K level).
  - Shortened refractory period of the muscle (epinephrine injection or following repetitive electrical stimulation).



Ventricular fibrillation

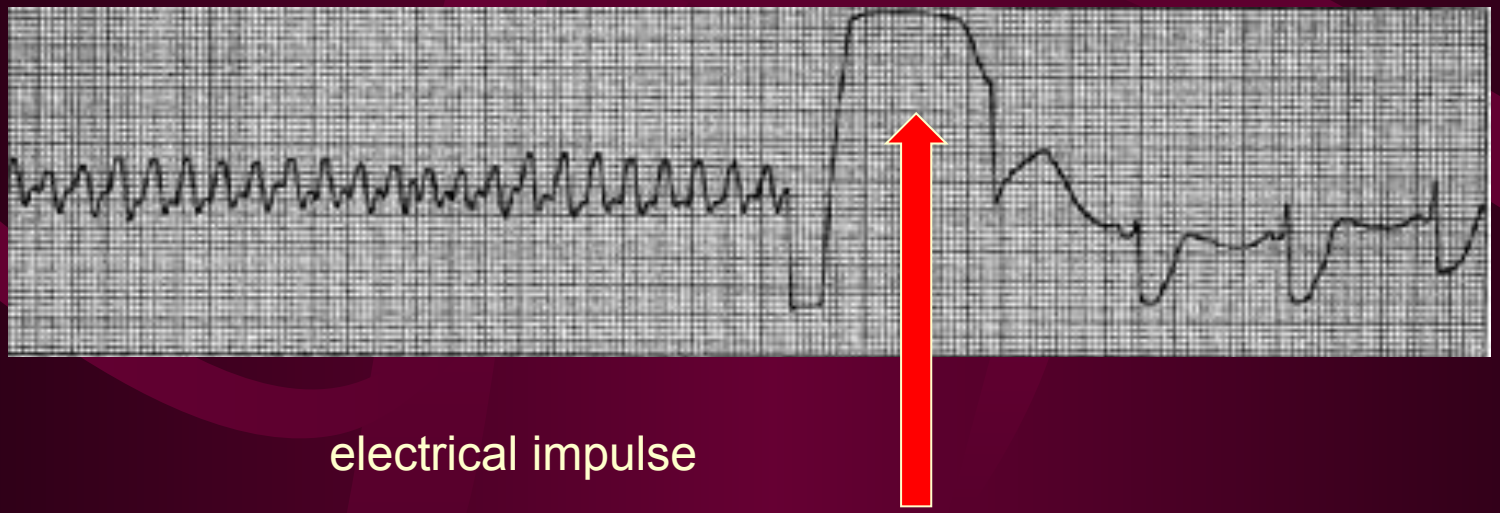
- **Ventricular fibrillation** irregular ventricular rate is 200-600 twitches/min.
- The heart **does not pump blood**.
- It leads to unconsciousness within 5 seconds.
- The trigger is anoxia.





Defibrillation of the heart

- Defibrillation – brings a maximum greater number of cardiomyocytes to one stable state – the phase of absolute refracterity. It will provide subsequent renewal of the cardiac rhythm if SA node is normally functioning.



Pathology of contractility

- Pulsus alternans – alternation of strong and weak pulse pressures during a sinus rhythm.
- Reasons: congenital heart diseases, cardiomyopathy, pericarditis, cardiac failure.