1. Noninvasive and invasive cardiovascular laboratory explorations.
2. Biological evaluation: laboratory tests, serum biomarkers (markers myocyte’s injury, inflammation, oxidative stress, neuro-hormones, myocyte’s stress, extracellular matrix remodeling).
4. ECG signal amplification and mediation. Indications
5. Prolonged ambulatory ECG monitoring (Holter). Indications.
7. Exercise ECG test. Indications and contraindications.
13. Inclined table test in the diagnosis of syncope.
16. Cardiac CT. Coronary CT angiography with contrast. Cardiac CT evaluation of cardiac chambers, pericardium and great vessels.
17. Cardiac MRI, coronary and great vessels.
19. Aortic stenosis. Etiology. Pathophysiology (effect of pressure overload on LV geometry, systolic and diastolic LV function, myocardial ischaemia)
22. Mitral stenosis. Diagnosis (clinical, laboratory investigations). Pharmacological treatment, medication, surgical and percutaneous mitral balloon.
24. Mitral regurgitation. Diagnosis (clinical, laboratory investigations). Pharmacological treatment, medication, surgical and cardiac resynchronization therapy.
34. Diagnosis of hypertension. Measurement of blood pressure. HTA of "white coat". Ambulatory hypertension or "masked".
35. Hypertension. Physical examination and laboratory investigations. Other test results.
37. Non-pharmacological treatment of hypertension.
41. Hypertension in special situations. Resistant hypertension. Treatment.
43. Accelerated-malignant hypertension with papilloedema.
44. Classical cardiovascular risk factors (age, sex, heredity, obesity, sedentary lifestyle, smoking, alcohol, hypertension, DLP) and recently described (homocysteine, lipoprotein (a), proinflammatory factors, prothrombinic factors). CV risk scores.
46. Atherosclerosis. Definition. Pathogenesis: initiation process, the progression of lesions, the role of inflammation and arterial calcification, plaque vulnerability. Clinical manifestations.
47. Dyslipidemia. Definition and terminology. Metabolism and transportation of lipoproteins (LDL-C, HDL-C, VLDL-C, non HDL-C, apolipoproteins, lipoprotein (a)).
49. Dyslipidemia as a risk factor for coronary heart disease.
50. Classification of hyperlipoproteinemias. Primary and secondary hyperlipoproteinemias.
52. Treatment of hyperlipoproteinaemias non medicated and medicated. Prophylaxis of hyperlipidemia.
Stable angina of effort. Classification. Diagnosis: clinical and laboratory investigations.


Ischemic syndromes (myocardial preconditioning, stunned and hibernating myocardium).


Acute coronary syndrome. Unstable angina and MI without ST segment elevation. Treatment (general measures, medication, interventional and surgical). Recovering unstable angina.

Acute myocardial infarction with ST-segment elevation. Criteria for definition of MI (detection increase and / or decrease biomarkers of myocardial necrosis, myocardial ischemic symptoms, ECG changes suggestive of ischemia new appearance of pathological Q wave on the ECG, imaging evidence of a recent loss of viable myocardium and so on). Pathogenesis (Pathology, changes in the VS).

Acute myocardial infarction with ST-segment elevation. Diagnosis (clinical, ECG, assessment of serum biomarkers and other biological samples, imaging).

Acute myocardial infarction with ST-segment elevation. Medical treatment during pre-hospital and hospital immediately (pain, oxygen, antianginal therapy, antiplatelet and anticoagulant).

Acute myocardial infarction with ST-segment elevation. Myocardial reperfusion therapy (coronary angioplasty, fibrinolytic therapy, antithrombotic therapy associated with reperfusion therapy treatment without reperfusion therapy).

Acute myocardial infarction with ST-segment elevation. Routine pharmacological treatment in the acute phase. Surgical revascularization.


Mechanical complications of AMI.

AMI complications. Arrhythmias and conduction disturbances.

AMI complications. Consequences of post-MI LV remodeling. Other complications.


Chronic heart failure. NYHA functional classification. Classification ACC / AHA based on structural abnormalities. Possible etiologies and mechanisms dominant (pressure and volume overload, decreased contractile efficiency and cardiac filling).

Contributing factors and precipitants of heart failure (or arrhythmia management, infectious disease, hypertensive crisis, myocardial ischemia, pulmonary embolism, anemia, hypoxemia of various etiologies, endocrine disorders, hyperkinetic states, noncompliant referrals, use of drugs with adverse effects).

Important pathophysiological mechanism in chronic heart failure: cardiac abnormalities (structural and functional) and neurohumoral (RAAS, SNS, bradykinin, NO, PG, BNP, endothelin, IL, TNF-α). LV remodeling. Mitral regurgitation. Arrhythmias and left bundle branch block.

Diagnosis of chronic heart failure (clinical, paraclinic investigations - ECG, imaging, laboratory).


Hipodiastolic heart failure and preserved LV ejection fraction.
82. Treatment of chronic heart failure. TREATMENT interventional (cardiac resynchronization therapy, implantable cardiac defibrillators), surgery (myocardial revascularization, ventricular reconstruction and mitral valve, heart transplantation).
84. Acute heart failure. Path physiology and forms of presentation of ICA (decompensate chronic HF / worse, EPA, IC hypertensive cardiogenic shock, isolated right IC, IC associated with ACS, increased cardiac output IC).
85. Acute heart failure. Diagnosis: clinical manifestation (symptoms and signs) and laboratory investigations (ECG, imaging tests, laboratory, blood gas meter and so on).
86. Treatment of acute heart failure. Treatment goals (TA, hypervolaemia, renal function, and so on). Ventilation techniques (oxygen, noninvasive ventilation, intubations and mechanical orotracheal ventilation).
89. Special cases of acute heart failure. Cardiogenic pulmonary edema. Clinical picture, laboratory tests (ECG, chest X-ray, EcoCG, gas meter pressure, blood counts, the BNP or NT-pro BNP plasma). Treatment.
98. Myocarditis. Treatment. Recognized certain therapies (heart failure, arrhythmias, and NSAIDs). Controversial therapies possibly additional (immunomodulatory, antiviral, etc.). Evolution and prognosis


108. Aritmogenesis. Physiological bases of cardiac electrical activity. Automatism or chronotropism. Conductivity or dromotropism. Excitability or batmotropism. Resting membrane potential. Transmembrane transport of ions and ion selective channels. The action potential of fast or slow type and respective phases.

109. Aritmogenesis mechanisms (abnormal impulse formation activity triggered - trigger, reentry).


113. Treatment and prevention of paroxysms of supraventricular tachycardia.


117. Ventricular arrhythmias. Classification. Particular form of ventricular tachycardia (monomorphic ventricular tachycardia heart normally reentrant branch block, form continuous bidirectional polymorphic, torsade de pointes, accelerated idioventricular rhythm).

118. Flutter and ventricular fibrillation. ECG diagnosis. Treatment.


120. Atrioventricular conduction disturbances. Atrioventricular block grade I, II and III.

121. Intraventricular conduction disturbances. Left bundle branch block.

122. Intraventricular conduction disturbances. Right bundle branch block.

123. Intraventricular conduction disturbances. Left bundle branch blocks anterior and posterior.

125. Antiarrhythmic drugs commonly administered to treat supraventricular tachyarrhythmias (adenosine, verapamil, diltiazem, esmolol, ibutilide, dofetilide, digoxin).

126. Antiarrhythmic agents commonly administered for the treatment of ventricular arrhythmias (lidocaine, mexiletine, dizopiramida, procainamide).

127. Antiarrhythmic therapy administered both supraventricular tachyarrhythmias and of the ventricular (quinidine, propafenone, flecainide, sotalol, amiodarone).
