• **Hypertension**

Symptoms
• Hypertension evolves most of the time quietly, being accidentally discovered by measuring BP.
Suggestive symptoms for the disease:
• headache, dizziness, visual disturbances, motor or sensory disorders, AIT, palpitations, precordial pain, dyspnea, thirst, polyuria, nocturia, hematuria, intermittent claudication, cold extremities or the presence of symptoms suggestive of secondary hypertension.

History:
• data on period of hypertension,
• the values reached,
• the degree of control of the hypertension,
• symptoms suggestive of secondary hypertension ,
• lifestyle, weight, BMI, consumption of saturated fat, salt, alcohol, fruits and vegetables, physical activity,
• the presence of sleep apnea,
• personality type,
• drug use (nasal drops, nonsteroidal and steroidal anti-inflammatory drugs, oral contraceptives, amphetamines, cocaine, erythropoietin),
• pathological history and associated coronary, cerebrovascular, peripheral vascular, renal, diabetes mellitus, dyslipidemia, asthma,
• heredocolateral history (kidney disease, cardiovascular disease, hypertension , dyslipidemia).

Physical examination:
• correct measurement of BP,
• search for suggestive signs for secondary hypertension : obesity, neurofibromatosis (pheochromocytoma), palpable (polycystic) kidneys, abdominal breath (renovascular hypertension), absence or decreased pulse in the femur, auscultation of the heart and large vessels.

Cardiac examination
• elements of cardiac hypertrophy, the presence of arrhythmias, gallop, pulmonary rales, peripheral edema.

Peripheral artery examination
• absence or diminution of the pulse, breaths, skin lesions, cold extremities.

Brain response
• detection of carotid breath, motor / sensory deficits, cognitive disorders,
• The examination of the eyelid is suggestive when papillary edema, exudates, bleeding appear.

Determination of BMI and type of obesity
• patients with abdominal obesity (abdominal circumference> 88 cm in women and> 102 cm in men) or those with metabolic syndrome are considered at cardiovascular risk.

Paraclinical investigations:
• hemoglobin, hematocrit,
• fasting blood glucose and glucose tolerance test (when fasting blood sugar exceeds 110 mg / dl),
• HDL lipid profile, LDL cholesterol, total cholesterol, triglycerides,
• serum creatinine, uric acid, serum potassium,
• urine summary, microalbuminuria, proteinuria.

Further investigations:
• ECG, echocardiography, carotid and femoral ultrasonography, ankle-arm index determination, highly sensitive C-reactive protein.

Secondary hypertension
• Secondary hypertension should be suspected by the family physician and sent to the specialist doctor for further paraclinic examination.
• The following signs and symptoms may raise the suspicion of a secondary hypertension:
• palpable abdominal mass,
• family history,
• modification of functional renal tests, hematuria, leukocyturia, proteinuria (polycystic kidney);
• hypertension difficult to treat, progression of renal impairment, hypopotassemia, difference> 1.5 cm between the two kidneys may suggest renovascular hypertension through renal artery stenosis or fibromuscular dysplasia;
• paroxysmal attacks of hypertension accompanied by sweating, pallor, headache, palpitations (pheochromocytoma);
• hypertension in childhood, resistance to treatment, hypokaliemia (primary hyperaldosteronism);
• characteristic facies, trunkular obesity, stretch marks (Cushing sd);
• hypertension in young people or children, anterior and posterior thoracic bleeding, absent of femural pulse or high BP in the upper limbs and low values in the lower limb (aortic coarctation),
• sleep apnea - frequent in the obese, daytime sleepiness, irritability, concentration disorders, decreased libido, night coughing,

Evaluation of the hypertensive patient:
• 1. Correct measurement of blood pressure;
• 2. exclusion of a secondary cause of hypertension ;
• 3. establishing the cardiovascular risk of the patient according to the level of systolic and diastolic BP; pulse pressure level (in the elderly), presence and number of cardiovascular risk factors

• 4. subclinical organ changes
- a) large heart and vessels: hypertrophy. Left ventricular (ECG- Sokolow-Lyon index> 38
- mm, echocardiographyI highlighting the thickening of the arterial wall or atherosclerosis plaques in the large arteries, carotid, femoral, measuring an ankle / arm index <0.9
- b) at the renal level: slight increase in plasma creatinine (1.3-1.5 mg / dl in men; 1.2-1.4 mg / dl in women), decreased glomerular filtration <60 ml / min / 1 , 73 mp, or creatinine Cl <60 ml / min, microalbuminuria 30-300 mg / 24 h
- c) at brain level: testing of cognitive function, CT and MRI (silent lacunar infarctions, micro-bleeding, white matter hyperreflective);
• 5. establishing the associated clinical conditions:
- a) cerebrovascular: cerebrovascular accident, cerebral hemorrhage, transient ischemia attack, at the bottom of the eyes we can identify advanced retinopathy - hemorrhages, exudates, papillary edema
- b) cardiac: AMI, angina pectoris, heart failure, reperfusion disorders, coronary revascularization, clinically manifest peripheral vascular disease
- c) renal: creatinine increase> 1.4 mg / dl in women and> 1.5 mg / dl in men, creatinine C decrease <30 ml / min, proteinuria> 300 mg / 24 hours;
• 6. assessment of cardiovascular risk according to the diagrams and establishing the therapeutic strategy and the patient follow-up plan.

• The peculiarities of antihypertensive therapy depending on the pathology:

1. Elderly patient
• - the decrease of BP will be done gradually, because there is an increased risk of orthostatic hypotension; therefore, BP measurement will also be done in clinostatism and orthostatisn:
• Many elderly people have target organ damage or other conditions that must be taken into account when choosing the hypotensive agent.
• It is recommended to lower the BP to the target values (140/90-mmHg) up to 80 years; over 80 years there is no evidence that lowering BPs to target values would benefit mortality, but lower cardiovascular events.
• The most commonly used hypotensive agents are: thiazide diuretics and calcium blockers;

2. hypertension in young people
• - requires first of all non-pharmacological measures
• BP should be lower than 140/90 mmHg, and if there are other associated cardiovascular risk factors, cautious pharmacological treatment will also be initiated.
• A particular aspect is the use of contraceptives in women and male anabolic steroids, which can cause the appearance of hypertension in young people, reversible upon stopping the use of these substances.
• New therapeutic agents will be used to prevent sexual dysfunction (angiotensin receptor blockers, IECs, calcium antagonists, vasodilating blockers)

3. hypertension in pregnancy
• BP over 160/140 mmHg will be compulsorily treated
• For BP over 140/90 mmHg, early pharmacological treatment will be initiated if:
• there is gestational tension with proteinuria
• the management pension is superimposed on the pre-existing hypertension with symptomatic impairment of target organs
• Medications that interfere with the renin angiotensin aldosterone system are contraindicated
• Diuretics reduce plasma volume
• Beta blockers cause fetal growth retardation at the beginning of pregnancy
• Hypertension agents of choice in pregnancy: methyldopa, labetalol
• In eclampsia: sodium nitroprusside, intravenous nitroglycerin

4. hypertension in diabetics
• target values below 130/80 mmHg
• it is recommended to lose weight and decrease the salt intake
• the drug treatment is started even at the values of the normal high BP
• cardiovascular risk is treated
• drug combination is required
• Renin angiotensin blockers are preferred for renovascular protection
• Metabolic neutral drugs are preferred: calcium blockers, IECs, pans
• Thiazide diuretics are avoided
• In the case of impaired glucose tolerance, beta-blockers are avoided because they can mask pre-existing diabetes

5. hypertension in metabolic syndrome
• the first measures consist in changing the lifestyle (diet, weight control, physical exercise).
• calcium channel blockers, IEC, BRAA, in combination with antidiabetic and hypolipidemic medication (statins)
• The use of beta-blockers and thiazide diuretics is avoided due to the increased risk of diabetes;

6. hypertension in patients with cardiovascular disease
• ischemic heart disease: beta blockers, platelet anti-aggregates (aspirin), hypolipemic agents (statins):
• heart failure - IEC, BRAA, beta blockers, diuretics;
• postIM- BB, IEC, BRAA, antialosteronone diuretic;
• paroxysmal atrial fibrillation - IEC, BRAA;
• permanent atrial fibrillation - BB, blockers of the nondihidropyridine calcium channels;

7. hypertension in patients with cerebrovascular disease
• IEC / BRAA + thiazide diuretics (the combination of perindopril + indapamide - the PROGRESS study);
• calcium channel blockers

8. hypertension in patients with chronic kidney disease
• The treatment aims to
- prevention of cardiovascular events,
- delayed renal damage.
• Target values of BP <130/80 mmHg require careful monitoring of renal function
- (proteinuria, creatinine);
• IEC, BRAA, loop diuretics;
• advanced renal impairment - precaution with decreasing doses to IEC, BRAA, avoiding thiazide and antialdosteronone diuretics;
• attention to bilateral renal artery stenosis - contraindicated IEC and BRAA; requires statin and antiplatelet combination;

9. Treatment resistant hypertension
• It represents the situations in which the control of BP is not obtained with non-pharmacological lifestyle change therapy and 3 drugs administered in appropriate doses, one of which is a diuretic.
• Diagnosis of resistant hypertension requires the exclusion of all causes of secondary hypertension .
• Resistant hypertension is associated with high cardiovascular risk of cardiovascular and renal events.
• For the correct diagnosis of resistant hypertension , it is necessary to exclude:
- reduced compliance to treatment,
- non - compliance with lifestyle recommendations,
- sleep apnea,
- consumption of drugs that increase BP (anti-inflammatory, contraceptive).

• The patient will be sent for investigation to the specialist.

Hypertensive emergencies
• symptomatic increase of BP> 220/130 mmHg frequently associated with signs of target organ suffering;
• it is necessary to recognize the situation, to grant the first aid until hospitalization (25% decrease of the BP in the first minutes, then to values of 160/100 mmHg and the patient taking over by a specialized service.

The most common situations are:
• hypertensive encephalopathy (headache, confusion, drowsiness, agitation, vision disorders, seizures, coma);
• menigocerebral hemorrhage (motor deficiency, meningeal signs, altered consciousness);
• eclampsia / preeclamsia;
• acute pulmonary edema (dyspnoea, orthopnea, cough, anxiety, agitation, intense sweating, pale and cold teguments, cyanosis of the extremities and lips);
• AMI;
• aortic dissection;
• severe symptomatic tension jump after: discontinuation of treatment, volemic overload, brain trauma

Hospitalization

• early diagnosis of hypertension , with the need to identify and treat risk factors, organ damage and associated clinical conditions, stratification of cardiovascular risk;
• scheduled visits to the family doctor - initially at 2-4 weeks until the establishment of the chronic treatment, the doses of medicines will be adjusted, the possible adverse reactions, the change of the medication will be considered;
• non-scheduled consultations in emergencies or when intercurrent events occur;
• after stabilizing the BP values, the patients at low risk will be monitored at 6 months; patients will be encouraged to measure BP at home, without overdoing it, which could cause anxiety, and without allowing self-medication, dose adjustment by the patient;
• patients at moderate, high or very high additional risk will be monitored more frequently, every 1-3 months, as appropriate;
• monitoring of target organ damage at 12 months: EKG, ankle-arm index, laboratory, eyelid, albuminuria, cognitive function testing, brain CT as needed;
• Collaboration with the specialist doctor at 6-12 months or as often as the case may be (suspicion of secondary hypertension , when BP control is not obtained after 6 months of correct treatment, sudden recurrence of high BP values, despite treatment);
• advising the patient on the elimination of risk factors, applying non-pharmacological measures, changing the lifestyle;
• the patient must be convinced that the treatment is lifelong and must be actively involved in achieving its objectives (BP control, reduction of risk factors, delay in target organ damage),

Difficulties in monitoring and treating hypertensive patients:

• motivating the need for permanent therapy, especially in asymptomatic patients;
• low adherence to non-pharmacological measures,
• the occurrence of adverse drug reactions,
• lack of information for patients,
• lack of medical information,
• non-adherence to current guidelines,
• maintaining old treatment practices,
• costs that patients cannot afford.

Solutions:

• periodic training of all doctors,
• educating and motivating patients,
• choosing the optimal solutions for each patient, taking into account their age, particularities, their cardiovascular risk, associated comorbidities, but also the economic possibilities,
• establishing a doctor-patient partnership that ensures good compliance.

Ischemic coronary artery disease

• Stable angina

• Typical angina is defined as retrosternal chest pain with the following characteristics:
- of constrictive nature, at retrosternal level, arms, jaw or throat;
- appears in effort;
- gives in to rest or NTG in about 5 minutes.

• Atypical angina has 2 of the 3 characteristics, and non-angina pain has one or none of the three characteristics.

Clinical evaluation:
1. Resting ECG (complete LBB, Q wave, ST / T changes that may indicate an ischemic event);
2. Treatment of CV risk factors;
3. Alternative diagnoses (eg GERD, musculoskeletal disorder);
4. Exclusion of other non-coronary causes of angina, such as severe aortic stenosis or hypertrophic cardiomyopathy;
5. Reassessment of blood tests to identify causes that may exacerbate angina (eg anemia).

• For patients presenting to the family doctor with stable chest pain who has recently started, after an initial clinical evaluation, the probability of a coronary artery disease based on the patient's history, age, sex and risk factors is subsequently determined.

• The pretest probability (PTP) of the existence of the ccm disease: Marian according to symptoms, age, sex, risk factors

Attitude

1. For patients with non-anginal chest pain, or with pretest probability (PTP) of BHF below 10%, no investigation is required;

2. For patients with PTP for BHF between 10 and 60%, the stress test (stress ECG, stress scintigraphy, stress echocardiography on dobutamine) is used to stratify the patient's risk. In addition, these patients should be treated for stable angina. For patients who cannot perform the stress ECG test, the myocardial infusion test or the stress echocardiography on dobutamine is used;

3. For patients at risk of BHF over 60%, they are directly eligible for diagnostic angiography.

Treatment
1. All patients are advised for the treatment of SA and for the measures to be taken in the case of prolonged angina that does not give up on NTG;

2. All patients suspected of having stable angina receive Aspirin, Statin, and beta-blocker (or calcium blocker if beta-blocker is contraindicated) and are advised to wear NTG to them;

3. Patients with a positive stress test are scheduled for elective angiography;

4. Patients with non-coronary pain are investigated for other conditions.

Is recommended:
• Aspirin daily 75 mg / day, as a platelet antiaggregant;
• Conversion enzyme inhibitors: Ramipril 2.5-10 mg / day, Perindopril 2.5-10 mg / day, Lisinopril 5-10 mg / day. In patients with ACE inhibitors intolerance (eg cough), angiotensin receptor blockers of the type Valsartan 50-100 mg / day, Candesartan 4-32 mg / day, Losartan 50-100 mg / day are administered;
• Statins: Simvastatin (10-40 mg / day, evening before bed), Atorvastatin (10-40 mg / day, evening, in patients with AS and diabetes), Rosuvastatin (5-20 mg / day, evening).
• Beta-blockers: bisoprolol (2.5-10 mg x 1 / day, preferably in the morning), carvedilol (3.125-25 mg x 2 / day), metoprolol (25-50 mg x 2-3 / day)
• Changing lifestyle

• Acute coronary syndromes

Acute coronary syndromes include:
• AMI with ST segment elevation (STEMI),
• AMI without ST segment elevation (NSTEMI)
• unstable angina

• Unstable angina

• Angina is understood to mean central, retrosternal or anterior chest pain, irradiated in the arms (more commonly in the left); at the base of the neck or lower jaw, described as a pressure, pressure, weight, sometimes as a jaw, constriction (claw), caused by myocardial ischemia.

• Pain / discomfort may be localized and epigastric and may be irradiated superiorly, and some patients may experience discomfort in one or more of these areas.

• Pain may be completely absent, being replaced by nonspecific symptoms such as dyspnoea (in old diabetic patients), palpitations, sweating, lipotomy, dizziness, anxiety, nausea and vomiting.

• Unstable angina is diagnosed on the basis of painful symptomatology and ischemic ECG changes (ST segment sub-leveling or T-wave negation), but without detection of myocardial necrosis series biomarkers.

Several forms of unstable angina are described:

1. transformation of stress angina over several days, with increasing frequency of painful episodes occurring at increasingly smaller efforts ("crescendo type angina");

2. newly installed angina, with episodes of angina appearing recurrently and unexpectedly unrelated to physical exertion (de novo);

3. angina post-IM.

These episodes may be relatively short (several minutes) and may subside spontaneously or after sublingual NTG (nitroglycerin) administration, before reappearing after a few minutes or hours.

An episode of prolonged effortless chest pain that does not relinquish / partially give up on sublingual NTG (nitroglycerin) raises the suspicion of an AMI, which necessarily involves presenting the patient to a UPU / cardiology ward.

• ECG in unstable angina:
(1) normal;
(2) acute ischemic changes - ST sub-level;
(3) non-specific changes (eg inversion of the T wave in certain derivatives).
In this case, the clinical symptomatology prevails and the patient will be transported to the UPU / cardiology ward.
Only if serial ECGs and coronary necrosis markers are within normal limits can other causes of chest pain be considered.

ST-segment elevation myocardial infarction (NSTEMI)

• clinical symptoms suggestive of AMI
• suggestive ECG aspect (ST sub-leveling and T wave inversion),
• increased myocardial necrosis markers (cardiac troponin I or T, CK, CK-MB),

Acute myocardial infarction with ST segment elevation (STEMI)

• prolonged retrosternal chest pain, which partially gives up on sublingual NTG administration or does not give up on this treatment and which
• ECG shows ST segment elevation (so-called "smiling face") in the adjacent bypasses, or newly emerged complete left branch block (LBB).
• myocardial necrosis markers (Troponin, CK, CK-MB) are increased

History
• Certain patients (the elderly, diabetics, newly operated on patients) may develop acute coronary syndrome in the absence of pain.
• Angina pain is confused with indigestion by both the patient (who neglects it) and the medical staff.
• Nausea and vomiting may accompany angina and AMI.

Clinical
• Angina pain can be accompanied by sweating, pallor, tachycardia, dizziness, vomiting.

• The value of the clinical examination is high when it identifies a clear cause of chest pain
- intercostal neuralgia, which has a clear localization and a normal ECG.

Investigations

• ECG

• The presence of ST segment changes (over / under-leveling, negating / flattening the T-wave on adjacent derivatives) on the initial ECG frequently confirms the diagnosis, especially if the anomalies are absent on a previous record

• Anteroseptal AMI
- ECG changes in V1-V2 derivatives
• Previous AMI
- ECG changes in V1-V4 bypasses
• Antero-lateral AMI
- ECG modifications in the Vl-V6, aVL derivatives
• are due to occlusion of the left coronary artery (LAD) - or its branches.
• The previous AMI has a poor prognosis, frequently reducing the performance of the left ventricle. in these patients, the greatest benefit is obtained by emergency intervention in the angiography laboratory.
• Subsequent treatment with conversion enzyme inhibitors prevents ischemic remodeling of the left ventricular myocardium and pump failure.

• Lower AMI
- causes changes in the derivatives D2, D3 and aVF,
- results as a result of occlusion of the right coronary artery, or less frequently of the occlusion of the circumflex artery.

• Lateral AMI
- causes changes in the V5-V6 and / or D1 and aVL derivatives (sometimes only in aVL),
- following the occlusion of the circumflex artery or the first diagonal, branch of the LAD.

• Later AMI
- reciprocal changes (in the mirror) in the previous derivations (V1-V3).
- more frequently as a result of lesions in the right coronary artery
- less frequently as a result of the circumflex artery injury (anatomical variant) that irrigates the posterior part of the left ventricle and the interventricular septum.

Laboratory tests - markers of myocardial necrosis
• Cardiac troponins (troponin T and troponin I).
- they grow 3-4 hours after the onset of necrosis
- reach the maximum value at 1-2 days
- it lasts 2 weeks

• Creatine kinase, aspartate aminotransferase (AST) and lacticdehydrogenase (LDH).

• To clarify the origin of the enzyme growth, the CK-MB isoenzyme is dosed, which cuts the diagnosis in an increased percentage of cases.
• AST and LDH increase in AMI, but have a slower dynamic, sometimes useful in retrospective diagnosis of AMI.
• Repeated and sequential dosing of CK may serve to approximate to some extent the affected myocardial mass.

• Management of acute coronary syndromes

• Morphine - titrated intravenously to relieve pain, avoiding sedation and respiratory depression (Diamorphine, 2.5-5 mg, iv);
• Oxygen- is administered in high concentrations and at high volumes of 6-8 1 / min, maintaining as much as possible a SpO2 95-98%;
• Nitroglycerin - is administered sublingually (cp. Or spray or in the ENP) for pain control and coronary vasodilation;
• Aspirin, loading dose - 325-500 mg, chewed or crushed as soon as possible.
• Statin, maximum dose (Atorvastatin 80 mg, po), to stabilize the atheroma plate,
• Other antiaggregant, in the loading dose (Clopidogrel 300-600 mg po, Prasugrel 60 mg po or Ticagrelor 180 mg po),

• Coronary reperfusion therapy. It can be obtained in 3 ways:
1. percutaneous coronary angioplasty (PHF);
2. thrombolytic therapy;
3. The emergency aorto-coronary bypass (CABG).

OBS.
• After 12 hours from the onset of precordial pain, the risks of reperfusion therapy outweigh the benefits, because extensive myocardial necrosis has already been established.

• In-hospital thrombolysis can reduce mortality when the transport time to hospital is greater than 30 minutes or when the time from presentation to hospital to onset of thrombolysis is greater than 30 minutes (door-to-needle time-DTN)

Cardiac rehabilitation.
• A cardiac rehabilitation program should be applied to all patients who have had acute coronary syndrome to speed up their return to normal activity and to act to reduce risks.

Secondary prophylaxis

Anti-thrombotic therapy:
• Low doses of aspirin 75-150 mg / day are effective and have low costs
• for those with aspirin intolerance (allergy, gastro-duodenal disorders), clopidogrel (Plavix) 75 mg / day may be used as an alternative.
Anticoagulant treatment with coumarin derivatives (Acenocumarol- Thrombostop, Sintrom):
• patients with persistent atrial fibrillation secondary to ischemic heart disease and at increased risk of systemic embolism

Reduction of high cholesterol levels.
- Statins reduce the risk of coronary event by about 30%.
- A diet low in fat and high in fiber, as well as regular exercise.
Quitting smoking.
- The definitive cessation of smoking should be made as soon as possible after the beginning of an SCA
- Bupropion, Champix and nicotine patches may be helpful.

Rhythm disorders

• Atrial fibrillation

• In the case of atrial fibrillation, the contraction rate of the atria is 400-600 per minute, which leads to the loss of the atrial pump, but most impulses are blocked at the AV node due to the installation of a variable functional block as a result of the period junction refraction, which leads to a heart rate of 140-160 per minute.

• At a heart rate of 90-100 per minute, the loss of the atrial pump results in a 10-15% decrease in heart rate.
• At higher frequencies, the decrease in heart rate is greater, favoring the occurrence of heart failure.

Clinical

• can be asymptomatic
• palpitations may occur,
• dyspnoea
• angina pectoris.

• Objective
• Auscultation: Heart sounds are arrhythmic,
• irregular peripheral pulse, pulse deficit

ECG
• absence of P waves,
• QRS complexes with preserved morphology
• Variable RR intervals
• the presence of waves of fibrillation (in the right inferior or precordial shunts).

• Atrial fibrillation is paroxysmal if the episode or episodes of fibrillation disappear spontaneously and last from 24 hours to 7 days.
• Most commonly, they disappear after 24 hours.

• Atrial fibrillation is persistent if it lasts longer than 7 days and does not cease spontaneously.

• Atrial fibrillation is chronic if the arrhythmia does not stop spontaneously or by treatment.

Anticoagulant treatment

• Due to the loss of the atrial pump and the risk of systemic embolism, anticoagulant treatment with acenocoumarol or anti-aggregate with aspirin 325 mg daily is required.
• In the case of chronic anticoagulant treatment (Thrombostop or Sintrom), it is important to monitor the anticoagulant effect by measuring the INR to be kept between 2-3
• Caution - New anticoagulants

Heart rate control

• digoxin with an initial loading of 1.5 mg per 24 hours and subsequently in chronic treatment 0.25 mg per day for 5 days per week.
• beta blockers (metoprolol 50- 100 mg daily in chronic treatment)
• calcium channel blockers (verapamil or diltiazem).

Electric conversion

• In the case of atrial fibrillation with recent installation and without thromboembolic risk, the electrical cardioversion may be attempted by the administration of an initial shock of 100-200 J synchronized with the R.
• In case of failure, the electric shock can be increased to 30 J.

• If the duration of the arrhythmia is longer than 48 hours, then the anticoagulant treatment will be started which will be maintained 3-4 weeks before the electrical cardioversion

• Atrial flutter

• The atrial flutter is a supraventricular tachyarrhythmia that appears frequently paroxysmal and in which the atria contract with a frequency of 250-350 per minute.
• Due to the refractory period of the junction there is an incomplete atrioventricular block 2: 1, (3: 1 or 4: 1), which reduces the heart rate to 140-150 beats per minute.
• Atrial flutter occurs frequently in mitral stenosis, ischemic heart disease and in patients with COPD, against the background of the chronic lung cord.

• Clinical: palpitations, lipotomy, angina pectoris and dyspnea.

• ECG: P waves are replaced with uniform flutter F waves in saw teeth with a frequency of approximately 300 per minute with 2, 3 or 4: 1 functional AV block, optionally variable block.

• Vagal pacing maneuvers such as carotid sinus compression only control the heart rate at most during compression, after which the heart rate returns to the initial 140-150 beats per minute.

• Antiarrhythmic treatment: beta blockers, calcium channel blockers or digoxin.

• In chronic treatment, amiodarone is the most effective because in addition to maintaining the sinus rhythm it can also control the heart rate if the flutter recurs.

• In chronic flutter, anticoagulant therapy can prevent thromboembolic injury.

• An alternative is electrical cardioversion, starting with 25-50 J shocks.

Paroxysmal supraventricular tachycardia

• Paroxysmal supraventricular tachycardia is a regular arrhythmia with a fixed frequency of 160-220 beats per minute.

• Clinical: palpitations, dyspnoea and pre-chondralgia that occur in accessions lasting minutes or hours.
• If paroxysmal supraventricular tachycardia is prolonged: dyspnoea as a result of left ventricular insufficiency leading to hypotension and collapse.
• Paroxysmal supraventricular tachycardia responds to vagal stimulation maneuvers such as carotid sinus compression or Valsalva maneuvers or intravenous adenosine administration, being converted to sinus rhythm.
• ECG: the regular succession of unmodified QRS complexes that are preceded by a P wave.

Ventricular tachycardia

• Ventricular tachycardia is a malignant rhythm disorder consisting of the succession of more than 3 ventricular extrasystoles (enlarged QRS complexes), achieving a relatively regular rhythm with a frequency of 150-180 b / min.
• It rarely occurs in healthy people in the form of paroxysmal access.
• Occurs especially in people with acute myocardial infarction, post-myocardial infarction, left ventricular aneurysm or after heart operations.
• Compression of the carotid sinus has no effect.

• Clinical: palpitations, dyspnoea, angina pectoris, lipotemia, syncope, collapse, Adams-Stokes syndrome by abrupt reduction of heart rate.

• ECG: the sequence of bizarre, large QRS complexes and the absence of P waves.

• Emergency treatment: electric defibrillation with electric shock 100 - 360 J or lidocaine 1 mg / kg body in intravenous bolus and it is due to the risk of death.

Ventricular fibrillation

• represents the most common rhythm disorder in the case of sudden death.
• occurs in about 60% of cases of sudden cardiac death.
• Clinical: syncope, seizures due to cerebral ischemia and cyanosis.
• ECG: chaotic rhythm with disorganized QRS complexes and indefinite frequency.
• Emergency treatment: electric defibrillation by external electric shock application 200-360 J

Sino-atrial block
• Break in cardiac activity lasting approximately two sinus intervals
• If the breaks exceed two sinus intervals, due to the decrease of the cardiac output, the syncope may be installed.
• sick sinus syndrome: episodes of flutter or paroxysmal atrial fibrillation or paroxysmal supraventricular tachycardia are successively associated

The atrio-ventricular block
• 1st degree atrioventricular block
• Delay of transmission of the electrical impulse to the ventricles, which on the electrocardiogram is observed as an extension of the PQ interval over 0.21 seconds

• The second degree atrio-ventricular block has two types:
- Mobitz type I or with Wenckebach periods: progressive depression of the transmission of the electrical stimulus to the ventricles, the duration of the PQ interval progressively increasing until a moment when the transmission of the electrical stimulus to the ventricles is blocked.

- Mobitz II type: intermittent blocking of the transmission of the electrical stimulus to the ventricles according to a block degree (block 2, 3, 4: 1), without a delay of their transmission.

• ECG: after a normal sequence of 2, 3 or 4 of atrial and ventricular depolarizations, a P wave without ORS complex follows.

• Clinically. bradycardia that responds poorly to effort.

• The 3rd degree atrio-ventricular block or the complete block consists of a complete and permanent block of the transmission of the electrical stimulus to the ventricles, producing atrio-ventricular dissociation,

• ECG: regular P waves corresponding to atrial contractions at a normal rate in total discrepancy with the QRS complexes that occur regularly, but at a rare rate of 40 per minute or even less.

• The production of the complete atrioventricular block is rarely asymptomatic, most often the cognitive functions being affected, going up to the syncope.
• The patient may lose consciousness by becoming cyanotic and with sneezing breathing with partial or generalized tonic convulsions

• Clinical: retrograde pulsations of jugular veins according to normal atrial rhythm, in discrepancy with ventricular contraction.

• When the atrium and ventricle contract at the same time, auscultation is noticed when the noise 1 (cannon noise) is accentuated as a result of the collision of the two blood columns at the mitral or tricuspid valves.

LBB major
• the duration of the QRS complex is over 0.12 seconds in the left precordials V5, V6 and Dl and aVL,
- R wave is notched in these derivatives having the appearance of the letter M (RR ').
• The electric axis of the QRS complex is deviated to the left at over - 30 degrees.
• A newly developed LBB may have the significance of an acute myocardial infarction.

Major RBB
• the duration of the QRS complex is over 0.12 seconds in the right precordials V1, V2 and DIII and aVF,
- R wave is notched in these derivatives having the appearance of the letter M (RR ').
• The electric axis of the QRS complex is deflected to the left at +110 degrees; usually between 120-150 degrees.

• **Heart failure**

• Most commonly, heart failure occurs as a result of the primary impairment of the myocardium by loss of muscle mass, as occurs in myocardial infarction, or due to pathological processes that diffuse myocardium such as ischemic heart disease, myocarditis, metabolic disorders or intoxication.

• Heart failure may occur as a result of increased pre-pregnancy in the valvular regurgitation due to aortic insufficiency or as a result of increased post-pregnancy in the case of aortic stenosis or severe hypertension .

• Another cause of heart failure is an increase in metabolic or oxygen needs such as thyrotoxicosis, severe anemia, arterio-venous shunts, infections, being called high flow heart failure.

• Another type of HF appears as a result of impaired diastolic function by increasing the ventricular filling pressure of the ventricles

• Decreased renal infusion due to HF leads to decreased glomerular filtrate with consecutive sodium and water retention, and by activating the renin-angiotensin-aldosterone system, peripheral vascular resistance increases and retention of sodium and additional water will increase the effort. left ventricle (VS).

• Sympathetic hypertonia that occurs in HF results in increased myocardial contractility, heart rate and venous tone.
• Increased contractility and tachycardia may exacerbate myocardial ischemia in a patient with pre-existing coronary artery disease, and increased pre-pregnancy by venous tonic effect may exacerbate pulmonary congestion.

• HF classification

• New York Heart Association:
• class I - dyspnoea appears in high effort
• class II - dyspnea occurs at moderate efforts;
• class III - dyspnoea appears in small efforts such as dressing up! or personal toilet;
• class IV - the patient has resting dyspnoea.

• HF is classified according to the times of the cardiac revolution in:
- Systolic HF in which the force of ventricular contraction is affected, (hypertension , ischemic heart disease)
- Diastolic HF in which the decrease of the cardiac output is caused by an increase of the ventricular diastolic pressure: constrictive pericarditis, cardiac tamponade, myocardial infiltrative processes (amyloidosis).

• Depending on the affected ventricles, the HF is classified into:
- Left HF by increased LV request within a hypertension ;
- Right HF by increased RV request in case of pulmonary hypertension;
- Global or congestive HF, when the function of both ventricles is affected.

• Symptomatology

• Left HF is characterized by exertional dyspnea, cough, fatigue, orthopnea, paroxysmal nocturnal dyspnea, cardiomegaly, bronchial rales, gallop rhythm and pulmonary congestion.

• Right HF is characterized by increased venous pressure, hepatomegaly and declining edema.

• The dominant symptom of left HF is shortness of breath initially and subsequently and at night as orthopnea, paroxysmal dyspnea at night and later at rest, as resting dyspnea.
• Especially at night, the chronic irritant cough may be amplified by the clinostatic position.
• Also during the night, the diuresis increases as a result of the decubitus position when renal perusion increases and the accumulated fluids during the day.

• Patients with HF may also exhibit fatigue and exercise intolerance depending on the modification of peripheral muscle perfusion.

• In patients with right HF, discomfort in the hypochondriac may occur! right through liver congestion, anorexia and nausea through blood flow disorders in the intestine! or declining peripheral edema

ECG
• left ventricular hypertrophy,
• terminal phase changes (ST segment or T wave) characteristic of ischemic heart disease,
• heart rhythm disorders.

Cardio-pulmonary radiography
• large size cord
• signs of pulmonary stasis, enlarged lungs, peribronovascular pattern

Echocardiographic examination
• the dimensions of the heart, of the cardiac chambers, the thickness of the ventricular walls, as well as the ejection fraction of the left ventricle can be measured

Doppler examination allows to determine the speed of blood flow and its meaning in the case of valvulopathies.

Radioisotope angiography allows the determination of ventricular volume and ejection fraction.

The presence of angina pectoris or ischemic heart disease in a patient with HF makes it necessary to perform a coronary artery for the subsequent application of a revascularization procedure by angioplasty or aorto-coronary bypass.

Laboratory investigations:
• Hemogram: Anemia that can trigger a high-flow HF or aggravate cardiac dysfunction.
• Investigating renal function (by determining glomerular filtration rate) is important to determine the impact that HF has on glomerular filtration.
• Determination of serum electrolytes is important for quantifying activated neuroendocrine mechanisms, such as the renin-angiotensin-aldosterone system and ADH antidiuretic hormone secretion, which produce hydrosaline retention.
• The evaluation of thyroid hormones is relevant for a possible hyperthyroidism, possible cause of HF.

• Attention - NT proBNP

Differential diagnosis

• Left HF (in which dyspnea predominates), the differential diagnosis is made with the conditions of the respiratory system such as
- COPD,
- asthma,
- pulmonary emphysema,
- pulmonary fibrosis, pulmonary thromboembolism.

Differential diagnosis

• The signs of pulmonary congestion in the HF should be differentiated from those that appear in pneumonia or pleurisy of infectious cause.
• The increased heart rate in an HF should be differentiated by pericarditis.
• The periferic edema characteristic of HF should be differentiated from the edema that appears by osmotic mechanism in states with hypoproteinemia due to cirrhosis, nephrotic syndrome or malnutrition.

Management of heart failure

Correcting reversible causes

• treatment of ischemic dysfunction of VS, hyperthyroidism, myxemem, valvulopathy, septal defects, arrhythmias and cardiomyopathy induced by alcohol or medicines.
• Some drugs, such as calcium channel blockers and antiarrhythmics, can aggravate HF and should be avoided.
• In the case of diastolic HF, reversible causes are pericarditis and secondary HVS hypertension .

Diet and activity

• Patients with HF should reduce their salt intake to 4-5 g / day (without salt, no salt added to the plate, bread can be salted).

• Reduction in physical activity may lead to temporary compensation of the HF.

• Depending on the HF degree, performing physical exercises according to the tolerance to the effort leads to the increase of the capacity of effort in these patients.

• Diuretics are the medication of choice for patients with moderate or severe congestive HF.

• In the case of mild HF, thiazide diuretics acting on the proximal segment of the distal contorted tube are administered, inhibiting sodium uptake and resulting in increased natriose and kaliuresis (hydrochlorothiazide 25-50 mg).
- Thiazides are effective at a glomerular filtration rate of over 30 ml / min.
- Adverse reactions: hypokalemia, prerenal azotemia by decreasing volume, hyperglycemia, hyperuricemia, neutropenia and thrombocytopenia.

• If the HF is more severe, a loop diuretic will be used, the diuretic and natriuretic effect induced by them having a faster onset and a shorter duration (furosemide 20-320 mg daily).
- Adverse reactions: decreased volemia with prerenal azotemia and hypotension. Hypokalemia induced by goat diuretics can be dangerous in the context of digital treatment.
• The hypokalemic effect produced by goose or thiazide diuretics can be counterbalanced by using potassium-sparing diuretics - spironolactone,

• ACE inhibitors improves the symptomatology of HF and increases the tolerance to the effort by decreasing the post-pregnancy and increasing the cardiac output.

• Counterbalances diuretic-induced hyponatremia and hypokalemia, which prevents arrhythmias.

• ACE inhibitors may delay the onset of HF in patients with asymptomatic ventricular dysfunction.

• Before treatment with lECA, other vasodilators will be discontinued and the administration of diuretics will be reduced or even interrupted for 24 hours.

• ACE inhibitors treatment of heart failure (HF) should take into account the patient's blood pressure and the degree of renal failure as a result of a deficient renal infusion.

• As ACE inhibitors increases potassium, the administration of potassium-sparing diuretics should be discontinued prior to treatment with ACE inhibitors. In case of cough, an adverse reaction of ACE inhibitors, the sardines may be used. However, their effect on HF is smaller because prostaglandin synthesis is lower.

• Treatment with beta-blockers is reserved for NYHA I-III associated with diuretic treatment and ACE inhibitors.

• Beta-blockers are contraindicated in the case of decompensated HFs.

• Carvedilol, which in addition to the beta-blocking effect also has a vasodilating effect by decreasing peripheral vascular resistance, is administered at the initial dose of 3.125 mg twice daily, increasing subsequently at a weekly interval of 6.25 mg x 2. / day, 12.5 mg x 2 / day and 25 mg x 2 / day, depending on patient tolerance.

• The application of the beta-blocker treatment should be carefully supervised by the doctor, because in about 10% of the patients treated, the signs of HF may worsen, increasing the dose of diuretic.

Tonicardiac treatment
• Digital treatment remains of primary intention in patients with HF and atrial fibrillation.
• The administration of digital treatment improves the symptomatology of HF, decreases the frequency of hospitalizations and increases the tolerance to effort of the patients.
• Digoxin administration is indicated in patients in whom the administration of diuretics and ACE inhibitors does not improve the symptoms of heart failure.

Anticoagulant treatment
• has indication in cases where the ejection fraction of the left ventricle decreases to 10-15% (dilated cardiomyopathy, extensive infarctions).

Monitorising of heart failure by the family doctor

• The role of the family doctor:
- to make the diagnosis of HF as early as possible
- to send the patient to the cardiologist in order to confirm the diagnosis, establish the cause of the HF and elaborate the therapeutic plan.

- to follow the evolution of the disease in the clinical context of the patient,
- to supervise the results of the treatment.

• The evolution factors considered in the monitorising of heart failure by the family doctor will be:
- ventricular allure;
- weight gain;
- the appearance of peripheral edema;
- degree of dyspnoea;
- limiting the level of physical effort of the patient;
- peripheral pulse.

• **Peripheral arterial disease**

• fatigue when walking,
• paresthesias in the calf and ankle;
• intermittent claudication - painful cramp usually located in the knob, which appears on the go and gives up at rest; pain may also occur in the thigh or buttock region (in high arterial obstruction) or in the plant (obliterating thrombangitis, diabetes).
• pain at rest appears in advanced stages - it is diffuse, atrocious, with nocturnal exacerbations; initially it is calmed by the declining position of the legs, later it becomes permanent, impedes sleep and is refractory to treatment.
• sexual dysfunction (in Leriche syndrome)

Clinical examination and history:
• association with diabetes, dyslipidemia;
• presence of cardiovascular risk factors (smoking, stress, sedentary lifestyle);
• other atherosclerotic determinations: cerebral, coronary; different values of BP in both hands;
• low skin temperature in advanced phases;
• skin trophic disorders:
• reduced hairiness,
• thickened and cracked nails,
• small petitions;
• atrophy of the leg muscles;
• stasis edema of the calves;
• late wound healing,
• increased tendency for skin infections;

• ischemic ulcers - small, located interdigitally, plantar - occur spontaneously or after minimal trauma;
• ischemic ulcers - located on the dorsal side of the foot and presumably, the bottom of the ulcer contains gray granulation tissue and the surrounding skin is pale and marbled;

• dry gangrene - necrotic, non-painful, black-purple tissue, covered by a crust; damp gangrene, with edema and lymphangitis;
• examination of the arteries - the diminution or disappearance of the pulse (the peripheral arteries symmetrically and the abdominal aorta will be palpated) may point to the level of obstruction;
• arterial breath at auscultation;
• Burger test-
- raising the lower limbs to 60-70 degrees in the dorsal position and performing movements in the ankle joint;
- the affected leg becomes pale;
- raising the feet to 30 degrees above the horizontal plane with
- accentuation of the hat and delayed capillary and venous filling time at the affected limb;
- moderate obstruction - refill in 25-30 sec; severe obstruction - recoil in more than 40 sec.

Paraclinical explorations in obliterating arteriopathy:
• usual: hemogram, blood glucose, lipid profile, C-reactive protein, kidney samples,
• ECG.
• The determination of the ankle-arm index is considered the non-invasive method of first intention for screening and diagnosis.
- It can be determined both in the hospitalized patient and in the ambulatory, the examination can be done including by the family doctor.
- The necessary equipment consists of a tensiometer and an arterial Doppler - calculates the ratio of systolic BP at the ankle level and systolic BP at the arm level, taking into account the highest value of the BP between the two arms.

• Duplex arterial ultrasonography (DUS) is used for the detection of vascular lesions, the location and quantification of their severity and extent, the measurement of intimate-medium thickness, the assessment of cardiovascular risk.

• Contrast substance angiography by computer tomography and / or magnetic resonance imaging are indicated for lesion localization and to consider revascularization options.

Treatment

• Combating cardiovascular risk factors and preventing systemic complications of atherosclerosis:
• quitting smoking
- decreases cardiovascular mortality and cardiovascular risk,
- decreases the severity of claudication and the risk of resting pain (counseling, nicotine replacement treatment);

• decreased blood pressure using IEC / angiotensin receptor antagonists, with significant effects on prophylaxis of cardiovascular or calcium channel blocker events

• beta-blockers should be administered with caution in advanced forms of the disease only if absolutely necessary;
• early detection of diabetes mellitus and blood glucose control;
• weight loss;
• daily walking training in relation to the claudication threshold - encouraging short-distance walking, until near the time of pain (at least 6 months);
• maintaining hygiene of the ischemic leg:
• wearing cotton and wool socks in winter,
• warm footwear in the cold season,
• the immediate avoidance and treatment of any infections occurring at this level.

Pharmaceutical treatment

• Antiplatelet therapy:
- acetylsalicylic acid 75-150 mg / day;
- ticlopidine 500 mg / day (risk of thrombocytopenia, thrombocytopenic purpura, neutropenia); clopidogrel 75 mg / day.

• Cilostazole
- phosphodiesterase III inhibitor, indicated in the treatment of intermittent claudication to increase the walking distance, in patients who do not have rest pain or semen of necrosis of the peripheral tissue, cadiac insufficiency;
- is given in doses of 100 mg twice daily,
- may induce arrhythmias, hypotension, bleeding (by antiplatelet effect), pancytopenia, aplastic anemia.

• Pentoxifylline
- methylxanthin derivative that inhibits phosphodiesterase having a weak vasodilator myotropic effect,
- is used in particular for its rheological effects,
- inhibits platelet aggregation,
- increases the elasticity of red blood cells
- the viscosity of the blood decreases.
- It is given intra-arterially, intravenously, intramuscularly 100-200 mg 3 times daily, subsequently per bone orally 400 mg 3 times daily and represents the second alternative for improving walking distance.

• Physiotherapy
- aims at developing the collateral circulation,
- Recommended resorts: Covasna, Vatra Dornei, Buziaş, Tuşnad.

• Treatment of ulcers and gangrene will be performed in collaboration with the surgeon or dermatologist,
• Endovascular treatment

• **Varicose vein disease**

• Varicose veins can be:
- primary, caused by the insufficiency of venous valves or abnormalities of the venous wall;
- secondary to deep vein obstruction by:
• thrombophlebitis,
• venous occlusion of neoplastic cause,
• arteriovenous fistulas

Favorable conditions:
• genetic factor,
• female sex (the estrogenic component),
• prolonged orthostatism,
• professions that require prolonged standing or sitting, lifting weights,
• pregnancy,
• obesity,
• chronic constipation.

Symptoms
• feeling of anxiety, fatigue, discomfort, numbness, weight in the calf,
• appearing after orthostatism, intense efforts, sitting position for a long time.

• They diminish or disappear in the lying position, with the feet slightly raised to the plane of the body.

Physical examination:
• edema,
• hyperpigmentation of the skin
• pruritus secondary to stasis dermatitis,
• thinning of the skin

Diagnostic
• symptoms and signs;
• the presence of favorable conditions;
• Doppler exam

Differential diagnosis
• primary varices / secondary varices;
• arteritis;
• root syndrome.

CEAP classification
• Stage C0: no visible, palpable or paraclinically detectable signs of chronic venous insufficiency;
• Stage C1: remote sensing and / or reticular veins;
• Stage C2: varicose veins present;
• Stage C3: the presence of edema;
• Stage C4a: dermis of stasis;
• Stage C4b: lipodermatosclerosis and / or white atrophy
• Stage C5: "healed" venous ulcer;
• Stage C6: active, open venous ulcer.

Treatment

• avoiding prolonged orthostatism;
• wearing their compression stockings (they will be dressed in the morning and dressed in the evening),
• the thickness of the stockings will be chosen according to the degree of affectation;
• weight loss;
• local wound care;
• avoidance of constipation;
• avoiding wearing heels higher than 5 cm, but also flat shoes;
• avoid sauna, hot baths, hot waxing, prolonged sun exposure;
• combating sedentary lifestyle through a physical exercise program,
• avoidable sports: horse riding, tennis;
• Recommended sports: jogging, swimming, cycling;
• persons carrying out sedentary activities (office work) will be advised to perform periodically movements of the legs, rotation, flexion-extension of the feet, walking on the tips, on the heel;
• nocturnal rest with raised legs;
• pregnancy monitoring;
• avoiding too tight cords and clothes.

• Diosminum (reduces venous stasis, distensibility and capillary permeability),
• Troxerutinum,
• Ginkobiloba,
• Vessel due (heparinoid indicated in venous pathology with risk of thrombosis),
• heparins or heparinoids for topical use.

Surgical:
• ligation or removal of varicose vein dilations and incompetent perforating veins;
• sclerotherapy - injection of sclerosing solutions in varicose veins (preferred procedure for varicose veins and venules 1-3 mm)
• intravenous lasers