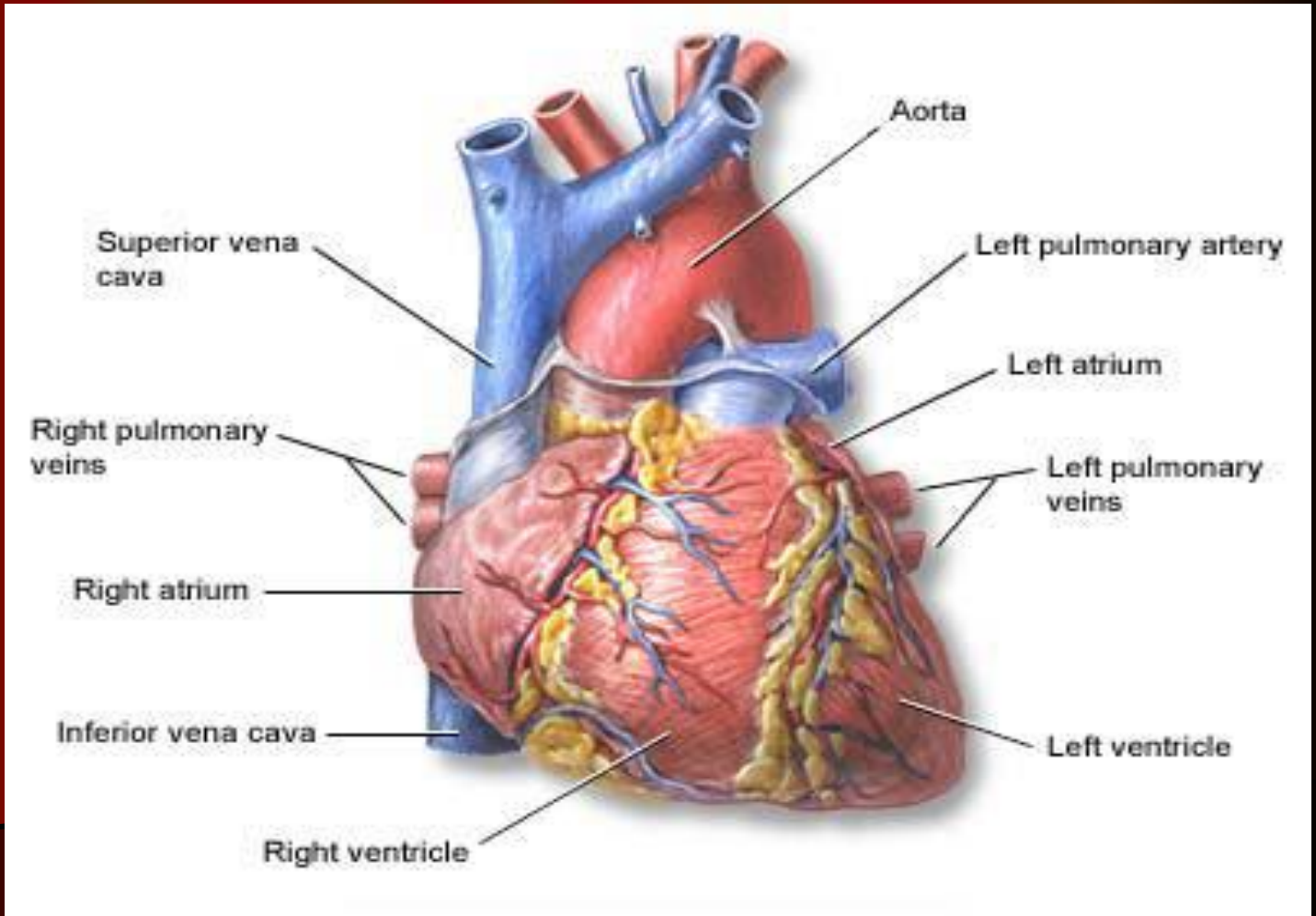
MANAGEMENT OF PATIENTS WITH CARDIOMEGALY AND HEART FAILURE



- **Cardiomegaly** is a considerable enlargement of the heart from its dilatation and/or hypertrophy, accumulation of waste products due to impaired metabolism or development of neoplastic processes.
- It is usually manifested by symptoms of heart failure, rhythm and conduction disorders.

COMMON SIGNS OF CARDIOMEGALY

- Enlargement of the heart
- Rhythm and conduction disturbances
- Physical findings: widened borders of the heart, dull sounds, weakened 1st sound over the apex, presystolic gallop, additional 3rd and 4th sounds, murmurs of regurgitation
- Signs of the underlying disease which has caused cardiomegaly

MAIN CAUSES OF CARDIOMEGALY

- IHD: atherosclerotic cardiosclerosis, post-infarction cardiosclerosis, ischemic cardiomyopathy, cardiac aneurysm
- Arterial hypertension
- Heart defects (congenital, acquired)
- Diffuse myocarditis
- Primary and secondary cardiomyopathy
- Pericarditis
- Cardiac tumours
- Athlete’s heart

DIAGNOSIS OF CARDIOMEGALY

- Interviewing the patient to find out the main complaints: dyspnoea, fatigability, weakness, less tolerance to physical exertion; feeling of heaviness in the right hypochondrium, peripheral oedemas, pains in the chest (angina or cardialgia), heart palpitations

DIAGNOSIS OF CARDIOMEGALY

- History. We should specify: consequence of development of heart failure symptoms (left or right ventricular failure separately or total heart failure at once), episodes of BP elevation, history of acute rheumatic fever, heart murmurs revealed in childhood, history of myocardial infarction, aches in the heart in the past, severe diabetes, connection with past infections, vaccination, alcohol consumption, medications, family history.

DIAGNOSIS OF CARDIOMEGALY

- PHYSICAL EXAMINATION:
- Inspection: cyanosis, acrocyanosis, paleness, ruddiness of cheeks, swollen veins in the neck, pulsating vessels or precardial area, widened venous network, enlarged abdomen, orthopnoea;
- Palpation: assessing the pulse rate (slow, of small amplitude or full, rapid; pulse deficit), chest tremors, apical pulse shifted to the left;
- Taking BP: normal, hypertension, high pulse pressure

DIAGNOSIS OF CARDIOMEGALY

- PHYSICAL EXAMINATION:
- Percussion: wider vascular bundle, wider borders of heart dullness
- Auscultation: dull sounds, weakened 1st sound, protodiastolic or presystolic gallop rhythm, extra 3rd and 4th sounds, murmurs of regurgitation, cardiac rhythm disorders;

DIAGNOSIS OF CARDIOMEGALY

- LABORATORY FINDINGS:
- CDC: diagnosis of anaemia, polycythemia (COPD, cyanotic congenital heart defects), leucocytosis and elevated ESR (infectious endo-, myocarditis, exudative pericarditis)
- Blood and urine glucose: diabetes mellitus
- Lipid profile: CHD, aortic atherosclerosis
- CRP test: endo-, myocarditis, systemic connective tissue diseases (SCTD)
- Antinuclear and rheumatoid factors: SCTD
- Bacterial blood culture: infectious endocarditis
- T3, T4, TSH: hypo-, hyperthyroidism
- Urea, electrolytes, creatinine: CKD

DIAGNOSIS OF CARDIOMEGALY

- INSTRUMENTAL INVESTIGATIONS:
- Chest X-ray (shape of the heart, enlargement of certain chambers, vessels): ‘mitral’, ‘aortic’ or spherical shape of the heart;
- ECG: the changes are non-specific and manifold (hypertrophy of chambers of the heart, rhythm and conduction disorders, changes due to scarring of the heart)

DIAGNOSIS OF CARDIOMEGALY

- INSTRUMENTAL INVESTIGATIONS:
- Echocardiography is the most valuable non-invasive methods of diagnosis assesses thoroughly morphological changes in the chambers and valves of the heart, peculiarities of movement of valves, thickened areas and calcinosis, impaired movement of blood in the heart, signs of pulmonary hypertension, elevated LVEDP, width of heart walls, asymmetry due to hypertrophy or symmetry, areas of hypo- or akinesia.

MANAGEMENT OF PATIENTS WITH CARDIOMEGALY

- To confirm cardiomegaly (to determine enlargement of the chambers, dilation or hypertrophy, to estimate the degree of enlargement of the chambers)
-
- To reveal the cause of cardiomegaly
-
- To assess its functional significance
-
- To plan management of the patient

MANAGEMENT OF PATIENTS WITH CARDIOMEGALY

- **ASSESSMENT OF FUNCTIONAL SIGNIFICANCE OF CARDIOMEGALY**:
- Symptoms of dyspnoea, weakness, fatigability
- Cardiac ventricular function (EF), congestive heart failure
- Determination of NYHA functional class of heart failure

MANAGEMENT OF PATIENTS WITH CARDIOMEGALY

- **PLANNING MANAGEMENT OF THE PATIENT:**
- Prevention: changing lifestyle, treatment of hypertension, CHD or any other underlying disease
- Medical treatment: diuretics, ACE inhibitors (or sartans), beta-blockers, nitrates, antiaggregants, anticoagulants, cardiac glycosides, antiarrhythmic drugs
- Surgical treatment

CARDIOMYOPATHIES (CM)

European Society of Cardiology (ESC), 2008

"Cardiomyopathies are structural and functional myocardial diseases in the absence of systemic hypertension, coronary atherosclerosis, valvulopathies, or congenital heart disease".

CARDIOMYOPATHIES

CM phenotypes

1. HCM (hypertrophic CM)
2. DCM (dilated CM)
3. ARVD (arrhythmogenic right ventricular dysplasia)
4. RCM (restrictive CM)
5. Non-classified:
   - Non compacted myocardium
   - Stress CM (Takotsubo CM)

ESC RECOMMENDATIONS (2008)

All CM phenotypes are divided into:

- Familial (inherited, genetic)
- Non-identified genetic disorder
- A disease subgroup (including those with known gene mutation, metabolic disorders, glycogen storage disease, impaired fatty acid metabolism, lysosome storage disorders)

2. Non-familial (acquired, non-genetic)

- Idiopathic
- A disease subgroup
  - Toxic CM
  - Endocrine CM
  - Alimentary (nutritional) CM (thiamine or selenium deficiency, hypophosphataemia, hypocalcaemia)
  - Alcohol-induced CM
  - Tachicardia-induced CM
  - Peripartum CM
  - Athlete's heart
  - CM in children born to mothers with insulin-independent diabetes
  - Inflammatory CM

HYPERTROPHIC CARDIOMYOPATHY

- Hypertrophic cardiomyopathy is defined by the presence of increased left ventricular (LV) wall thickness that is not solely explained by abnormal loading conditions.
- HCMP occurrence is 0.02-0.23% in adults and unknown in children, incidence being equal approximately to 0.3-0.5 per 100000 of population (0.005-0.07%). Most studies note that men develop the disease more often than women; CM prevalence rate in different races is similar.

HYPERTROPHIC CARDIOMYOPATHY

2014 ESC guidelines on diagnosis and management of hypertrophic cardiomyopathy

- HCM is prevalently a genetic disease of the muscle of the heart characterised by a set of specific morphological and functional changes and progressing steadily with a high risk of development of severe life-threatening arrhythmia and sudden cardiac death.
- HCM synonyms
  - Idiopathic hypertrophic subaortic stenosis
  - Muscular subaortic stenosis
  - Hypertrophic obstructive cardiomayopathy

HYPERTROPHIC CARDIOMYOPATHY

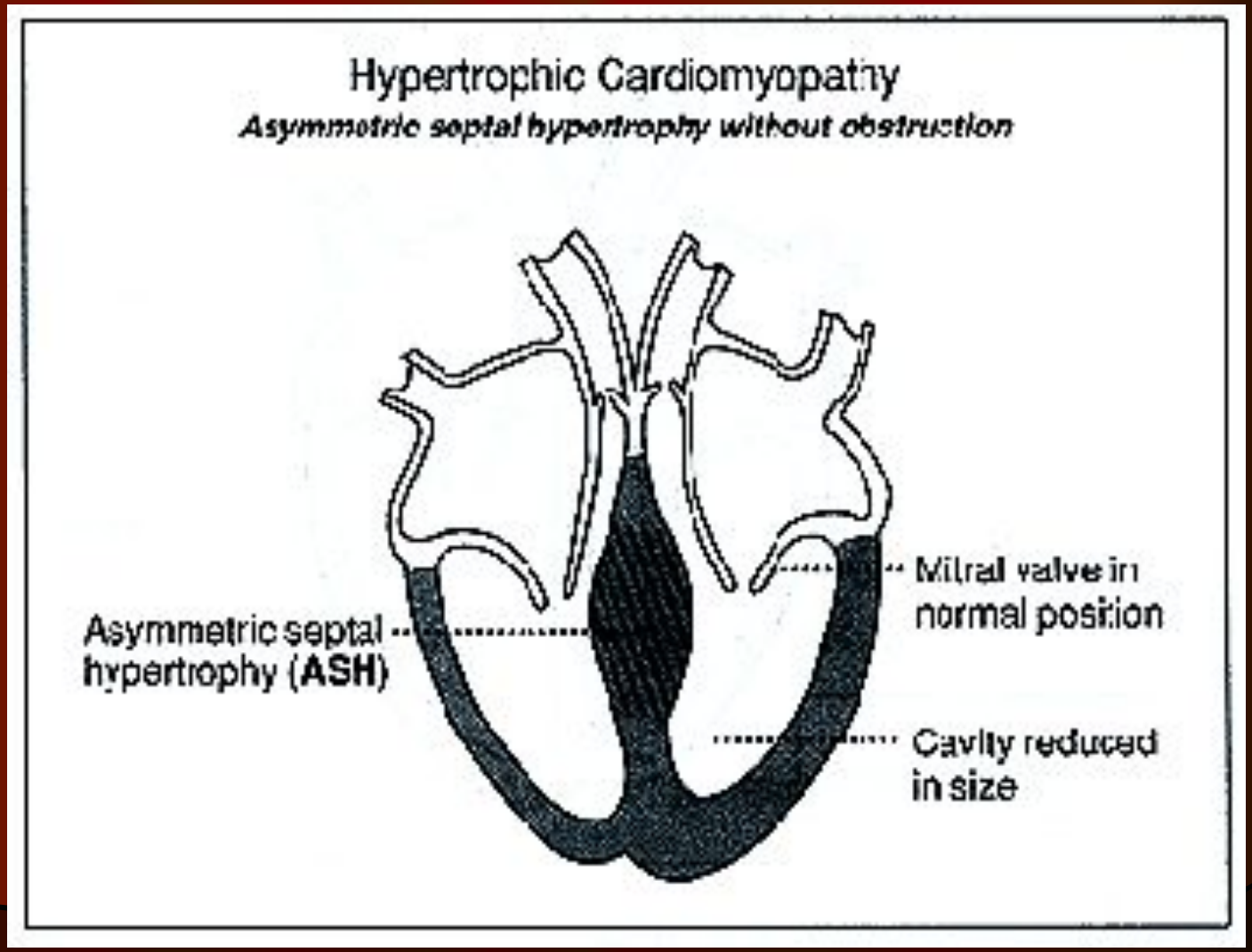
- HCM is the main cause of sudden cardiac death (SCD) in the young, in sportsmen in particular

- The findings of autopsies of sportsmen who died of SCD show that death may have been caused by non-revealed or clinically silent HCM in 36% of cases.

HYPERTROPHIC CARDIOMYOPATHY

- **HCM is characterised by considerable (more than 15 mm) hypertrophy of myocardium of the left and/or in rare cases right ventricle, often asymmetric due to thickened IVS, with frequent development of left ventricular outflow tract obstruction (LVOTO) in the absence of known causes (hypertension, heart defects and specific heart diseases).**

HYPERTROPHIC CARDIOMYOPATHY



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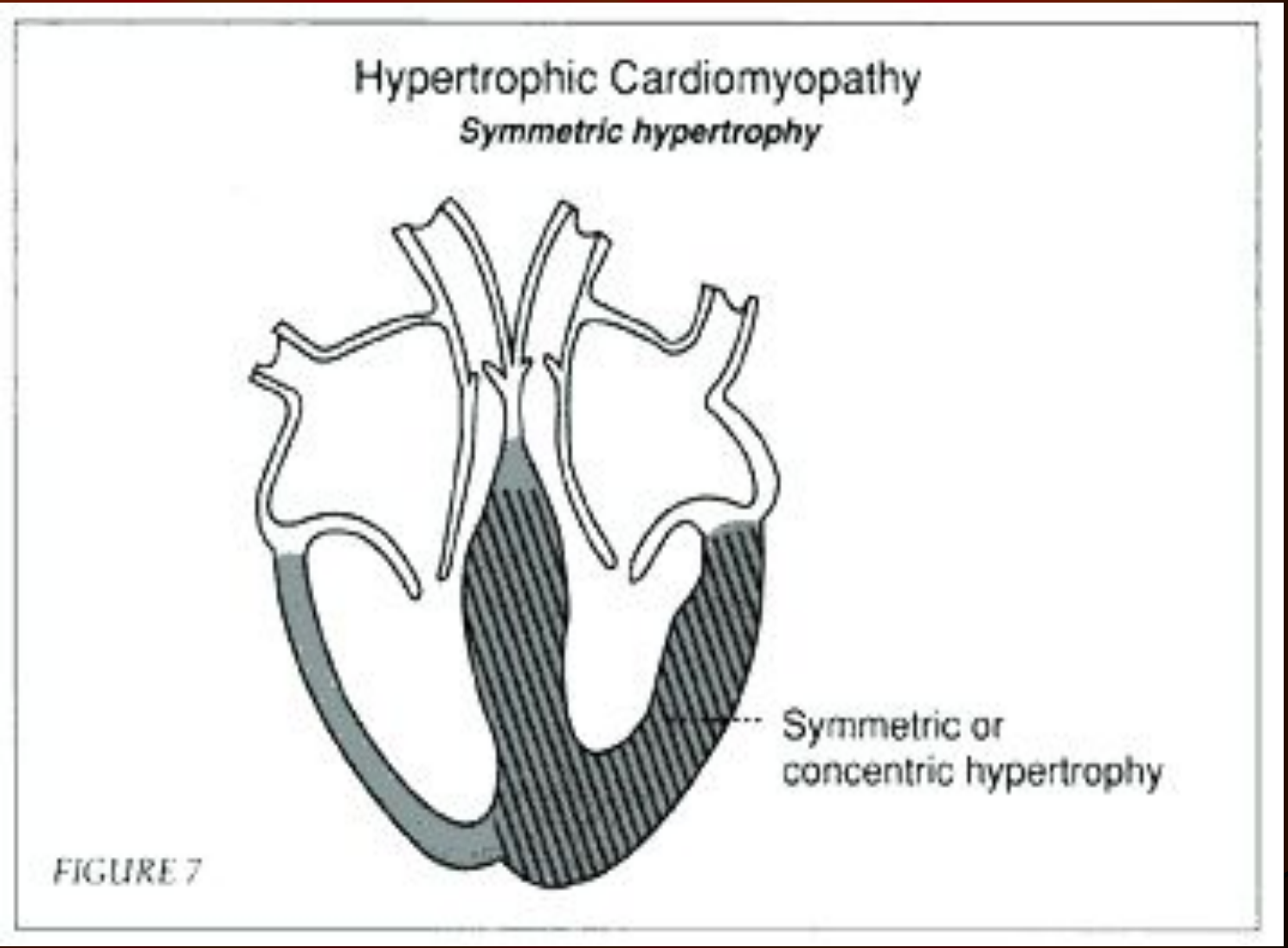

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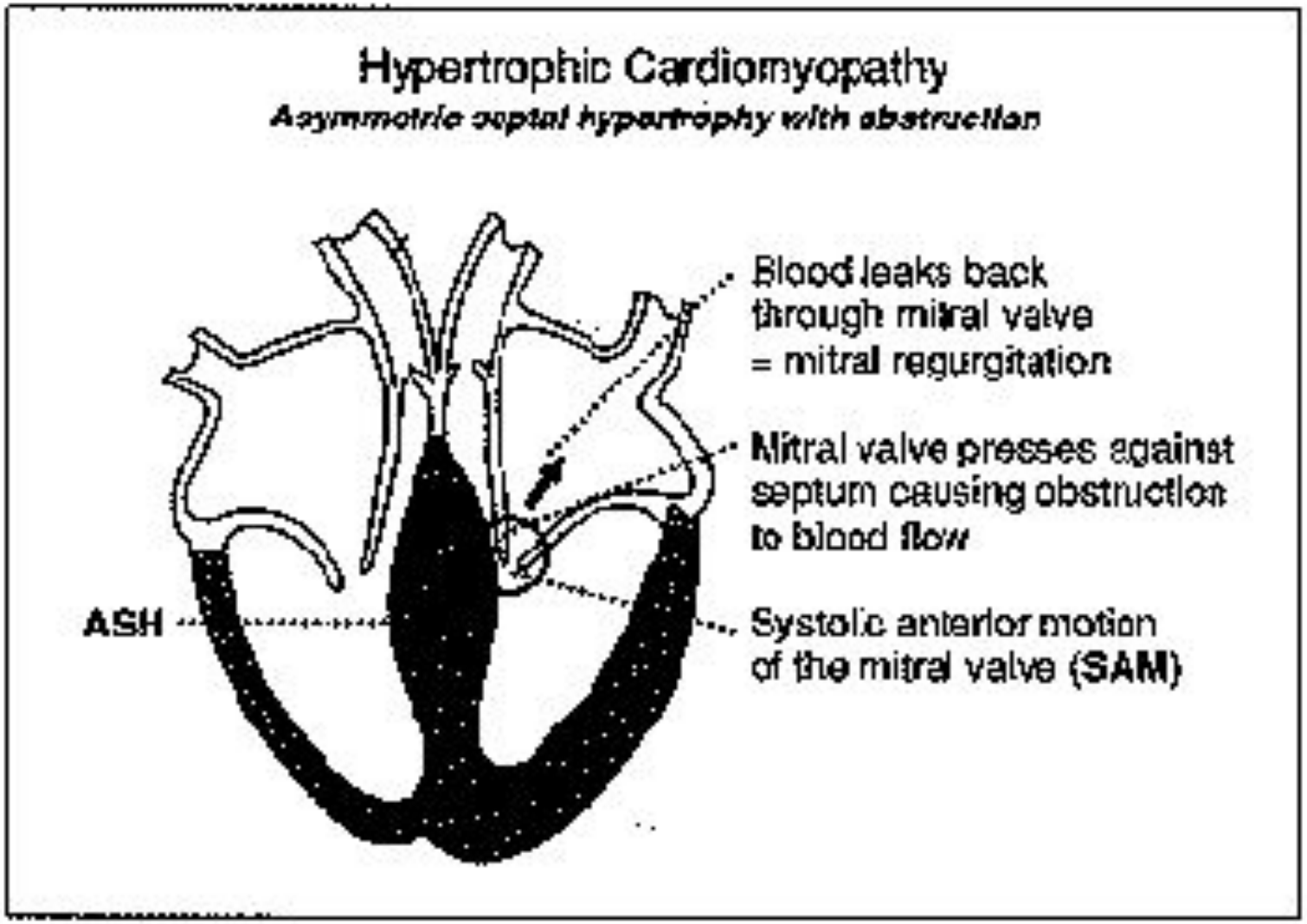
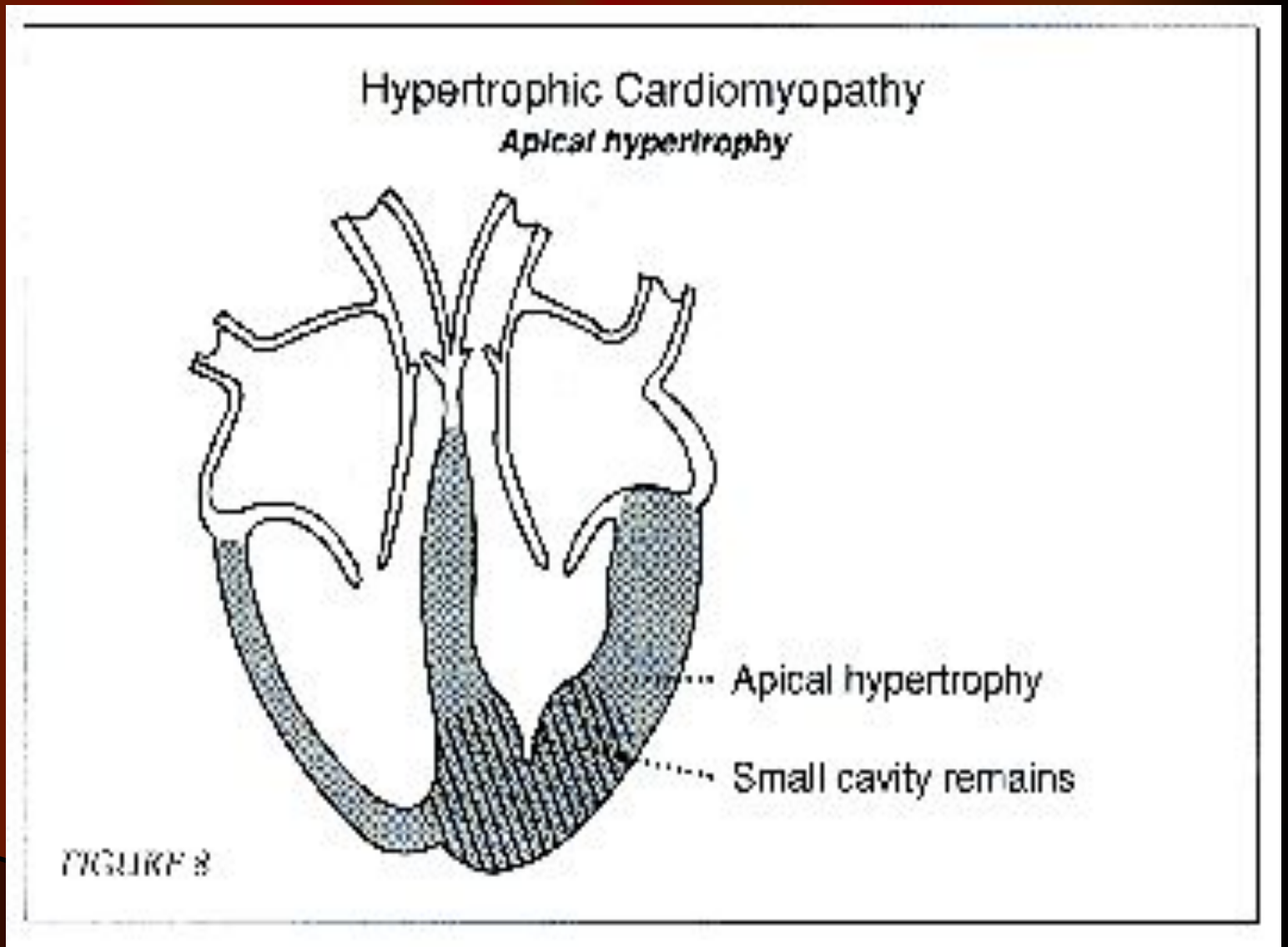


HYPERTROPHIC CARDIOMYOPATHY



HYPERTROPHIC CARDIOMYOPATHY

Pathogenesis of HCM includes 4 interrelated processes:

1. Left ventricular outflow tract obstruction (LVOTO)
2. Diastolic dysfunction
3. Myocardial ischaemia
4. Mitral regurgitation

HYPERTROPHIC CARDIOMYOPATHY

CLINICAL MANIFESTATION:

- Asymptomatic course in 25% cases
- Dyspnoea on exertion (90%), orthopnoea;
- Angina (70-80%);
- Syncope (20%), presyncope (50%)
  - Greater obstruction in augmentation of cardiac contractility due to exertion;
- Cardiac arrhythmias (90%)
- Thromboembolic risks of atrial fibrillation
- Sudden cardiac death

HYPERTROPHIC CARDIOMYOPATHY

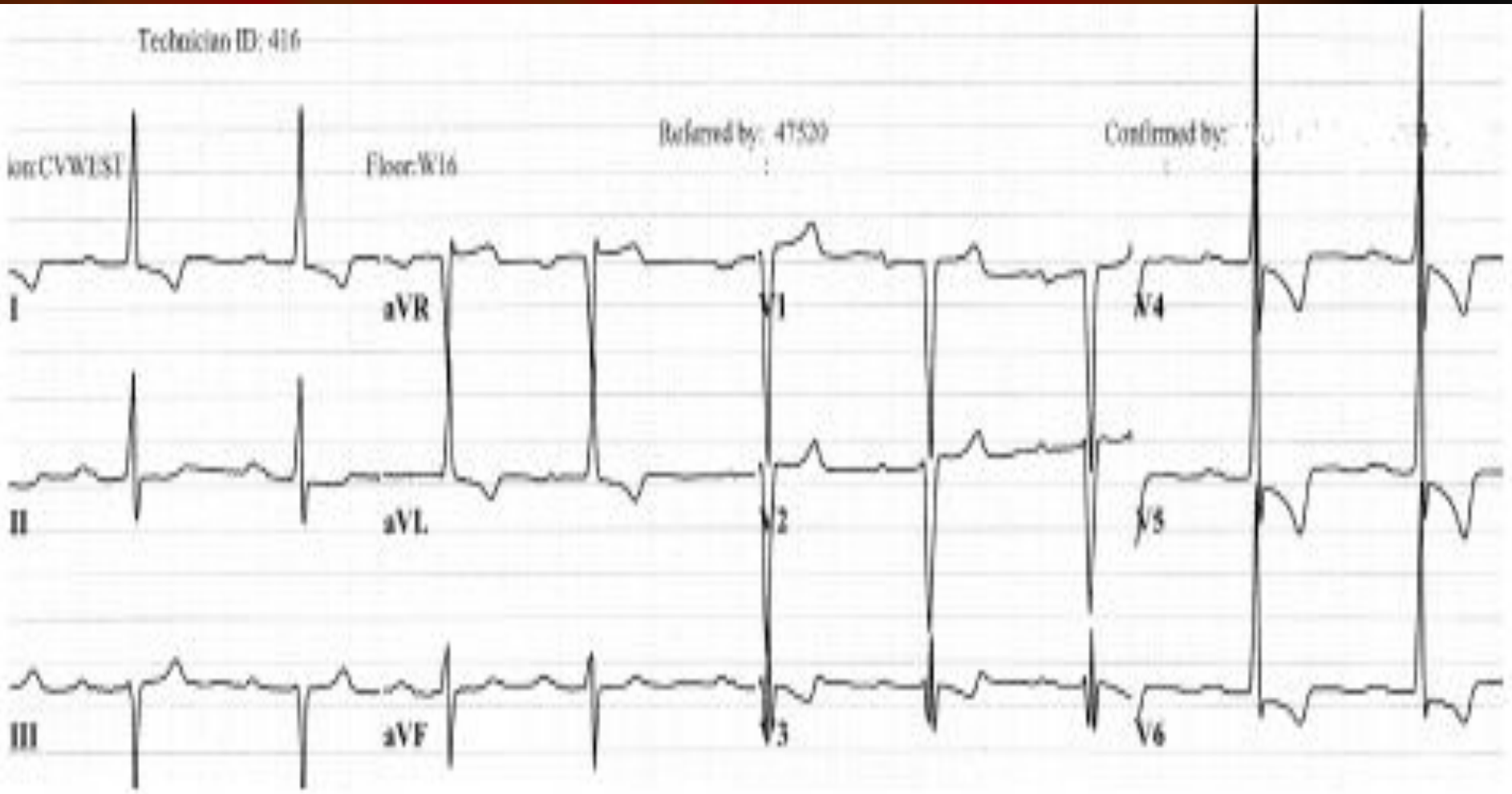
- ON EXAMINATION:
- intense, raised cardiac impulse shifted slightly to the left
- double, triple or even quadruple impulse over the apex of the heart
- alternating pulse
- An ejection systolic murmur over the apex or in the 3rd-4th intercostal space at the left sternal edge, ‘rhomboid’ character of systolic murmur

HYPERTROPHIC CARDIOMYOPATHY

DIAGNOSIS:

- DNA-diagnosis using polymerase chain reaction (PSR)
- Genetic testing of relations in the first degree to assess the risk of development of HCM
- ECG and daily monitoring of ECG
- Chest X-ray, EchoCG, MRI
- Cardiac stress tests
- Coronary angiography

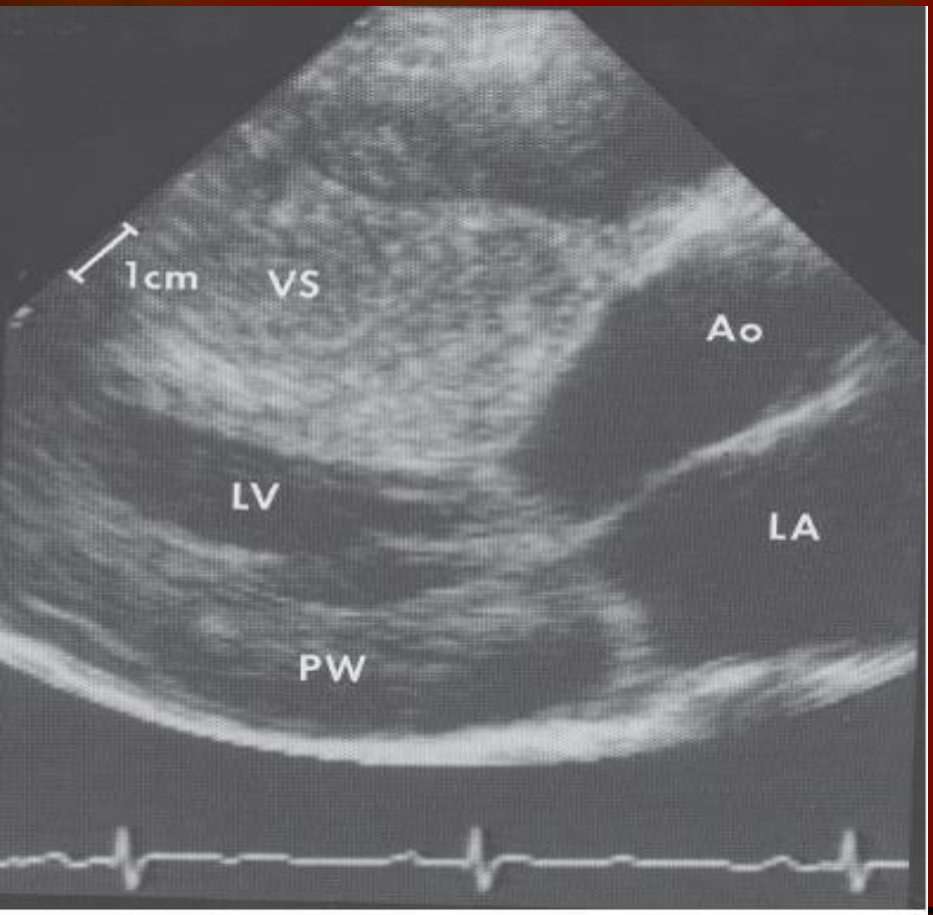
HYPERTROPHIC CARDIOMYOPATHY

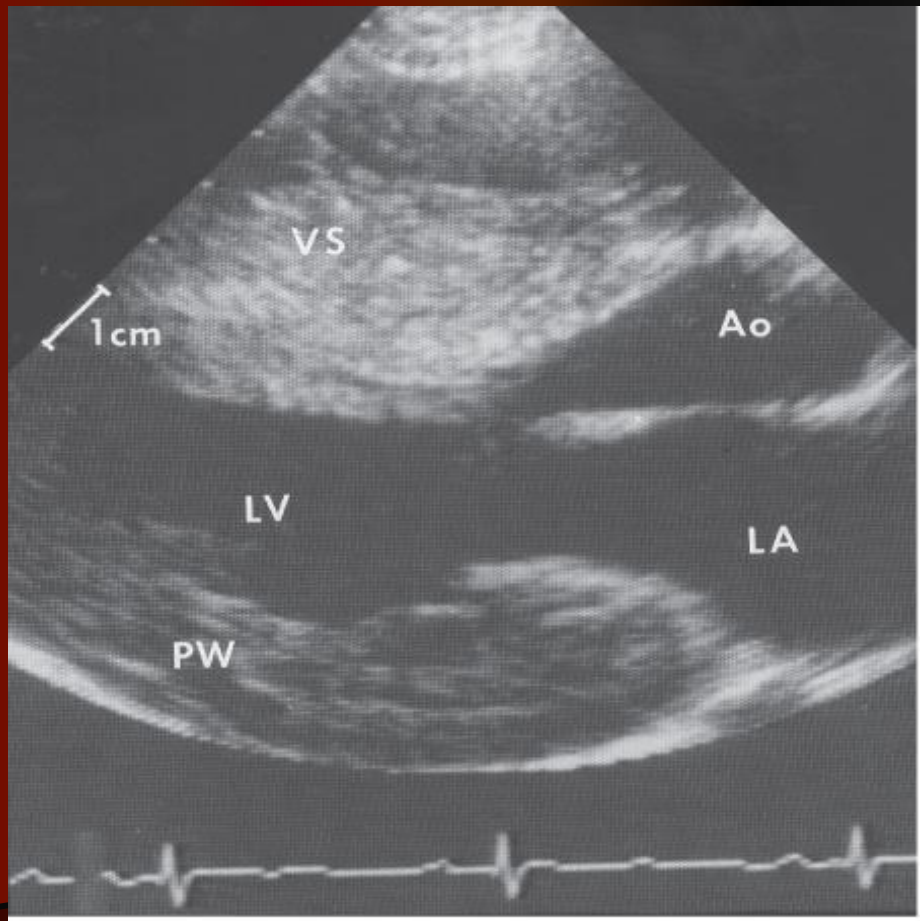


Source: Fuster V, O'Rourke RA, Walsh RA, Poole-Wilson P: *Hurst's The Heart*, 12th Edition: http://www.accessmedicine.com

HYPERTROPHIC CARDIOMYOPATHY

- Left ventricular wall or IVS thickness >15 mm





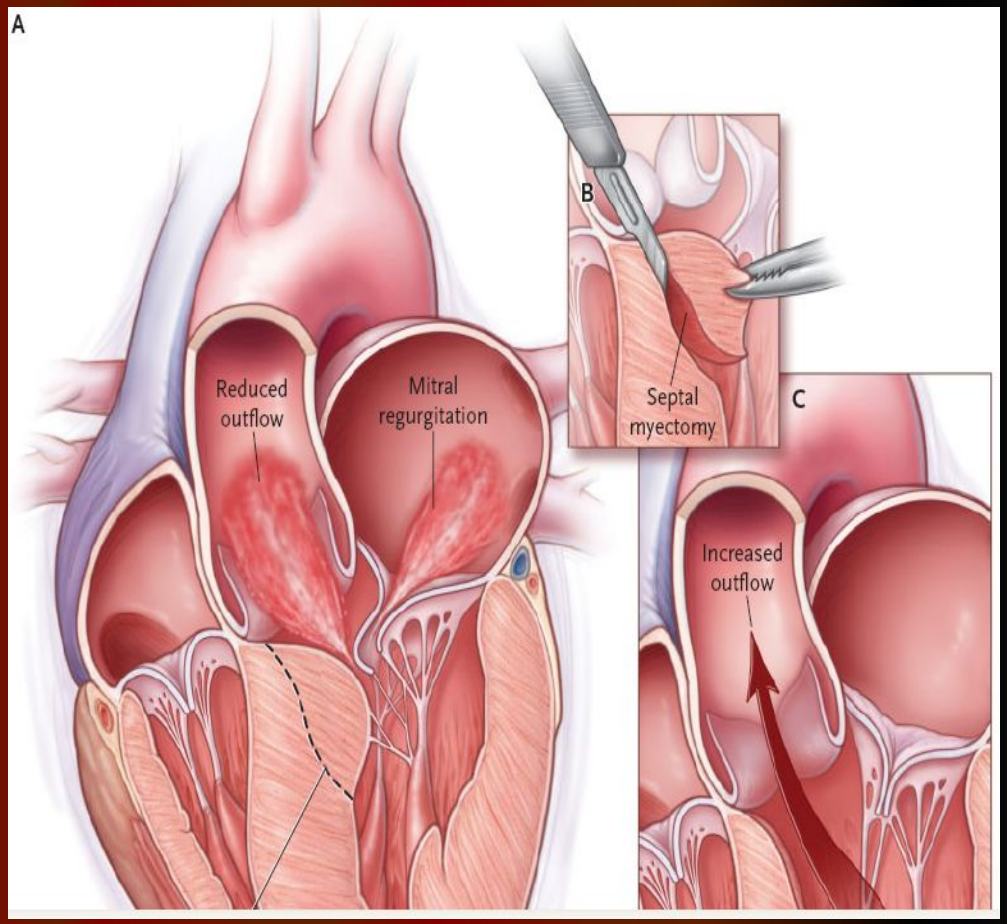
HYPERTROPHIC CARDIOMYOPATHY

- MEDICAL TREATMENT:
- ß-blockers
  - Increase diastolic filling/relaxation of the LV
  - Are first choice in obstructive and non-obstructive forms
  - Decrease myocardial oxygen demand
  - Have shown no effect on SCD risk
- Verapamil (480 mg)
  - Improve diastolic function of the LV
  - Relieve symptoms (especially pain behind the breastbone)
- Disopyramide
  - Is used in combination with ß-blockers
  - Negative inotropic effect
- Diuretics
- Amiodarone, Sotalol (to treat arrhythmia)

HYPERTROPHIC CARDIOMYOPATHY

- Invasive methods of HCM management
- Transaortic septal myectomy (Morrow's procedure)

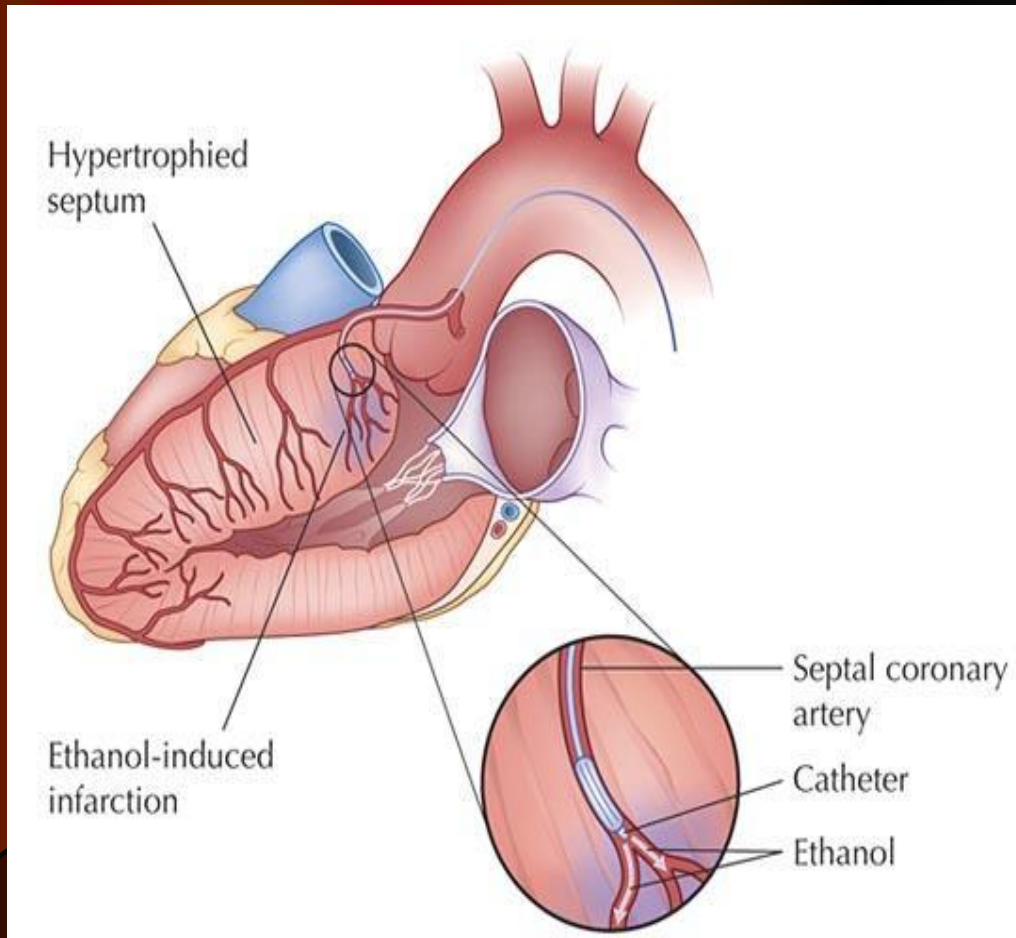
is a 'gold' standard to decrease left ventricular outflow tract obstruction (LVOTO) for both children and adults.



HYPERTROPHIC CARDIOMYOPATHY

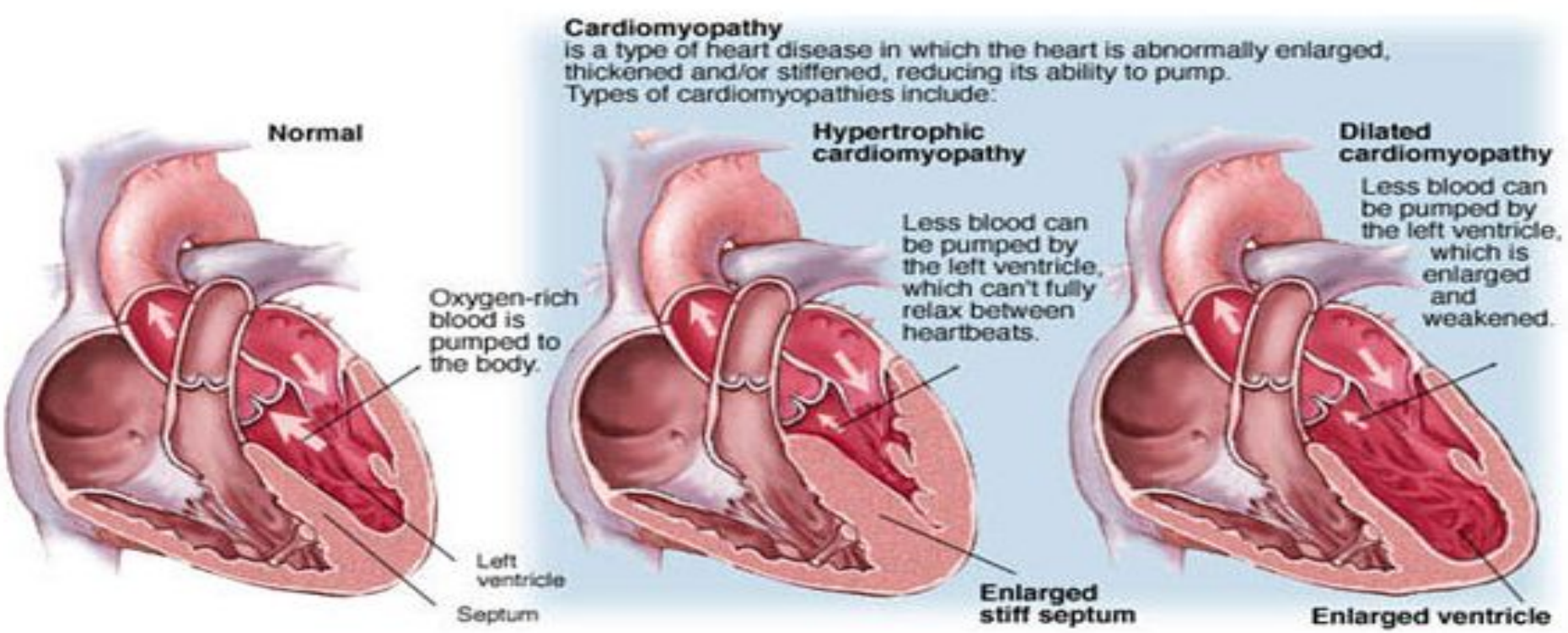
- Percutaneous transluminal septal alcohol ablation

May be chosen for highly symptomatic adult patients with LVOTO, resistent to medical treatment and if other methods are undesirable for them



DILATED CARDIOMYOPATHY (DCM)

- ***Is a disease of the cardiac muscle characterised by dilation and impaired contractility of the left ventricle or both ventricles of the heart (WHO).***



DILATED CARDIOMYOPATHY

- Dilated cardiomyopathy is responsible for 9% of all cases of heart failure. Incidence of dilated cardiomyopathy is 3 to 10 cases per 100 000 people.
- It affects men more often, occurring mostly in adults 20 to 50
- **ESC (2008) DCM diagnosis is confirmed if** there is dilation and impaired contractility of the left ventricle in the absence of CHD, valvular pathology or hypertension.

CLINICAL MANIFESTATIONS OF DCM

- Symptoms: palpitation, syncopes, weakness, dyspnoea, reduced exercise tolerance and sudden cardiac death.
- Most often DCM symptoms occur in adults from 30 to 40
- DCM clinical manifestations are connected with:

✔ Progressing CHF
✔ Reduced heart output
✔ Ventricular and supraventricular arrhythmia
✔ Conduction disorders
✔ Thromboembolism, including pulmonary embolism and acute impaired cerebral circulation
✔ Sudden death or death caused by heart failure

- Sudden death may occur before Class III HF develops

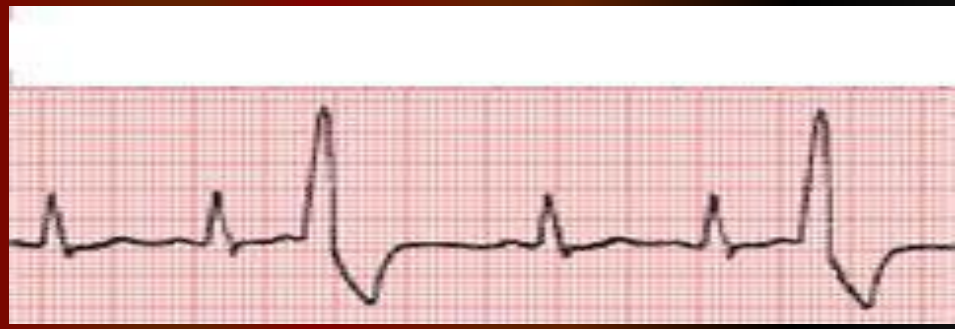
CLINICAL MANIFESTATIONS OF DCM

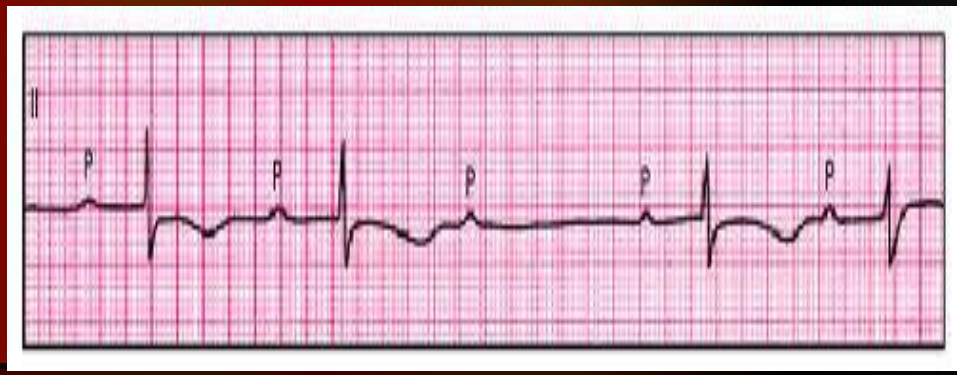
- **Physical changes**
- Inspection, palpation:
- Swollen, pulsating jugular veins
- Diffuse apical pulse shifted to the left
- Tachypnoea, orthopnoea
- Oedemas, anasarca
- Percussion: widened borders of the heart to the left, to the right
- Auscultation:
- *The heart* – **regurgitation murmurs:**
- – mitral or mitral-tricuspid
- **Gallop rhythm**
- **(Often) arrhythmia** (tachycardia, extrasystolia, atrial fibrillation)
- *The lungs* – ***rales*****:** moist, stagnant

DIAGNOSIS OF DCM

- ECG: no specific changes

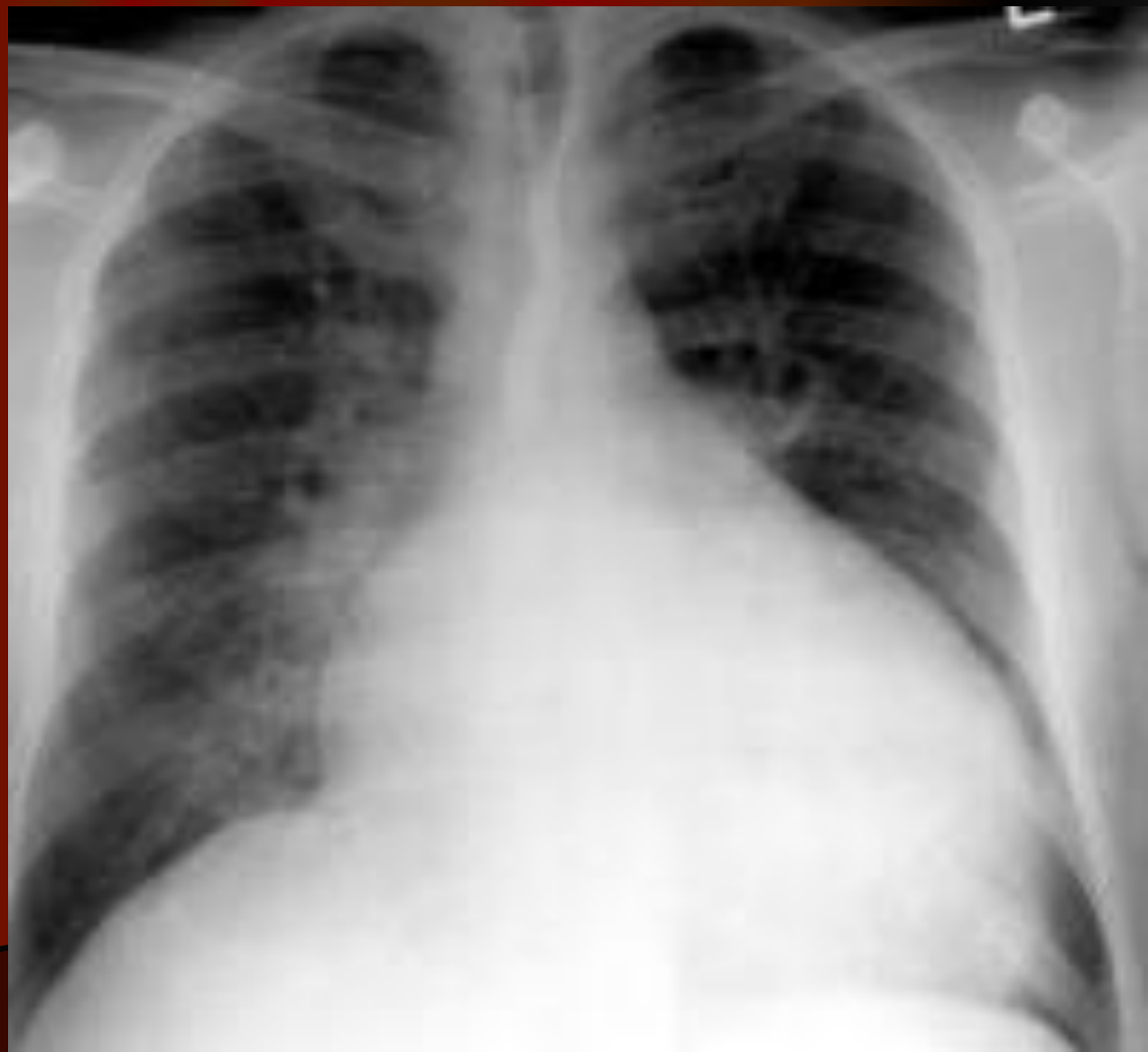
- Ventricular arrhythmia
- Atrial fibrillation
- Impaired contractility
- Complete left bundle branch block (LBBB)
- Non-specific ST – T changes
- increase in the amplitude of the R-wave between leads V1-V4





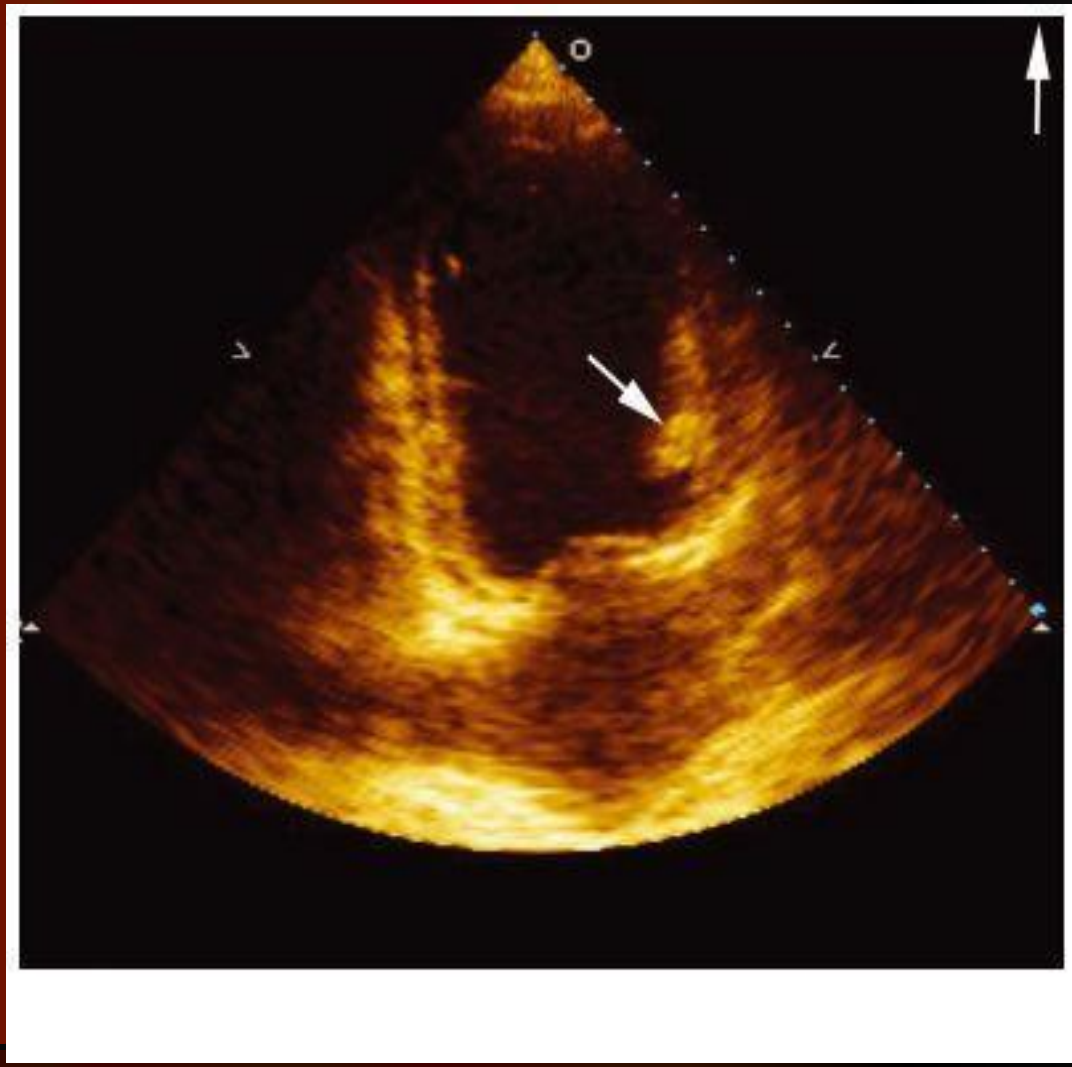
DIAGNOSIS OF DCM

- Cardiomegaly (cardiothoracic ratio > 50%)
- Pulmonary congestion



DIAGNOSIS OF DCM

- Dilation of heart cavities
- EF < 40%
- Sings of pulmonary hypertension
- Hypokinesis of walls
- No findings to support IHD, defects and other cardiac problems
- Sings of dyssynchrony of myocardium



DIAGNOSIS OF DCM

- **Radionuclide methods**
- Can be used to assess the size of heart chambers, contractility of the left and right ventricles, dyssynchrony, focal changes. Differential diagnosis with IHD. Allow to make an early diagnosis of impaired areas and take a biopsy from these areas
- **MRI and MSCT**
- 1. Differential diagnosis with other cardiomyopathies: ARVD, endocardial fibroelastosis (EFE), amyloidosis, sarcoidosis, myocarditis, between infiltrative and inflammatory CM.
- 2. Identifying patients with a high risk of sudden cardiac death (with vast areas of fibrosis).
- **Coronary ventriculography**
- To reveal intact arteries. Invasive measurement of parameters
- ***Endomyocardial biopsy***

EXCLUSION CRITERIA FOR DCM

- Systemic arterial hypertension (> 160/100 mm Hg)
- Ischaemic heart diseases (50% coronary stenosisin one or several vessels on coronary ventriculography)
- Alcohol abuse (> 40 g/day for females ,> 80 g/day for males)
- Systemic diseases of the connective tissue
- Specific diseases of pericardium
- Congenital heart defects
- Acquired heart defects
- Pulmonary heart disease

MANAGEMENT OF DCM

1. To exclude factors which may worsen dysfunction of myocardium
2. Medical treatment:

- Management of heart failure
- Treatment and prevention of arrhythmias/ sudden cardiac death
- Prevention of thromboembolism

3. Surgical treatment

MYOCARDITIS

- Inflammatory impairment of the heart muscle due to influence (direct or indirect through immune mechanisms) of a number of factors; associated with damage to mechanical and electric functions of the heart.
- The true incidence of myocarditis in population is unknown.
- A cause of sudden death.
- Clinical symptoms vary from subclinical disease to sudden death in newly developed atrial or ventricular arrhythmia, complete heart block or acute symptoms resembling myocardial infarction.
- Most researches mention prevalence of the disease in males.

ETIOLOGY OF MYOCARDITIS

- Bacteria
- Rickettsiae and Spirochaete
- Viruses
- Protozoa
- Fungi
- Parasitic diseases
- Deficiencies (hypophosphataemia, hypomagnesemia, hypocalcaemia, carnitine or selenium deficiency)
- Allergic and toxic reactions
- Action of some medications and cardiotoxic factors
- Autoimmune diseases
- Sequelae of burns, corrosions and frostbite
- Post-transplantation conditions

VIRAL INFECTION IN MYOCARDITIS

- Coxsackie of A and B groups, ECHO, A and B flu, herpes (herpes virus type 6), cytomegalovirus, Epstein-Barr virus , parvovirus B 19, coronavirus, arbovirus, hepatitis B, C, D viruses, HIV, epidemic parotitis, polio.
- The most common viral genome identified in biopsy of myocardium in European population is parvovirus B 19 and human herpes virus type 6.
- Among pathogenic bacteria, intracellular pathogens (of Chlamidia genus) have been the most significant recently.

MYOCARDITIS

THE COURSE OF THE DISEASE

- Mild: mostly focal, without cavity dilation, systolic dysfunction, potentially dangerous arrhythmias, heart failure stages 0-1.
- Moderate: focal or diffuse with initial dilation, moderate impairment of LV contractility, without malignant arrhythmia
- Severe: diffuse myocarditis with cardiomegaly, systolic dysfunction, life-threatening rhythm and conduction disorders

DIAGNOSIS OF MYOCARDITIS

1 CRITERIA OF INFLAMMATION, INFECTION:

- Fatigue, hyperthermia, accelerated ESR, leucocytosis, elevation of C-reactive protein
- Routine microbiologic and serologic reactions (positive neutralisation reaction, complement-binding reaction, haemagglutination) are of significance to make a diagnosis of non-viral myocarditis only
- Immune, histochemical study of biopsy material, PCR-guided diagnosis to confirm viral myocarditis

DIAGNOSIS OF MYOCARDITIS

2 CRITERIA OF MYOCARDIAL INVOLVEMENT:

- Clinical: cardialgia, heart palpitations, irregular heart work, HF symptoms associated with infection, allergy or other underlying disease, weakened I(II) sound, systolic murmur at the apex, widened borders of the heart
- ECG: tachycardia, bradicardia, arrhythmias, blockades, decreased voltage, repolarisiation disorders, long QT
- EchoCG: cavity dilation,< EF, hypokinesis of myocardium, thicker walls, fluid accumulation in a pericardial cavity, valve regurgitation, blood clots in the cavities
- Biochemical: elevated cardiac troponin levels, CPK-MB, LDH, level of antimyocardial antibodies (to sarcolemmal and microfibrillar proteins of cardiomyocytes)

DIAGNOSIS OF MYOCARDITIS

New York Heart Association (NYHA)

- History of infection confirmed clinically and biochemically or another cause (allergy, toxins, medications, burns, etc.)
- Sinus tachycardia
- Weakened S1
- Gallop rhythm
- Enlarged heart
- Congestive heart failure
- Pathological changes on an ECG
- Elevated serum enzyme or isoenzyme activity

MANAGEMENT OF MYOCARDITIS

1 Etiotropic treatment

- Antibacterial, antiviral, antiparasitic drugs

2 Pathogenic treatment

- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Glucocorticoids (GCs) (if severe)
- Immunosuppressive drugs (second-line therapy)

3 Symptomatic treatment

- Management of HF
- Treatment of rhythm and conduction disorders
- Prevention and treatment of thromboembolism

HEART FAILURE (HF)

- HF is a clinical syndrome characterized by typical symptoms (e.g. breathlessness, ankle swelling and fatigue) that may be accompanied by signs (e.g. elevated jugular venous pressure, pulmonary crackles and peripheral oedema) caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/or elevated intracardiac pressures at rest or during stress.

HEART FAILURE (HF)

- *A state in which the heart cannot provide sufficient cardiac output to satisfy the metabolic needs of the body*
- It is commonly termed congestive heart failure (CHF) since symptoms of increase venous pressure are often prominent

HEART FAILURE (HF)

- HF – is an imprecise term used to describe the pathological state that develops when the heart cannot maintain an adequate cardiac output or can do so only at the expense of an elevated filling pressure.
- In practice,HF may be diagnosed whenever a patient with significant heart disease develops the signs or symptoms of a low cardiac output,pulmonary congestion or systemic venous congestion.

CLASSIFICATION

- Heart failure can be classified in several ways
  1 - Acute and chronic HF
  2 – Left , right and biventricular HF
  3 - Systolic and diastolic dysfunction
  4 - Forward and backward HF
  5 - High-output HF
  6 - Functional classes (NYHA)

ACCF/AHA stages of HF

- **Stage A**: At high risk for HF but without structural heart disease or symtoms of HF
- **Stage B**: Structural heart disease but without signs or symptoms of HF
- **Stage C**: Structural heart disease with prior or current symptoms of HF
- **Stage D**: Refractory HF Requiring specialized interventions

- *ACCF/AHA guidelines, 2001*

ESC Guidelines for diagnostic and treatment of acute and chronic HF (2016)

- Definition of **heart failure** with: preserved **(HFpEF**), mid-range **(HFmrEF**) and reduced ejection fraction **(HFrEF)**

- 1) LVEF < 40% with reduced EF
- 2) LVEF – 40-49% with mid-range EF
- 3) LVEF > 50 % with preserved EF

NEW YORK HEART ASSOCIATION (NYHA) FUNCTIONAL CLASSIFICATION OF CHF

- *I class.* Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnoea or anginal pain.
- *II class.* Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dysponea, or anginal pain.
- *III class.* Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dysponea or anginal pain.
- *IV class.* Patients with cardiac disease resulting in inability to сап on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

MANAGEMENT OF HEART FAILURE (HF)

The main purposes:

1. To reduce mortality !!!
2. To relieve HF symptoms
3. To slow down HF progress
4. To improve the quality of life (QOL)
5. To reduce duration of hospital treatment
6. To improve prognosis

THE MAIN PRINCIPLES OF HF MANAGEMENT

- To reveal and exclude triggering factors
- To normalise cardiac output
- To eliminate fluid retention in the body
- To reduce peripheral tension
- To reduce sympathoadrenal effects
- To improve blood supply and metabolism of myocardium

METHODS OF HF MANAGEMENT

- **Non-medical** (changing lifestyle)
- **Pharmacotherapy** (ACE inhibitors or ARBs, beta-blockers, aldosterone antagonists, diuretics, cardiac glycosides, ivabradine, anticoagulants, antiarrhythmic drugs, statins, cardiometabolic drugs)
- **Mechanical** (thoracocentesis, paracentesis, dialysis, ultrafiltration)
- **Surgical** (pace-makers, ICD (implantable cardioverter defibrillator), coronary revascularisation, heart transplantation)

Pharmacotherapy for HF

1 DRUGS PROVED TO BE ABLE TO REDUCE MORBIDITY AND MORTALITY RATES IN CASE OF CHF EXACTLY

- used for all patients (ACE inhibitors or ARBs, beta-blockers, aldosterone antagonists);
- used under certain clinical conditions (diuretics, cardiac glycosides, ivabradine, anticoagulants);

2 DRUGS NOT INFLUENCING PROGNOSIS FOR CHF BUT RELIEVING SYMPTOMS IN CERTAIN CLINICAL SITUATIONS

(antiarrhythmic drugs, statins, calcium channel blockers (CCB), antiaggregants, cytoprotectants, vasodilators)

ACE inhibitors recommended by Russian Cardiology Society

- Enalapril 2,5×2 - 20×2
- Captopril 6,25×3 - 50×3
- Fosinopril 5×1 - 20×1
- Perindopril 2×1 - 8×1
- Lisinopril 2,5×1 - 20×1
- Ramipril 2,5×2 - 5×2
- Spirapril 3×1 - 6×1
- Trandolapril 1×1 - 4×1
- Chinapryl 5×1 - 40×1
- Zofenopril 7,5×1 - 30×1

RULES FOR ADMINISTRATION OF ACE INHIBITORS

1. **To discontinue active diuretic therapy or to reduce the dosage of diuretics within 24 h.**
2. **To discontinue or to reduce the dosage of systemic vasodilators.**
3. **Not to start treatment if BP is < 90 mm Hg, plasma K > 5.0 mmol/L, creatinine > 220 mcmol/L.**
4. **To monitor BP, plasma K, creatinine after each dose titration, then once in three months.**
5. **If GFR is 15-30% reduced the dose may remain the same; in 30-50% the dose should be decreased by two times, in >50% the drug should be discontinued**

ESC recommendations. ARBs II with proved influence on prognosis

- Candesartan from 4-8 mg daily to 32 mg daily
- Valsartan from 20-40 mg twice a day to 160 mg twice a day
- Losartan from 50 mg daily to 150 mg daily

ESC recommendations. β-blockers with proved influence on prognosis

- **Bisoprolol**
- from 1.25 mg daily to 10 mg daily
- **Carvedilol**
- from 3.125 mg twice a day to 25-50 mg twice a day
- **Metoprolol succinate**
- from 12.5 mg daily to 200 mg daily
- **Nebivolol**
- from 1.25 mg daily to 10 mg daily

Peculiarities of taking ß-blockers

- To all patients with manifestations of CHF due to IHD or DCM, if the level of EF <45%.
- If the level of EF <25%, carvedilol is preferable.
- Treatment should start after correction of hypervolemia.
- Individual titration, aiming at reaching target doses.

ESC recommendations. Aldosterone antagonists

- **Eplerenone**
- from 25 mg daily to 50 mg daily
- **Spironolactone**
- from 25 mg daily to 25-50 mg daily

ESC recommendations. Aldosterone antagonists

Contraindicated:

- K level >5.0 mmol/L, creatinine >220 mcmol/L,
- While taking other sparing diuretics, concomitant use of ACE inhibitors and ARBs II.

Treatment only monitoring potassium and creatinine levels

- Initial dosage is 12.5-25 mg (50 mg for patients not taking ACE inhibitors or ARBs).
- Therapeutic dosage is 25-75 mg (100-150 mg for patients not taking ACE inhibitors or ARBs).

IVABRADIN, a standard medication for CHF management

- **Reviewing European recommendations on HF (2012):**
- Ivabradin should be included into medical treatment for CHF of every patient with CHF Class II-IV and LVEF <35%, heart rate 72≥70 bpm, sinus rhythm.
- Heart rate has been recognised as a routine parameter determining the further management
- Heart rate <70 bpm is included into the algorithm of management of patients with CHF
- Ivabradin improves outcomes in patients with CHF

Indications for administration of diuretics:

- To eliminate clinical symptoms of fluid retention. They contribute to better exercise tolerance.
- Preventive consumption for haemodynamically stable patients disposed to hypervolemia.

Doses of diuretics during active stage of HF treatment

- **Furosemide** from 20–40 mg to 40-240 mg
- **Torasemide** from 5–10 mg to 10–100 mg
- **Hydrochlorothiazide**

  from 12.5-25 mg to 25-100 mg
- **Indapamide** from 2.5 mg to 2.5 - 5 mg
- **Spironaloctone** from 12.5-25 mg in combination with ACE inhibitors or ARBs II to 25-75 mg in combination with ACE inhibitors or ARBs II

  from 50 mg without ACE inhibitors or ARBs II to 100-150 mg without ACE inhibitors or ARBs II

TAKE CARE OF YOUR HEART !!!