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# **HEART FAILURE**

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## Definition

Cardiac insufficiency (CI) is characterized by incapability of the heart to maintain the cardiac output necessary for the body metabolism with normal or high venous return.

## Incidence

In infants: 5 – 6% of infants with cardiovascular pathology.

## Etiology

1. Pressure overload in right ventricle and left ventricle representing the obstructive lesion like.
  - a) outlets from the cavities (aortic stenosis, pulmonary stenosis, coarctation of aorta, idiopathic hypertrophic subaortic stenosis, obstructive hypertrophic cardiomyopathies).
  - b) incoming blood in the cavities (mitral stenosis, tricuspid stenosis, tricuspid atresia, cor triatrium, anomalous pulmonary venous return).
2. Volume overload of right ventricle and left ventricle representing the large left shunt, valvular regurgitation and system A – V fistula. In these children first appears dilatation and then hypertrophy.  
A considerable large left to right shunt may produce CI earlier (PDA, ASD, VSD, A-V canal etc.).
3. Alteration in the myocardial contraction:
  - a) primitive: myocarditis, primitive cardiomyopathy, endocardial fibroelastosis.
  - b) secondary: coronary circulation affection, infantile calcifying arteriopathy, myocardial infection, medication toxicity, genetic diseases (glycogen storage disorders, mucopolysaccharidosis, collagenoses, intoxication with copper, iron, metabolic and endocrine, electrolyte imbalance (hyper and hypokalemia), myxoedema, thyrotoxicosis, uremic cardiomyopathy, neurologic diseases (Duchenne, Friedrich ataxia, severe tachyarrhythmia, restrictive cardiomyopathy, septicemia, Anemia, iatrogenic CI (cardiac intervention), etc.
4. Mixed origin.

## Pathophysiology

I. Factors determining the myocardial performance.

- Cardiac frequency of heart rate – acceleratory (sympathetic) and inhibitory (parasympathetic). In tachycardia (200 b/min) CI does not worsen as a result of diastolic shortening which disturbs the ventricular filling but still ventricular ejection systole drops. Unfavorable oxygenation conditions of the heart may lead to lessening of the myocardial contractibility. In bradycardia (less than 40 – 45 b/min) CI may be only VES, so telediastolic volume which may lead to extreme dilatation of the heart which interferes with myocardial oxygenation provoking ischemia and lessening of the myocardial contractibility.
- Preload is estimated by the telediastolic volume.
- Afterload is the force which opposes the shortening of myocardial fibres.
- Myocardial contractibility is dependent on the length of myocardial fibres (Starling – Franki

law). Decrease in the myocardial contraction is due to the fall in the ejection fraction and cardiac output in ratio with an increased volume or modification at the end of diastole. Ejection fraction represents the ratio between systolic ejection volume and telediastolic volume.

### III. Adaptation mechanism in CI.

1. In CI the compensatory mechanism is by increase in the contractibility by strong stimulation of sympathetic system:
  - a) tachycardia;
  - b) ventricular dilatation;
  - c) ventricular hypertrophy;
  - d) relaxation and distensibility of the heart;
  - e) peripheral resistance – local reaction.
2. Biochemical factors.
3. Excitation – contraction coupling.
4. Adrenergic mechanism.
5. Oxygen transport by erythrocytes.
6. Pulmonary dynamics.
7. Renal mechanism.

#### Clinical manifestations:

- Direct signs: cardiomegaly, loud II sound of PA, gallop rhythm, systolic murmur due to tricuspid regurgitation or due to functional mitral regurgitation (pulsus paradoxus, alternative, tachycardia, difficulty in eating, stature deficit).
- Signs of pulmonary congestion: tachypnea, dyspnea on effort and paroxysmal nocturnal dyspnea (PND), cough, crackles, obstructive signs, cyanosis.
- Signs of venous systemic congestion: hepatomegaly, raised JVP, peripheral edema, ascitis in case of severe CI.

#### Laboratory findings:

- Chest X-ray: cardiomegaly, pulmonary arterial trunk dilatation, pulmonary venous stasis.
- ECG: non-specific findings: flat or inversed T waves and short ST segment, repolarization disturbances in precordial leads, P pulmonale, large P waves, sharp P waves in leads I, II and III, right axis deviation in V<sub>1</sub>, V<sub>2</sub>, left deviation of QRS and complex RS in V<sub>1</sub> – V<sub>2</sub>.
- Echo-caediogram: it can measure the dimensions of LV and RV. Myocardial function can be appreciated.
- Laboratory investigations: blood analysis: P, electrolytes, hemogram, cardiac enzymes.

#### Diagnosis:

It is based on clinical manifestations and laboratory reports explained earlier. CI is graded according to New York Heart Association (NYHA), 1964:

- I functional class: absence of dyspnoea and/or weakness; activities are unaffected.
- II functional class: mentioned symptoms are absent in rest, but present during normal activities.
- III functional class: symptoms are absent in rest but lessening in the capacity of activities and physical effort.
- IV functional class: symptoms are present in rest and disability for the physical activities.

### Treatment

#### General measures

- Alimentation of the infant.
- -by reducing the volume by  $\frac{2}{3}$  –  $\frac{1}{3}$  of the necessary volume for the corresponding age at short intervals. In infants the Ryle's tube aspiration is done to prevent regurgitation and aspiration. In older children the liquid is administered  $750 \text{ ml/m}^2/\text{day}$  and is increased on amelioration. Potassium rich diet is given once in 2 – 3 days (dried grapes, egg, gruel, boiled potatoes, a cup of yoghurt, cheese from cow's milk).
- The position of upper body is raised while sleeping which eases respiration.
- Humid  $\text{O}_2$  by 30 – 40% concentration is given.
- Normal temperature.
- Sedation.
- Treatment for the basic disease.
- Correction of acidosis by  $4\% \text{ Na}(\text{HCO}_3)_2$  2 – 4 ml/kg/day.
- Anticoagulants (if necessary).
- In case of arrhythmia – antiarrhythmic treatment.

#### Digoxin

Digoxin is the largely preferred in infants and children, it is administered orally and parenterally. The half-life is 1-1,5 days. Maximum effect is seen in 4 hours after administration and has 4 – 7 days of action. 20 – 25% are inactivated daily. The level of digoxin in the serum in a responsive child after 4 hours is  $3,69 \pm 0,4$  -  $2,41 \text{ mcg/L}$ . In cardiac failure the digitalizing

dose is given in the first 24 hours and then the maintaining dose is given:  $\frac{1}{4}$  at 8<sup>00</sup> and the other  $\frac{1}{4}$  at 16<sup>00</sup>. In the case of chronic cardiac insufficiency the digitalizing dose is given for the first 2 – 3 days and then continued with the maintaining dose.

- In preterm infants the digoxin is administered carefully starting from 0.005 – 0.01 mg/kg. There are various digitalizing doses according to the age:
- In preterm infants - 0.035 mg/kg/day – oral;
- In newborns – 0.05 mg/kg/day – oral;
- In 2 years – 0.05 – 0.07 mg/kg/day – oral.
- Maintaining dose is  $\frac{1}{5}$  –  $\frac{1}{4}$  -  $\frac{1}{3}$  of the digitalizing dose administered in 2 divided doses.
- Oral maintaining dose is 0.01 – 0.02 mg/kg/day divided in to two doses.

#### Reducing the effort of heart:

General measures:

- Absolute bed rest 7 – 10 days.
- No physical effort.
- 40-50% humidized O<sub>2</sub> – through mask 30 min – 2 hours till symptoms subside.
- 30° oblique decubitus position.
- Decreasing afterload.

#### Maintaining treatment:

- Hydralazine – vasodilator effect on arterial bed. Initially 0.1 – 3 mg/day in 3-4 doses and may be increased to 7 mg/kg/day.
- Captopril (Enalapril) – ACE inhibitor – oral dose – 0.3 – 1 – 5 mg/kg/day in 3 – 4 divided doses. It is administered with diuretics and ionotropics.
- Prasosin (minipress) – initial dose – 5 mcg/kg – oral.
- $\alpha$  and  $\beta$  adrenergic blockers.

#### Decreasing the volume overload:

- Less sodium diet. In older children – 1 g salt/day.
- Nr 5 or 10 diet accordingly to insufficiency.
- Liquid restriction. In IIB – III degree maximum of 40-50 ml/kg/day is recommended.
- Diuretic therapy: furosemide – 1 mg/kg if not effective then up to 2 – 3 mg/\*day. Chlorthiazide 20 - 30 mg/kg/day. Spironolactone – 1-3 mg/kg/day with the previously mentioned diuretics.
- At the same time the patient should not be manipulated unnecessarily. Sedation – Phenobarbital 3 mg/kg; droperidol – 0.25 – 0.5 ml/kg/day; phenytoin – 0.001 mg/kg i/m or 0.0015 – 0.0025 mg/kg/day – i/v.

### ACUTE PULMONARY EDEMA

It is an emergency of the left heart failure. The manifestations are due to water retention, protein in the extravascular space of the pulmonary parenchyma.

#### Pathophysiology:

In the production of acute pulmonary edema the following things take place:

- Increased pressure in pulmonary capillaries.
- Decreased plasma osmotic pressure.
- Increased vascular permeability in pulmonary circulation.
- Increased filtration surface.
- Increased pulmonary tissue capacity for the fluid accumulation.
- Disturbances of lymphatic drainage.

#### Classification of pulmonary edema (WHO, 1990):

I. Clinical forms:

- Acute interstitial pulmonary edema (cardiac asthma);
- Acute alveolar pulmonary edema;

II. According to clinical evolution:

- Fulminant, instantaneous or sudden pulmonary edema duration.

- Acute pulmonary edema – duration less than an hour, if not treated results in death.
- Slow acute pulmonary edema – till 2 hours.

III. According to X-ray findings in chest:

- Massive acute pulmonary edema.
- Infiltrative acute pulmonary edema.
- Lobular acute pulmonary edema.
- Alveolar acute pulmonary edema.

#### Clinical picture:

Dyspnoea, tachypnoea, pulmonary obstruction, orthopnoea, cough, agitation, cyanosis, pallor, transpiration, serous expectoration, hemoptysis are seen. On auscultation the respiratory sounds are diminished bilaterally, bilateral crackles, crepitations, increasing cyanosis, tachycardia, abundant frothy expectorations are observed. Diminished cardiac beats. Weak pulse. In infants the clinical picture is worse.

#### Diagnosis:

General signs: sudden ataxia, orthopnoea, severe dyspnoea, confused status. On examination: anxiety, agitation, pallor, acrocyanosis, cold sweating, tachypnea more than 40 c/min, stridor, dry cough in the first phase. Brown-red frothy sputum. Auscultative findings: diminished respiration, humid crackles, crepitations, low cardiac beats, protodiastolic gallop rhythm. ECG findings: tachycardia or severe tachydysrhythmia, cor pulmonale, modifications in ventricular myocardium. X-ray findings: “butterfly wings” appearance.

#### Treatment:

- Maintaining the airway patency.
- Semidecubitus position.
- Antisputum therapy: O<sub>2</sub> inhalation through 30 – 75% alcohol, antifomsilan, silicon, etc.
- Decreasing the venous return to the right ventricle or decreasing the pulmonary overloading.
  - BP cuff in the lower extremities every 20-30 min. in older children.
  - Rapidly acting diuretics – Lasix – 1-2 mg/kg up to 3-6 mg/kg.
  - Aminophylline 2-4-6 mg/kg – i/v.
  - To increase the osmotic pressure – 20% hypertonic solution.
  - Ganglion blockers.

#### Treatment in alveolar acute pulmonary edema:

- Aspiration and maintaining of the air way.

- Absolute rest.
- Sedation.
- Semidecubitus position.
  - O<sub>2</sub> inhalation through 30-75% alcohol or 10% antifomsilan 0.2 – 0.5 – 0.7 ml.
  - Tracheal intubation and mechanical ventilation, endotracheal 70-96% of ethyl alcohol – 1-3 ml i/v bolus.
  - BP cuff to the extremities.
  - If the systolic BP is 60 – 70 mm Hg then Dopamine 2 – 10 – 15 mg/kg/min in perfusion or noradrenaline 2 – 12 mcg/min in perfusion – maximum – 20-30 mcg/min. Prednisolone – 1-2 mg/kg i/v bolus.
  - Hemodynamic support – 100 IU/kg i/v in bolus, strophantine 0.25 – 0.5 mg i/v or sol. Digoxine 0.025% - 0.03 – 0.05 mg/kg.
  - Calming agents: diazepam 0.5% - 0.05-0.1 ml/kg i/v or 20% sol. Of sodium oxybutirate 100 mg/kg in perfusion.

#### **Bibliography:**

1. Kliegman: Nelson Textbook of Pediatrics, 18<sup>th</sup> edition. ISBN-13; 978-1-4160-2450-2457.
2. Maydannic V.G. Propedeutics of pediatrics. Kharkiv National Medical University. 2010, p.348
3. Susan M., White, Andrew J. Washington Manual TM of Pediatrics, The, 1st Edition, 2009, Lippincott Williams & Wilkins.
4. Colin D. Rudolph. Rudolchs Pediatrics, The 21 st Edition, 2003.