**LECTURE SYLLABUS**

**(General and dental medicine)**

**Cardiovascular pathophysiology**

**General physiological rules**

Basic role of pressure gradient in circulation (delta P) and resistance (peripheral resistance, R) – general condition analogous to Ohm´s law: blood flow (Q) is directly proportional to pressure gradient and inversely proportional to resistance - Q = ΔP / R

Heart muscle as a pump – main source of pressure gradients, continuous circulation (cardiac output)

Series connection of systemic and pulmonary circuit

Unidirectional blood flow

Resting and loading state

Final pressure in circulatory system results from mutual dependance of bloodstream volume, compliance of vessels and blood volume

**Hypertension**

Blood pressure measurement (mmHg, kPa), physiological regulation

Etiopathogeny – mosaic theory, risk factors – endogenous and exogenous

Classification of systemic hypertension:

* primary (essential)
* secondary (renal, renovascular, endocrinopathies, CNS disorders, circulatory disorders (aortic coarctation)

Complication, hypertension as a predisposed factor to another disturbances

Pathophysiological base of therapy

Pulmonary hypertension

**Hypotension**

Undefined range between normal and hypotensive state

Usual criterion for systemic arterial hypotension – < 100 / 65 mmHg

Constitutionally decreased blood pressure – not real disease

Idiopathic, orthostatic, secondary (diarrhea, vomiting, sweating, immobilization)

**Circulatory collapse**

Immediate total block of circulation

Causes: cardiac arrest or syncope, pulmonary thromboembolism, generalized vasodilatation

Main sign – disturbance of consciousness (resulting from reduced brain perfusion), usually blood pressure decline by 50 mmHg and more

Consequence: brain hypoxia: aerobic metabolism max. 10 – 20 sec, then anaerobic glycolysis (max. 4 – 5 min), then irreversible changes (highest sensitivity – neocortex, brain stem RF)

* Pathophysiological base of therapy; cardiopulmonary resuscitation

**Syncope**

Cardiac (AV block III ), neurogenic (strong vagal stimulation)

**Heart failure (insufficiency)**

Determinants of the heart work as a pump, preload, afterload

Left heart failure, compensation, complication (pulmonary edema, asthma cardiale)

Right heart failure, compensation, complication (peripheral edemas)

Concentric and eccentric hypertrophy of myocardium as a main chronic adaptation; consequence of volume and pressure overload, i.e. preload resp. afterload increasing

**Shock**

Definition of shock as a generalised hypoperfusion in microcircullation

Etiopathogeny: different causes and mechanisms, common pathophasiological consequence

Basic classification: hypovolemic, cardiogenic, distributional (including special types)

Hypovolemic shock – decrease of effective volume of circulating fluids: external or internal bleeding, burns, nephrotic syndrome, vomiting, diarrhoea, crush syndrome, peritonitis)

Cardiogenic shock – heart failure as a pump: acute MI, malignant arrhytmias, myocarditis, pericard disturbances (tamponade), massive pulmonary embolism

Distributional (also vascular, low-resistant) – generalised vasodilatation: septic shock caused by bacterial endotoxins, anaphylactic, neurogenic

Reversible and irreversible phase; pathophysiological base of therapy

**Atherosclerosis**

Etiology and pathogeny; risk factors

Consequences of atherosclerotic process in cardiovascular pathophysiology; arteriosclerosis

**Ichemic heart disease**

Etiopathogeny, risk factors

Acute and chronic manifestation of IHD

Acute coronary syndrome – classification (STEMI, NSTEMI, unstable angina, ECG signs

Pathophysiological base of therapy

Complication of acute myocardial infarction (cardiogenic shock, ventricular tachyarrhythmias, tamponade)

**Ischemic – reperfusion damage**

2 main areas:

1) Reperfusion in occlusion (spasm) of coronary artery

* spontaneous – opening of collaterals, thrombus recanalization, spasms relaxation
* therapeutical – thrombolysis or PTCA (percutanneous transluminal coronary angioplastic)

2) Reperfusion after cardiac arrest

Pathogeny of myocardial damage: calcium and oxygen paradox

Pathophysiological base of therapy, prevention (importance of hypothermia)

**Compartment syndrome**

Symptoms resulting from pressure increase in anatomically closed space (compartment); it leads to vascular occlusion and local ischemia

**Heart defects**

Inborn (developmental) and acquired changes in cardial morphology; affecting the pumping function of heart (importance of unidirectional bloodstream and pressure gradient!)

Functional consequences: left to right circulatory shunt – increasing blood amount in pulmonary circulation, right to left shunt – mixture of venous and arterial blood (increasing blood amount in systemic circulation)

Inborn heart defects: with and without cyanosis

Valvular stenosis – change of pulse volume, congestion before a barrier

Valvular insufficiency – blood regurgitation (inverse direction of bloodstream)

**Arrhythmias (dysrhythmias)**

Electrical instability of myocardium; reentry mechanism

Basic classification, etiopathogeny, ECG markers

**Other pathogenetic mechanisms**

Hyperemia – active, passive, inflammatory

Cyanosis – peripheral, central

Edema – local, generalized