# ABSTRACT BOOK

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## for medical students and young doctors

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index). Echocardiography was considered as reference method and performed in M- and B-modes. Criterion of LVH was left ventricular myocardial mass indexing by BSA >125 g/m$^2$.

**Results**: LVH was found in 17 (85%) patients by echocardiography criterion. LVH was determined by Sokolow-Lyon voltage index in 4 (20%) patients, according to Cornell voltage index - in 4 (20%) patients. Only for 1 case there was agreement on both Sokolow-Lyon and Cornell voltage indexes. LVH in all patients with positive Sokolow-Lyon and Cornell voltage indexes was confirmed by echocardiography (i.e., 100% of specificity). For Sokolow-Lyon voltage index in 13 cases LVH was undiagnosed (i.e., 24% of sensitivity). There were same results for Cornell voltage index. LVH by Cornell product index was determined in 12 (65%) patients. In 4 cases there were coincidences with Cornell voltage index and in 4 cases - with Sokolow-Lyon voltage index. Moreover, comparing with results of echocardiography, in case of Cornell product index the coincidence of LVH was 14 (100% of specificity). At same time for Cornell product index in 3 cases LVH was undiagnosed (i.e., 82% of sensitivity).

**Conclusion.** These data suggest that both Sokolow-Lyon and Cornell voltage indexes are very specific methods, but they may recognize only pronounced left ventricular hypertrophy. Results obtained by Cornell product index not significantly differ from the ultrasound data, showing acceptable sensitivity and specificity.

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## THE ROLE OF NEUROENDOCRINE DISORDERS IN PATIENTS WITH VIBRATIONAL DISEASE IN COMBINATION WITH HYPERTENSION

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**Introduction.** In the structure of professional diseases vibration disease occupies one of the first places. According to modern ideas, vibration disease is a systemic angiotrophoneurozis in its pathogenesis there are disoders of universal homeostatic mechanisms - neurohumoral regulation, microcirculation, tissue and cellular metabolism [Sukharevskaya T.M. 2010]. The clinical picture of vibration disease is characterized by polimorphal symptoms with involvement in the pathological process the various stages of homeostasis of many organs and systems, which in progression has a tendency to generalization. First of all, peripheral blood vessels are changed. Vibratory stimulation of vascular smooth muscle cells leads to changes in hemodynamics and angiospazm in the microcirculation. As a result there is a development of arterial hypertension (AH) and coronary heart disease, which in their turn increase the course of the underlying disease and worse the prognosis in general.

**Results.** At the present stage of study vibration disease covers the following links of pathogenesis, as activation of proinflammatory cytokines. It also draws attention to dynamics of biometal, depending on the severity of vibration disease against concomitant hypertension. This direction of medical researches is perspective and actual, as the receved data are enough contradictory and require a further study.

Biological effects of vibration caused by both their direct effects on cells and subcellular structures, and indirectly - through neurohumoral and neyroreflektorni mechanisms that appear on the immune system. An important effect of cytokines is that they make the connection between systems of the body (immune, nervous, endocrine, cardiovascular) and used for their involvement in regulatory protection. The blood plasma of patients with hypertension, regardless of its etiology, the maintenance of proinflammatory cytokines. There is an assumption that proinflammatory cytokines determine the pathological processes of vascular remodeling by regulating the degree of apoptosis. Tumor necrosis factor (TNF) - causes the activation of free radicals that can cause intensification and strengthening the processes of apoptosis and inactivation of vascular endothelial nitric oxide in the endothelium.

**Conclusions**. Cytokines can increase the density of intracellular membranes of neutrophils, activating the oxidase, enzymes peroxidation and therefore lead to a rapid, but reverse, lowering intracellular calcium.

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## CHANGES IN THE OXIDATIVE STRESS INDICES IN DIFFERENT TYPES OF ACUTE HEART FAILURE

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**Introduction.** Oxidative stress (OS) is an important pathogenetic component of acute heart failure (AHF). The increase in the induction of active forms of oxygen, intensification of peroxide lipids oxidation (POL) and lower levels of antioxidant protection is tightly connected by direct and indirect linkages to the neurohumoral and immuno-inflammatory activation, and is associated with the destructive and metabolic disorders, which are specific to the AHF.

The **aim** and objectives of the study: increase of efficiency of diagnostics of acute cardiac insufficiency on the basis of the research intensity of oxidative stress in patients in all clinical types.

**Material and methods.** 50 patients with ischemic heart disease, accompanying by the AHF were examined. The control group included 10 patients with ischemic heart disease and heart failure I FC (NYHA).

**Results**:In the total group of patients with AHF the level of malondialdehyde (MDA) was 10,94 ± 1,09 mmol / l, dienal conjugate (DC) - 56,27 ± of 9.9 mmol / l, catalase 2,39 ± 0, 29 usd. / l, ceruloplasmin (CP) - 120,52 ± 11,6 c.u. / l, that in 2,5, 2,2, a 1.3, 1.6 times more than the value of the control ($p < 0.01$, $p < 0.01$, $p < 0.05$, $p < 0.05$ respectively). It was noticed that levels of MDA and DC increased more than in 2 times, but the stage of the compensatory activation of catalase and the CP was much lower.

**Conclusion:** Thus, increased free radical activity and the processes of peroxidation at AHF was not balanced by an adequate reaction of the antioxidant system.