ROLE OF HEART DYSSYNCHRONY IN DEVELOPMENT AND PROGRESSION OF CHRONIC HEART FAILURE. SURGICAL METHODS AND TREATMENT OF HEART DYSSYNCHRONY

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Chronic heart failure is a widespread disease with a progressive course and poor prognosis. Remodeling of a left ventricle (LV) myocardium, insufficient formation and the use of power substrata and heart dyssynchrony (HD) are the main causes of chronic heart failure (CHF) in patients with coronary heart disease.The actual problem is the treatment of patients with impaired intraventricular conduction, left ventricular systolic dysfunction and severe stenosis of coronary atherosclerosis. Revasculization for a myocardium can be one of the most effective methods of restoration of contractile abilities of a myocardium at the expense of influence on the basic pathogenetic mechanisms of CHF. This review examines the role of myocardial dyssynchrony in development of CHF, diagnostic criteria, as well as the possibility of surgical treatment.

Keywords: chronic heart failure, heart dyssynchrony, echocardiography, coronary revascularization

According to the statistics the prevalence of CHF in the population is 7% [18], the prevalence of CHF functional class (FC) I-IV in the European part of Russia – 12,3%, severe CHF, corresponding to FC III-IV, occurs in 2,3% of cases [12]. The annual mortality in patients with symptomatic heart failure is 12% that is up to 612 thousand patients with CHF die in the Russian Federation a year [6]. Meanwhile, sudden cardiac death (SCD) reaches 50% of the total [4].

Heart dyssynchrony (DS) is one of the main causes of CHF [15]. DS is the dissociation rate of its chambers and/or segments of the myocardium, due to violations of the pulse, which leads to the decrease of the pump function of the heart and increase of myocardial energy consumption. Thus, DC promotes the development and progression of heart failure [5]. In 15% of the patients with CHF [17], the pathological process causes a decrease in myocardial contractility and impaired cardiac conduction system [10].

The most common disorders of impulse conduction in the cardiac conduction system are the blockade of the left bundle branch block (LBBB) – from 25 to 36%, and less – due to the blockade of the right bundle branch block (4-6%) violations of inter-and intraventricular conduction, that causes in its turn the extension of the QRS complex on the ECG [14].

According to C. Wiggers [24], the abnormal ventricular apex activation during the right ventricle (RV) stimulation, as during LBBB leads to depressed left ventricular (LV) function and to its structural changes as well. The modified sequence of electrical activation of the ventricles during LBBB leads to mechanical dyssynchrony of ventricular and cardiac cycle with negative effect on hemodynamics. Due to the fact that the electrical impulse spreads from the right to the left (eccentric ventricles activation), directly on the myocardium, and not according to His-Purkinje system, it reaches the left ventricle later, and its propagation velocity is less than normal. As a result, mechanical left ventricular systole significantly lags behind the RV systole and becomes more prolonged. Excitation of the posterolateral wall of the left ventricle occurs later than the interventricular septum (IVS), which significantly reduces left ventricular systolic hemodynamic performance because of asynchrony of left ventricular wall contraction. So, the phase of pre-exile of izovolumic left ventricular contraction and relaxation takes place. As a result, left ventricular diastolic pressure significantly reduces, which interferes normal left ventricular filling. The phase of fast and slow (atrial systole) LV filling superimpose on each other, thus reducing the contribution of atrial systole. Disunited activation of the mitral valve papillary muscles leads to late diastolic or presystolic regurgitation, which is not related to the anatomical substrate and is reversible [23].

The extension of QRS interval ≥ 120 ms is one of ECG signs of DS [5]. Evaluation of this parameter according to the electrical mechanical DC marker shows its presence in 15% of all the patients with CHF [17]. For the patients with severe CHF, the incidence of widened QRS complex increases by more than twice and is revealed in more than 30% of all the patients [10].

DS is subdivided into atrioventricular, interventricular, intraventricular, and atrial [2]. Mechanical heart DS cannot exist without its visualization on ECG. In such cases, echocardiography is the primary method of dyssynchrony identification [16].

The main DS indicators on the Echocardiography are the following: the presence of presystolic mitral regurgitation, delayed activation of the poster lateral left ventricle wall in comparison with the interventricular septum (normally less than 30 ms), interventricular

mechanical delay (the difference in the duration of asynchronous and isometric phase of LV contraction in combination with the time of induction and isometric contraction of the pulmonary artery (normally no more than 40 ms)), delay of pre-exile from the aorta (the beginning of *Q* wave on ECG till aortic valve opening (normally less than 140 ms)) [11].

Coronary revascularization, particularly coronary artery bypass grafting (CABG), is one of the most appropriate methods of contractile synchrony recovery and infarction functionality [3].

The indications for revascularization in patients with heart failure of ischemic etiology are unstable angina pectoris and severe stenosis of the coronary arteries [9]. The risk of death is increased, ranging from 5 to 30%. Treatment of heart failure of ischemic genesis without unstable angina pectoris is a problem, taking into account the lack of randomized controlled investigations in this sample of patients. It is necessary to include the assessment of viable myocardium in the plan of survey of patients with heart failure and coronary artery disease. The improvement of LV function and survival of patients with ischemic but viable myocardium who underwent revascularization were revealed during several prospective and retrospective studies, as well as Meta-analyzes [1]. In the absence of viable myocardium revascularization is ineffective, and it is necessary to exclude surgical treatment, associated with high risk. In patients with severe left ventricular dilatation the probability of ejection fraction improvement is low, even in the presence of viable myocardium. The effectiveness of myocardial revascularization in combination with surgical ventricular reconstruction in order to reverse the development of its remodeling has been examined in several randomized studies [13]. The aim of surgical reconstruction is the removal of scar tissue and the restoration of physiological LV volume and shape. The STICH study compared the efficacy of CABG separately and in combination with surgical ventricular reconstruction in the patients with ejection fraction $\leq$ 35%. The frequency of the primary endpoint (death from any cause or hospitalization because of heart disease) did not differ between groups. However, the combined intervention resulted in the reduction of end systolic volume index of the left ventricle to 16 ml/m^2 (19%), which was more than after CABG, but less than in previous observational studies. The latter fact allows us to express concern about the prevalence of surgical procedures for reconstruction, which took place in this randomized study [7]. The feasibility of this intervention is necessary to evaluate individually on the basis of symptoms (signs of heart failure should be more severe than unstable angina pectoris), measurement of LV volumes, and the extent of scarring in the myocardium.

The choice between CABG and (Percutaneous coronary revascularization) PCI should be based on careful analysis of the anatomy of coronary arteries, the expected completeness of revascularization, accompanying diseases, and lesions of the heart valves [22]. The results of some studies have shown that CABG is superior to PCI [21]. The risk of SCD, despite revascularization remains high in many patients with coronary artery disease and reduced LV function, so it is necessary to assess carefully the feasibility of cardioverter defibrillator implantation [8].

The investigations of Research Institute of Cardiology named after A.L. Myasnikov convincingly show that successful revascularization in patients with CHF and the presence of viable myocardium can significantly improve the course of the disease [20]. In patients with myocardial revascularization a reduction in the frequency of the complex QRS expansion $\geq$ 120 ms is revealed to 31%, presystolic mitral regurgitation to 16,5%, interventricular mechanical delay – 14,5%, delayed activation of the posterolateral wall to 20,1% and the delay of pre-exile from the aorta to 19,3%. These changes signify the improvement of myocardial synchrony provided improved myocardial blood flow because of revascularization. It is possible by reducing the mass of stunned myocardium and energy and conductivity improvement, because other positive changes associated with revascularization usually develop later [11, 19].

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