The dynamic sports as running and swimming generate eccentric left ventricular hypertrophy (LVH) similar to the diastolic overload of the left ventricle. There were already described two morphological patterns of LVH in trained athletes – strength-trained heart and endurance-trained heart [1], [2]. In this study we tried to assess some electrical features in the LVH caused by swimming exercise comparing to the pathological pattern of LVH from patients with cardiac ischemia.

We investigated by echocardiography (ECHO), electrocardiography (ECG) and vectorcardiography (VCG) 15 elite swimmers (group 1) and 30 patients with LVH caused by ischemic heart disease (group 2). We calculated VCG parameters of the QRS loop as the spatial perimeter (PQRS), the spatial surface (SQRS), the speed of inscription (VQRS), and similar parameters of ST-T loop. We also examined a group of 20 normal subjects for comparing the VCG parameters values. ECHO revealed eccentric LVH for all subjects. In group 1 we found moderate LVH, with mean diastolic left ventricle diameter (DD) about 52mm, and the thickness of the interventricular septum (IVS) or posterior wall (PW) at the superior normal values limit (10 – 10.4mm); the left ventricular mass (LVM) was moderate increased (120.5g/m²). In group 2 the eccentric LVH had significant increased LVM (280g/m²) by elevated values of DD (65.5mm), IVS and PW (13mm). The classical ECG revealed variable LVH criteria: in group 1 LVH was expressed by indirect signs in chest leads 1-2 and in group 2 the classical pattern of LVH was found in 69%. VCG in Frank system revealed different electrical behaviour in our groups. We found elevated values for PQRS and for SQRS by increasing amplitudes of the QRS vectors in both groups. PQRS, SQRS were mean correlated with LVM only in group 2 (Pearson correlation index: 0.5). Like in a previous study, we did not find any correlations in group 1 [3]. We also found an increased speed of inscription of the QRS and ST-T loops in group 1, comparing to the normal findings and to the group 2.

VCG results could suggest an improvement in the impulse conduction in the physiological hypertrophied myocardium comparing to the decreased conduction during the depolarization and repolarization in pathological LVH caused by ischemia. Our study emphasise a different electrical profile of the eccentric LVH in trained swimmers comparing to pathology better investigated by VCG than by ECG and propose to add these particular VCG findings to the features of the athlete’s heart syndrome [4].

References


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