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ORIGINAL ARTICLE

The influence of water-based training on arrhythmia in patients with stable coronary artery disease and preserved left ventricular function

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Abstract

Background: *Water immersion may cause adverse cardiovascular events, including arrhythmias in patients with damaged cardiac muscle, e.g. with cardiac failure. So far, there have been rather few reports on arrhythmia induced by water training in patients with coronary artery disease (CAD). The aim of the study was to assess the influence of exercise training in moderately cold water (28–30°C) on arrhythmia and physical capacity in stable CAD patients with preserved left ventricular (LV) function.*

Methods: *Sixty-two post-myocardial infarction male patients, mean age 50.9 ± 7.9 years, participated in 16 water-based trainings (WBT), which lasted 55-min, twice a week in water at 28–30°C. Each subject underwent 24 h Holter on-land monitoring (Holter-24) once during the study and twice in-water Holter monitoring (Holter-W) during WBT. Before and after WBT cardiopulmonary exercise test (CPET) was performed. The following parameters were analyzed: peak oxygen consumption (peak VO_2), mean number of ventricular ectopic beats (VEBs) and supraventricular ectopic beats (SVEBs) during Holter-24 and Holter-W, the percentage of men who developed arrhythmia during CPET vs. Holter-24 and vs. Holter-W.*

Results: *WBT significantly improved patients' physical capacity, and more often provoked arrhythmia, mainly SVEBs, than CPET or daily activity assessed during Holter-24. During WBT 58% men developed VEBs and 62% SVEBs.*

Conclusions: *1. WBT provoked arrhythmias significantly more often than did CPET and normal daily activity. 2. Owing to WBT patients improved their physical capacity which was still maintained at 1-year follow-up.* (Cardiol J 2016; 23, 1: 93–99)

Key words: coronary artery disease, rehabilitation, water-based exercises, arrhythmias

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Introduction

Swimming is very popular in the general population and most patients with coronary artery disease (CAD) and after myocardial infarction (MI) wish to continue this activity but many physicians are reluctant to allow cardiac patients to practice water activities, especially in cold water, and particularly, if left ventricular (LV) function is impaired.

To date, there have been only limited data in very small and heterogeneous groups on the effects of cold water immersion on heart rhythm disturbances in cardiac patients. Schmid et al. [1] showed that during water immersion and swimming in patients with congestive heart failure (CHF) ventricular ectopic beats (VEBs) increased significantly in cold (22°C) water but not in CAD patients with preserved LV function. The definition of cold water, however, is much more difficult. Water temperatures between 32°C and 34°C are in general perceived as warm, whereas temperatures between 30°C and 32°C are initially perceived as cold with the sensation passing within 3–5 min and becoming comfortable thereafter for at least 1 h [1, 2]. Water immersion at neutral (35 ± 0.5°C) and cold temperatures activates different regulatory and effector mechanisms. Thermoneutral water immersion strengthens the influence of the parasympathetic nervous system by the stimulation of baroreceptors which induces bradycardia, a decrease in blood pressure and a drop in systemic vascular resistance [1, 3]. Moreover, central blood volume, mean stroke volume and mean cardiac output increase. Immersion in cold water tends to overcome this effect by increasing the sympathetic tone which is mediated mainly by thermoreceptors, activated by the rapid fall in skin temperature [1, 4]. Exposure to cold water leads to enhanced sympathetic activity which is further accentuated by exercise and is able to provoke especially ventricular arrhythmias [1, 5].

The aim of this study was to assess the influence of exercise training in moderately cold water (28–30°C) on arrhythmia and physical capacity in CAD patients with preserved LV function.

Methods

The study comprised 62 consecutive post-MI male patients, aged 50.9 ± 7.9 years, who were referred to the second phase of comprehensive cardiac rehabilitation in water (water-based training [WBT]). Inclusion criteria were: age < 65 years, sinus rhythm, stable CAD, preserved LV function (ejection fraction > 50%), clinical stability for at least 2 weeks prior to entry to the study plus optimal and stable medical treatment, the patient's willingness to comply with the proposed training program. Exclusion criteria were: unstable angina, CHF, uncontrolled hypertension, valvular heart disease, left bundle branch block, impaired renal or hepatic function. The study protocol was approved by the Institutional Ethics Committee on Human Research, and each participant gave their written informed consent.

Study protocol

All patients, meeting inclusion criteria mentioned above, participated in the WBT program which included 16 trainings, twice a week, 55 min each, in moderately cold water at 28–30°C. The cardiopulmonary exercise test (CPET) was performed before and after ending WBT, then 6 and 12 months after its completion.

Moreover, during the study once 24 h Holter electrocardiogram (ECG) on-land monitoring (Holter-24) and twice, at entry and at the end of WBT, in-water Holter ECG monitoring (Holter-W) with a waterproof pack were performed.

To be able to assess the incidence of arrhythmic events the two periods of 55 min and 40 min from Holter-24 recordings adjusted for the time with swimming exercise were chosen and compared with those from Holter-W.

The mean number of VEBs and supraventricular ectopic beats (SVEBs) as well as the percentage of men who developed arrhythmia during CPET vs. Holter-24 and vs. Holter-W were evaluated.

Cardiopulmonary exercise test

Each subject performed a symptom limited CPET according to the modified Bruce protocol. The test was performed using a Schiller treadmill (Carrollton, USA) which was connected to a computerized breath-by-breath spiroergometry system (ZAN 600, Zan Messgeräte GmbH, Germany). As described previously, oxygen consumption (VO_2) was measured continuously using breath-by-breath analysis and used an index of exercise capacity [6]. Peak VO_2 was defined as the highest oxygen uptake level achieved during the final 30 s of CPET. The formula used for the prediction of VO_2 was the Wasserman standard calculation, which incorporated sex, age, height and weight of the subject and is valid for patients aged over 20 years. A 12-lead ECG and heart rate (HR) were recorded continuously at rest, during the CPET and during recovery until HR, ECG and VO_2 returned to the baseline value. Blood pressure (mm Hg) was measured manually every 2 min using a sphygmomanometer. Subjects were encouraged to exercise until they reached a self-determined limit of their functional capacity (perceived exertion or dyspnea) or until the physician terminated the test according to the European Society of Cardiology guidelines [7].

The following parameters were analyzed: peak VO_2 (mL/kg/min), exercise duration (min), walking distance (meters), double product (mm Hg/min × 100), e.g., the product of HR and systolic blood pressure, at rest and at peak effort. Moreover, the incidence of arrhythmic events and the percentage of patients who developed arrhythmia were assessed.

Water-based training

The WBT program included 16 supervised sessions, twice a week, at water temperature 28–30°C. WBT was conducted in mid-afternoon, 3 h postprandially, in groups of 8 subjects each. Patients spent a mean time of 55 min in water. Each training unit started with a slow water immersion lasting ca. 10 min and then patients underwent swimming training for 40 min, which consisted of three parts: part one — a 10-min of warm up; part two (main) — a 20-min training including exercises improving strength and endurance of the trunk, upper and lower extremity muscles, as well as free swimming with particular attention paid to breathing; part three — a 10-min period of calm-down and relaxing exercises. The limit of training HR was calculated as the sum of resting HR and 60–80% of HR reserve, i.e., the difference between maximal HR at peak CPET and HR at rest.

Each subject was informed about his HR limit and how to measure HR as self-control during training. HR recorded during Holter-W allowed training intensity to be adjusted. WBT was accompanied by music as an additional element of therapy.

Holter ECG recording during water-based training

A novel and unique device was designed to enable ECG to be registered in water. Aria recorder Del Mar Reynolds (USA) was put in a water-proof box from which 4 cables went through water-tight holes to 4 electrodes. This made it possible to monitor ECG and count arrhythmic events during the whole training unit (55 min) including water immersion and swimming exercise (40 min).

Table 1. Clinical characteristics of the study group.

Age [years]	50.9 ± 7.9
Time after MI to rehabilitation [days]	66.9 ± 50.5
Family history	16 (25.8%)
Smoking history	36 (58.1%)
Hypertension	37 (59.7%)
Hyperlipidemia	53 (85.5%)
Body mass index [kg/m^2]	27.4 ± 2.9
Diabetes mellitus	5 (8.1%)
LVEF [%]	57.8 ± 12.6
Medications:	
β-blocker	61 (98.4%)
ACEI	53 (85.5%)
Statins	61 (98.4%)
Clopidogrel	56 (90.3%)
Aspirin	62 (100%)

Data are expressed as mean ± standard deviation or number (percentage); MI — myocardial infarction; LVEF — left ventricular ejection fraction; ACE-I — angiotensin-converting enzyme inhibitor

Statistical analysis

Statistical analysis was performed using SAS statistical software (version 8.2; Cary NC, USA). All data were expressed as mean ± standard deviation. Student's t-test for matched pairs was used to compare the parameters of a continuous type, when the distribution of variables did not differ significantly from the normal distribution. When it did, a non-parametric rank test was used. In order to assess the differences of categorized parameters χ^2 test was used. A p value of < 0.05 was considered statistically significant.

Results

The clinical characteristics of the patients are listed in Table 1. The majority of patients had hypertension, smoking and hyperlipidemia. Most of them were overweight and had standard medical therapy. The doses of medication were unchanged during the study period. All patients had preserved LV systolic function.

Owing to WBT, patients improved their physical capacity. This result was maintained 1 year after training completion. After WBT, peak VO_2 increased by 15.3% ($p < 0.05$) just after training, by 17.5% after 6 months ($p < 0.01$) and by 16.7% after 12 months ($p < 0.05$) (Fig. 1).

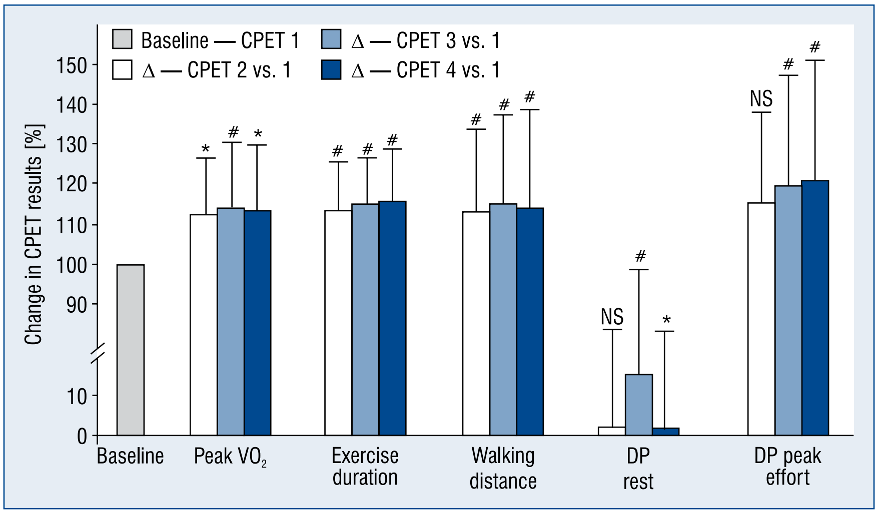

Figure 1. Changes in the results of cardiopulmonary exercise test (CPET) just after water-based training (WBT), 6 and 12 months after ending the training program. Percent changes from the results of the baseline CPET 1 are plotted. All values are mean ± standard deviation; *p < 0.05; #p < 0.01 vs. baseline; CPET 2 — exercise test performed immediately after ending the training program; CPET 3, CPET 4 — exercise tests performed after 6 and 12 months, respectively; peak VO_2 — peak oxygen consumption; DP — double product [mm Hg/min], i.e. product of heart rate and systolic blood pressure.

Similarly, WBT caused a significant improvement in exercise duration, walking distance and double product at peak effort and these effects maintained until 1 year. None of the patients complained of chest pain and no signs of ischemia on ECG were recorded during initial CPET and those performed subsequently.

Mean numbers of VEBs recorded in water vs. on land during 40 min (3.6 ± 0.17 vs. 4.8 ± 0.22, p = NS) and 55 min (4.93 ± 0.26 vs. 6.92 ± 0.32, p = NS) did not differ significantly (Fig. 2). Of note, mean number of SVEBs registered on Holter-W vs. Holter-24 during 40 min (2.8 ± 0.12 vs. 0.4 ± ± 0.02, p < 0.05, respectively) and 55 min (3.63 ± ± 0.15 vs. 0.7 ± 10.03, p < 0.05, respectively) was significantly higher in water (Fig. 2). No complex arrhythmia and signs of ischemia were found.

During WBT, 36 (58%) patients developed VEBs and 39 (62%) SVEBs. WBT provoked arrhythmias significantly more often than CPET: VEBs (p < 0.05), SVEBs (p < 0.01) and normal daily activity registered on Holter-24: VEBs (p < 0.01), SVEBs (p < 0.01) (Fig. 3).

Discussion

To the best of our knowledge, this study was the first to assess the influence of moderately cold water-based exercises on arrhythmia in a relatively large and homogenous group of CAD patients with preserved LV function. Swimming is one of the most popular form of physical activity not only in healthy people but also in patients with established cardiovascular diseases. Moreover, there is growing use of water-based exercises in rehabilitation programs in patients with CAD. An important aspect of swimming in CAD patients is, however, the occurrence of symptoms and signs of ischemia on ECG during this type of training. Immersion in water increases venous return, which in turn may increase LV workload and myocardial oxygen demand [3, 8]. Although none of our CAD patients had angina or ECG evidence of ischemia, they were more prone to develop arrhythmia. We found that during WBT performed at water temperature 28–30°C 58% patients developed VEBs and 62% SVEBs. No complex forms of arrhythmia were recorded. Mean numbers of VEBs recorded in water vs. on land did not differ significantly, but the mean number of SVEBs was significantly higher in water. It is noteworthy that WBT provoked arrhythmia significantly more often than CPET and normal daily activity. Moreover, water immersion itself generated arrhythmic events which were further increased during the remaining parts of the training unit. A possible explanation for this effect is that water immersion and horizontal body position cause a greater venous return and higher preload to the right and left heart [1, 3, 8]. This shift in blood volume into the thoracic cavity increases

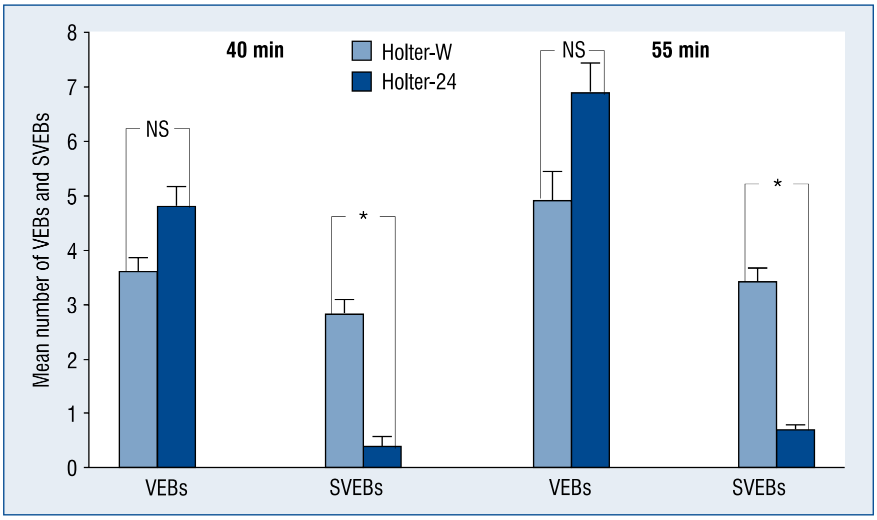

Figure 2. Influence of swimming exercise and daily activity on arrhythmic events registered on Holter electrocardiogram (ECG) in patients studied. The mean number of ventricular ectopic beats (VEBs) and supraventricular ectopic beats (SVEBs) registered on Holter ECG during 40 min and 55 min in water (Holter-W) and on land (Holter-24) monitoring are plotted. See 'Methods' section for more details. All values are mean ± standard deviation; NS — non significant; *$p < 0.05$.

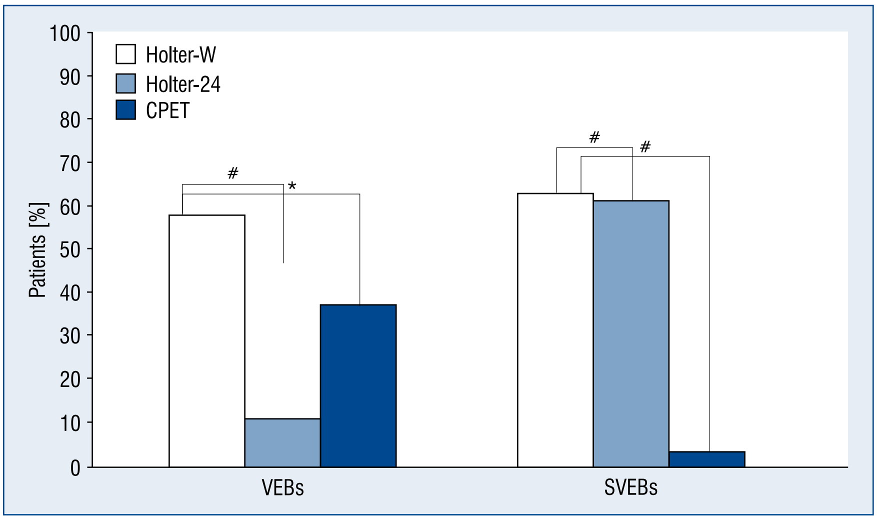

Figure 3. Occurrence of arrhythmic events during swimming exercise, daily activity and cardiopulmonary exercise test (CPET) in patients studied in percent. The percentage of patients who developed arrhythmia registered during Holter ECG monitoring in water (Holter-W) vs. on land (Holter-24) vs. CPET are plotted. See 'Methods' section and Figure 2 for more details; *$p < 0.05$; #$p < 0.01$; VEBs — ventricular ectopic beats; SVEBs — supraventricular ectopic beats.

end-diastolic pressure and volume not only in the ventricles but also in the atria. We cannot exclude that atrial overload might have been an arrhythmogenic trigger in our patients.

It should be mentioned that our classical, comparative statistical analysis did not contain assessment of spontaneity, quantity and quality of arrhythmia variability. Recently, these parameters changed the definition of antiarrhythmic and proarrhythmic effects of various drugs [9]. Proarrhythmia should be considered if antiarrhythmic therapy induces unexpected and sometimes fatal reactions by either producing new symptomatic arrhythmias or by aggravating existing arrhythmias. Fortunately, taking into account these new criteria in our patients neither water immersion nor water-based exercise therapy were associated with a proarrhythmic side effects.

Of note, exposure to cold water is thought to enhance sympathetic activity, which may further increase cardiac load and provoke arrhythmias, especially of ventricular origin [2, 4, 5].

Schmid et al. [1] compared arrhythmic effects of cold water (22°C) in a rather small group of 12 CHF patients and 10 CAD patients but with preserved LV function. In contrast to CAD patients, in CHF patients the number of VEBs recorded during a 30-min stay in water was significantly higher than that recorded during a 30-min mean period from Holter-24 on land. As mentioned previously, in our study VEBs were generated in significantly more patients during WBT than during CPET or normal daily activity recorded on Holter-24. But, they were only single VEBs. Similarly, in the study by Schmid et al. [1] no complex arrhythmia was observed.

The authors of several papers reported that arrhythmia during swimming could result from exercise-induced ischemia, and suggested exercise stress test and Holter-24 should be performed before starting WBT [1, 8, 10]. It should be emphasized that in our study, all patients had stable CAD at entry confirmed by CPET and Holter-24 and none of them had ischemic symptoms during swimming. Moreover, all of them significantly improved exercise capacity after WBT and this effect was still observed at 1 year follow-up.

Similar results were reported by other authors in groups of patients with CHF and CAD with preserved LV function [1, 11–13]. In addition, our previous paper comparing patients exercising in water vs. on land reported a significant improvement of peak VO_2 in both groups [14]. Interestingly, in the majority of our patients the rate of SVEBs was significantly higher than VEBs during water-based exercises than during daily activity or exercise stress test. As mentioned previously, none of them experienced ischemia. Thus, one explanation might be that water immersion resulted in cardiac, especially atrial, overload. On the other hand, similarly to some authors, we cannot exclude that enhanced sympathetic activity caused by immersion at water temperature below body temperature but not ischemia itself provoke arrhythmias, predominantly SVEBs in our patients [4, 5, 15, 16].

As it was described in 'Introduction', water of a temperature below 30°C is initially perceived as cold. It is the temperature in open waters. Since swimming is a popular leisure-time activity also in patients with cardiovascular diseases, it should be recommended with caution, especially in those with CHF or immediately after MI.

Based on our findings, swimming in moderately cold water seems safe for patients with stable CAD and preserved LV function provided exercise test and 24 h Holter ECG are performed at entry to exclude patients with ischemic episodes. Moreover, Holter monitoring during water-based exercises can be considered a valuable addition in identifying patients at risk for swimming-related arrhythmias. Further studies are warranted to establish duration and intensity of water-based exercises to optimize improvement in cardiorespiratory fitness in patients with various cardiac diseases. Moreover, future research is needed to allow WBT prescribing and conduction with modality specific test.

Limitations of the study

The present study population is limited to optimally treated group of male patients after MI without LV systolic dysfunction and does not represent a random sample of CAD patients. As such, our findings cannot be considered representative of the CAD population and extended to higher risk patients. Moreover, it was only non-randomized exercise study without any assessment of cardiovascular risk factors. Despite the study limitations listed above, the results of our study encouraged WBT to be applied in low-risk groups of male patients with stable CAD and without LV systolic dysfunction.

Conclusions

1. Water-based training provoked arrhythmias significantly more often than did exercise stress test and normal daily activity.
2. Owing to WBT patients improved their physical capacity which was still maintained at 1 year follow-up.
3. Water-based training in moderately cold water is an effective and safe form of physical training in men with stable CAD and preserved LV function.

Conflict of interest: None declared

References

1. Schmid JP, Morger C, Noveanu M et al. Haemodynamic and arrhythmic effects of moderately cold (22°C) water immersion and swimming in patients with stable coronary artery disease and heart failure. Eur J Heart Fail, 2009; 11: 903–909.
2. Craig AB, Dworak M. Thermal regulation during water immersion. J Appl Physiol, 1996; 21: 1577–1585.

3. Schmid JP, Noveanu M, Morger C at al. Influence of water immersion water gymnastics and swimming on cardiac output in patients with heart failure. Heart, 2007; 93: 722–727.
4. Weiss M, Hack F, Sthele R et al. Effects of temperature and immersion on plasma catecholamines and circulation. Int J Sports Med, 1988; 9: S113–S117.
5. Sramek P, Simeckova M, Jansky L et al. Human physiological responses to immersion into water of different temperatures. Eur J Appl Physiol, 2000; 81: 436–442.
6. Korzeniowska-Kubacka I, Dobraszkiewicz-Wasilewska B, Bilińska M et al. Effects of interval training in patients with coronary artery disease and positive exercise stress test: A pilot study. Advances Rehabilitation, 2011; 2: 37–42.
7. Perk J, De Backer G, Gohlke H et al. European guidelines on cardiovascular disease prevention in clinical practice (version 2012): The fifth joint task force of the European Society of Cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of nine societies and by invited experts). Int J Behav Med, 2012; 19: 403–488.
8. Niebauer J, Hambrecht R, Hauer K et al. Identification of patients at risk during swimming by Holter monitoring. Am J Cardiol, 1994; 74: 651–656.
9. Dąbrowski A. Assessment of antiarrhythmic therapy. In: Dąbrowska B, Dąbrowski A, Piotrowicz R eds. Holter electrocardiography. Via Medica, Gdańsk 2004: 158–159.
10. Itoh M, Araki H, Hotokebuchi N et al. Increased heart rate and blood pressure response, and occurrence of arrhythmias in elderly swimmers. J Sports Med Phys Fitness, 1994; 34: 169–178.
11. Teffaha D, Mourot L, Vernochet P at al. Relevance of water gymnastics in rehabilitation programs in patients with chronic heart failure or coronary artery disease with normal left ventricular function. J Cardiac Fail, 2011; 17: 676–683.
12. Cider A, Schaufelberger M, Sunnerhagen KS, Andersson B. Hydrotherapy: A new approach to improve function in older patient with chronic heart failure. Eur J Heart Fail, 2003; 5: 527–535.
13. Meredith-Jones K, Waters D, Legge M, Jones L. Upright water-based exercise to improve cardiovascular and metabolic health: A qualitative review. Compl Therapies Med, 2011; 19: 93–103.
14. Dobraszkiewicz-Wasilewska B, Baranowski R, Korzeniowska--Kubacka I et al. The comparison of the effects of interval training and swimming pool training in post myocardial infarction patients and in patients post CABG: The preliminary results. Folia Cardiol, 2004; 11: 831–837.
15. Leclerq JF, Maisonblanche P, Cauchemez B, Coumel P. Respective role of sympathetic tone and of cardiac pauses in the genesis of 62 cases of ventricular fibrillation during Holter monitoring. Eur Heart J, 1988; 9: 1276–1283.
16. Roelke M, Garan H, Mc Govern BA, Ruskin JN. Analysis of the initiation of spontaneous monomorphic ventricular tachycardia by stored intracardiac electrocardiograms. J Am Coll Cardiol, 1994; 23: 117–122.