ORIGINAL ARTICLE

Influence of warm-up ischemia on the effects of exercise training in patients with stable angina

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KEY WORDS

coronary artery disease, exercise training, warm-up phenomenon

ABSTRACT

INTRODUCTION It is still unknown whether ischemia-inducing training in patients with stable angina is superior to the training conducted below the ischemic threshold (IT) according to the current guidelines.

OBJECTIVES The aim of the study was to assess the influence of warm-up ischemia prior to training on the effects of training conducted either at or below the IT in patients with stable angina.

PATIENTS AND METHODS Thirty male patients aged 56 ±8 years, after myocardial infarction, with stable angina and positive exercise test (ET1) were divided into 2 groups: group A included 18 patients with the warm-up effect, group B – 12 patients without this effect. All patients followed an 8-week interval training program (TP). The intensity of training was planned to reach the heart rate at the IT. Successive ETs were performed immediately after the TP (ET2), at day 3 (ET3), day 10 (ET4), and at 1 month (ET5).

RESULTS After the TP, there was a statistically significant improvement in group A in all analyzed variables except maximum ST depression (max STD). Maximal workload increased by 28%, walking distance by 24%, duration by 20%, and time to 1-mm STD by 28%. Max STD reduction amounted to 14% ($P = 0.13$). The beneficial effect of training on exercise-induced ischemia was maintained for up to 10 days (ET4) and on physical capacity for up to 1 month (ET5). In group B, the TP did not affect time to 1-mm STD, but physical capacity improved significantly and was maintained for up to 1 month (ET5).

CONCLUSIONS The warm-up effect appears to be necessary to attenuate myocardial ischemia after training.

INTRODUCTION Exercise training improves functional capacity, reduces clinical symptoms and, subsequently, cardiovascular mortality in patients with stable coronary artery disease (CAD).[1-5] Based on the documented benefits, even patients at increased risk for cardiovascular complications during exercise should engage in individually designed training programs (TPs).

The guidelines on exercise prescription in CAD patients recommend that training intensity should stay below the ischemic threshold (IT) and correspond to the heart rate (HR) that remains 10 beats per minute (bpm) below the rate at which 1-mm ST-segment depression (STD) on electrocardiogram (ECG) occurs.[6] There is still unresolved question whether a more intensive ischemia-inducing training regime would be superior to a guideline-driven approach. A previously published paper established that even mild exercise with no detectable myocardial ischemia appeared sufficient to reduce subsequent angina on exertion. However, only exercise of greater intensity that provoked myocardial ischemia was able to attenuate ischemia on re-exercise.[7,8] These observations are consistent with patients' descriptions of the warm-up effect in everyday life. The time-course of this phenomenon describing attenuation of angina when preceded by recent angina-provoking exertion strictly resembles that of ischemic preconditioning demonstrated in experimental studies.[9-11] Such findings should be of practical relevance in developing exercise training protocols in CAD patients. Therefore, the aim of this study was: first, to examine whether the presence of the initial warm-up effect influenced myocardial ischemia after training; second, to evaluate whether repeated ischemic training sessions were necessary to attenuate

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myocardial ischemia after training; and third, to determine how long a potentially anti-ischemic effect of training persisted.

PATIENTS AND METHODS From January 2009 to October 2011, 30 male patients, aged 56 ±8 years, with stable angina pectoris (Canadian Cardiovascular Society class I or II) and positive exercise test agreed to participate in an 8-week comprehensive cardiac rehabilitation program, 3 to 4 weeks after uncomplicated myocardial infarction (MI).

CAD was angiographically confirmed and documented by all subjects who had history of MI before recruitment to the study. The inclusion criteria were: stable angina for 1 month before the inclusion in the study, age 65 years or younger, and preserved left ventricular (LV) systolic function (LV ejection fraction [LVEF] ≥45%).

Patients were excluded if they were older than 65 years, had unstable angina, congestive heart failure or LV dysfunction with LVEF less than 45%, uncontrolled hypertension, arrhythmia, valvular heart disease, left bundle branch block, LV hypertrophy or resting ST-segment changes, and diabetes. Patients' medications were not altered throughout the study.

The study protocol was approved by the Institutional Ethics Committee on human research and each participant gave their written informed consent.

Study protocol At entry, all patients underwent physical examination, electrocardiography (ECG), echocardiography, and baseline exercise tests (ET) on a treadmill according to the modified Bruce protocol (Cambridge Heart, Spacelabs, United States) and had to report chest pain and/or fatigue accompanied by STD of at least 1 mm.

On the day of the study, the subjects were instructed to avoid any unusual exertion; they rested for at least 30 minutes before ET, which was always undertaken in the mid-afternoon, 3 hours postprandially and was conditioned by the absence of angina on the day before exercise testing.

A standard 12-lead ECG and blood pressure (BP) measurements were taken at baseline, at the end of each stage, at peak exercise, and during a 10-minute recovery period. A 3-lead ECG was monitored continuously before, during, and for 10 minutes after ET. ET was considered positive at the first appearance of ≥1-mm of horizontal or downsloping STD 80 ms after J point, detected in at least 2 consecutive precordial leads. All tests were supervised by the same cardiologist and interpreted in a blind fashion by two other investigators. Any discrepancies were resolved by consensus. The following variables were determined: maximal workload (METs), walking distance (WD, m), exercise duration (ED, min), time to 1-mm STD (min), maximum STD (max STD, mm), HR (bpm), and BP (mmHg) at rest, at 1-mm STD, and at peak exercise.

Following the first positive ET (ET1), which was considered an initial ischemic stimulus, all patients re-exercised after 30 minutes (ET-30) so that patients who experienced the warm-up effect could be identified. Myocardial ischemia at the peak of ET-30 was considered reduced when the time to 1-mm STD was prolonged by at least 10%.

Patients in whom time to 1-mm STD prolonged significantly (8.37 ±3.43 vs. 10.24 ±4.08, $P = 0.01$), i.e., those in whom the warm-up effect during ET-30 was observed, were in group A (n = 18), whereas group B (n = 12) consisted of patients with unchanged time to positivity onset during ET-30 (8.17 ±4.04 vs. 8.31 ±4.13, P = nonsignificant). Then, all patients underwent an 8-week exercise TP, and echocardiography and ET (ET2) were repeated at the conclusion of the training protocol. Afterwards, all patients underwent successive ETs performed at days 3 (ET3), 10 (ET4), and at 1 month (ET5) after ending the TP.

Two-dimensional echocardiography Imaging was performed in the left lateral decubitus position using the VIVID 7 device (General Electric, United States) with a multifrequency transducer equipped with the Tissue Doppler Imaging (TDI) software. Cardiac chamber dimensions were measured according to the recommendations of the American Society of Echocardiography and the European Association of Echocardiography.[12] Transmitral LV filling velocities at the tips of the mitral valve leaflets were obtained from the apical 4-chamber view using pulsed-wave Doppler echocardiography.

The following variables were analyzed from the TDI recordings: peak velocity of early transmitral LV filling (E, m/s) and diastolic velocities of lateral (E'l), septal (E's), anterior (E'a), and posterior (E'p) parts of the mitral annulus in m/s. In addition, the E to E´ ratio was calculated because it appeared to be correlated with LV filling pressure.[13,14] The LVEF was obtained by the Simpson's biplane method.

Exercise training program The TP lasted 8 weeks and consisted of 24 interval trainings on a cycloergometer 3 times a week. Each session lasted 40 minutes, included a 2-minute warm-up, cycling under continuous electrocardiographic monitoring during which loads were applied in an interval manner, i.e., 4-minute exercise bouts separated by 2-minute rest. The TP was planned individually for each patient. The intensity of a training session was calculated to obtain HR reached at 1-mm STD during ET1. BP and HR were measured at baseline, at the end of each interval, and at recovery.

Statistical analysis A statistical analysis was performed using the SAS statistical software v. 8.2 (Cary, North Carolina, United States). All data were expressed as mean ± standard deviation or as percentages. The t test for matched pairs or

TABLE 1 Clinical characteristics of the study groups

Characteristic		Group A (n = 18)	Group B (n = 12)	*P*
age, y		54.72 ±8.37	57.18 ±9.78	0.53
smoking history, n (%)		7 (38.8)	6 (50.0)	0.55
hypertension, n (%)		15 (83.3)	10 (83.3)	1.0
hypercholesterolemia, n (%)		18 (100)	12 (100)	1.0
history of MI, n (%)		18 (100)	12 (100)	1.0
history of angina, mo		32.8 ±20.8	31.8 ±20.7	0.76
LVEF, %		59.0 ±10.30	52.41 ±8.87	0.22
medications	β-blockers, n (%)	17 (94.4)	9 (75.0)	0.13
	ACEIs, n (%)	15 (83.3)	10 (83.3)	1.0
	statins, n (%)	18 (100)	12 (100)	1.0
	aspirin, n (%)	18 (100)	12 (100)	1.0
	clopidogrel, n (%)	18 (100)	12 (100)	1.0

Data are expressed as mean ± standard deviation or number (percentage).

Abbreviations: ACEIs – angiotensin-converting enzyme inhibitors, LVEF – left ventricular ejection fraction, MI – myocardial infarction

TABLE 2 Results of baseline exercise tests in the study groups

	Group A (n = 18)	Group B (n = 12)	*P*
workload, METs	6.9 ±1.9	7.3 ±2.1	0.74
walking distance, m	578.1 ±177.3	624.4 ±217.2	0.32
exercise duration, min	9.3 ±3.2	8.6 ±3.6	0.69
time to 1-mm STD, min	8.4 ±3.4	8.2 ±4.0	0.82
max STD, mm	1.3 ±0.3	1.4 ±0.5	0.26

Data are expressed as mean ± standard deviation.

Abbreviations: MET – standard metabolic equivalent is a unit used to estimate the amount of oxygen (O_2) used by the body during physical activity, 1 MET = 3.5 ml O_2/kg/min used by the body at rest, max STD – maximum ST-segment depression

the analysis of variance were used to compare the parameters of a continuous type in the study groups, when the distribution of variables did not differ significantly from the normal distribution; when it did, the nonparametric rank test was used. To assess the differences of categorized parameters in the groups, the χ^2 test was used when the number was high enough or the Fisher's exact test when it was not. *P* value less than 0.05 was considered statistically significant.

RESULTS We studied male patients with stable angina and similar clinical status. The clinical characteristics of the patients are listed in TABLE 1. There were no intergroup differences in age, coronary risk factors, angina duration and MI history, LVEF, and concomitant medications. The doses of medications were unchanged during the study period. Moreover, there were no differences in the results of ET1 between the 2 groups (TABLE 2). Exercise training sessions were performed to obtain HR reached at 1-mm STD during ET1. Training HR limits were comparable between group A (106.2 ±11.7 bpm) and group B (107.8 ±11.0 bpm). In group A, 7 patients (39%) trained at an intensity that induced 1 mm of STD, whereas in group B, only 3 patients (25%) trained at the IT. The remaining patients from either group were not able to attain the IT at peak of each training session. In group A, all analyzed variables improved significantly during ET2 performed after completing TP in comparison with ET1, except max STD (FIGURE 1). An increase in workload amounted to 28%, in WD to 24%, in ED to 20%, and in time to 1-mm STD to 28%. The reduction in max STD, although statistically nonsignificant, was 14%. Of note, HR assessed at IT during ET1 and ET2 was comparable (107 ±10 vs. 107 ±11 bpm) despite significant prolongation of time to 1-mm STD suggesting that exercise of greater intensity provokes less ischemia. Moreover, there was a further improvement in all analyzed parameters, except for max STD, during the tests performed after 3 (ET3) and 10 days (ET4) after finishing the TP compared with ET1. During ET3, an increase in workload amounted to 33%, in WD to 32%, in ED to 26%, and in time to 1-mm STD to 39%. During ET4, an increase in workload amounted to 47%, in WD to 40%, in ED to 28%, and in time to 1-mm STD to 33%.

Furthermore, a beneficial effect of training on physical capacity (PC) was maintained for up to 1 month (ET5). Of note, there were no additional beneficial effects on myocardial ischemia

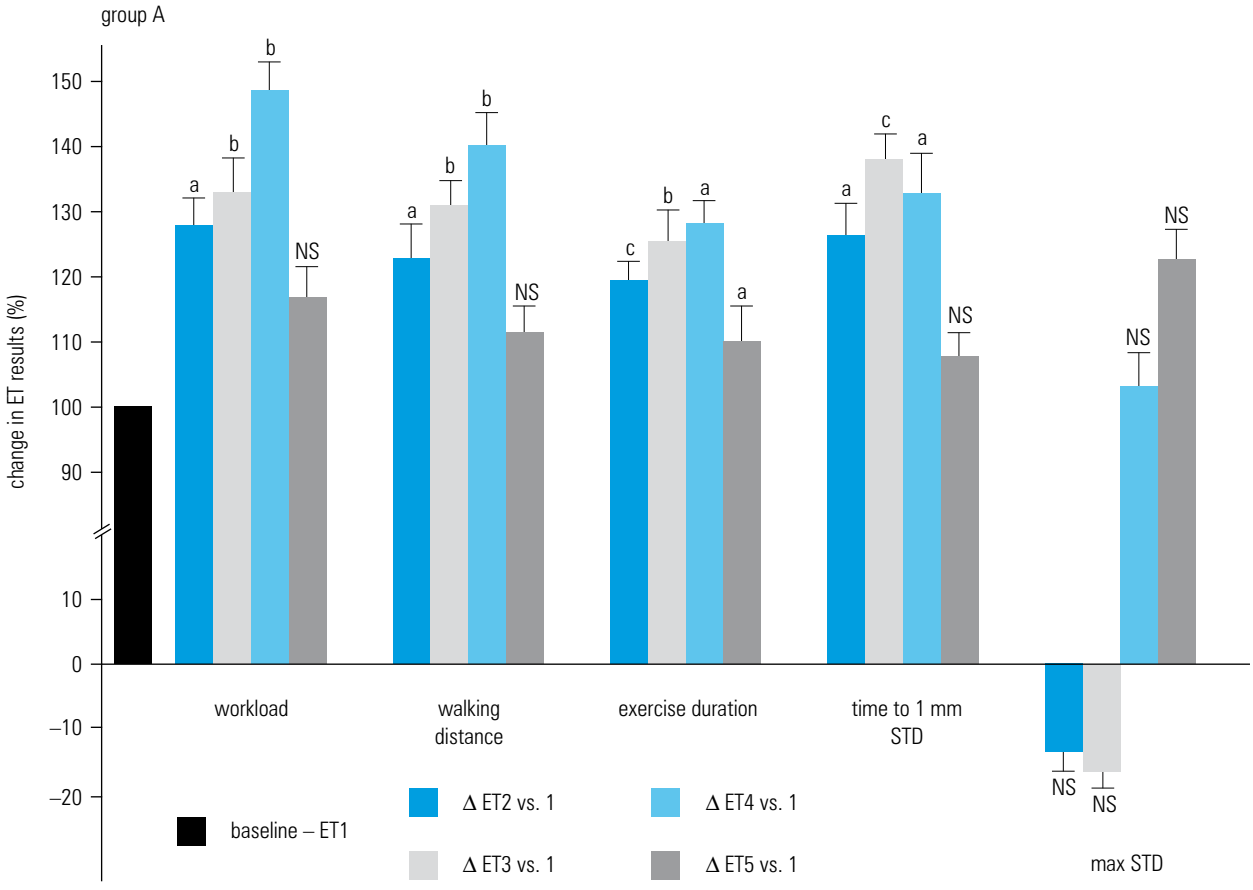

FIGURE 1 Changes in the results of exercise tests performed in group A; percent changes from the results of the baseline exercise test are plotted; all values are mean ± standard deviation

a $P < 0.05$

b $P < 0.01$

c $P < 0.001$ vs. baseline

Abbreviations: ET1 – baseline exercise test, ET2 – ET performed immediately after ending the training program, ET3, ET4, ET5 – ETs performed at day 3, day 10, and at 1 month, respectively, NS – nonsignificant, others – see TABLE 2

attenuation in the subgroup of patients (A_1) trained at the IT compared with A_2 (FIGURE 2).

In group B, there was no influence of the TP on time to 1-mm STD and HR assessed at IT during ET1 in relation to ET2 (110 ±11 vs. 112 ±11 bpm); however, the parameters of PC improved significantly in comparison with ET1. An increase in workload amounted to 24%, in WD to 20%, and in ED to 25%. During ET3, an increase in workload amounted to 23%, in WD to 18%, and in ED to 26%. During ET4, an increase in workload amounted to 19%, in WD to 13%, and in ED to 24%. Moreover, similarly to group A, this improvement was maintained for up to 1 month (ET5) (FIGURE 3).

Finally, participation in the TP did not provoke deterioration of either systolic or diastolic LV functions as measured by echocardiography (TABLE 3).

The health status was stable in all patients during the study and none of them had any adverse effects of exercise training.

DISCUSSION The main finding of this prospective observational study was that interval training on a cycloergometer induced a short-term attenuation of the ECG signs of myocardial ischemia only in those CAD patients (group A) who presented warm-up ischemia before training.

We observed that an 8-week interval training in group A increased workload, WD, ED, and time to 1-mm STD at the ET performed immediately and 10 days after the TP. In addition, this effect was found to be independent of whether training was conducted under the IT or provoked myocardial ischemia. Importantly, HR as a determinant of myocardial oxygen consumption assessed at IT did not change significantly in group A after the TP, suggesting that for the greater workload, the patients developed less pronounced ischemia.

In contrast, patients in group B, without the initial warm-up effect improved exercise performance but failed to attenuate time to 1-mm STD after training. Thus, the favorable effect of training on myocardial ischemia seems to require the initial warm-up ischemic stimulus. Moreover, it should be emphasized that during the 8-week TP, we observed no deterioration of the LV function as measured by echocardiography.

The warm-up phenomenon is a well-known clinical observation reported by more than half of the patients with stable CAD.[15,16] They report that their anginal symptoms are worse in the morning but improve during the day. The mechanism of this phenomenon has not been fully elucidated. Bogaty et al.[8] showed that neither an improvement in myocardial perfusion nor wall motion dysfunction accounted for the attenuation of myocardial ischemia observed with repeated exercise.[8] Another possibility is that the warm-up effect might be analogous to ischemic preconditioning observed in experimental animals, in which brief episodes of ischemia and reperfusion render myocardium more resistant to subsequent ischemic insult.[17,18]

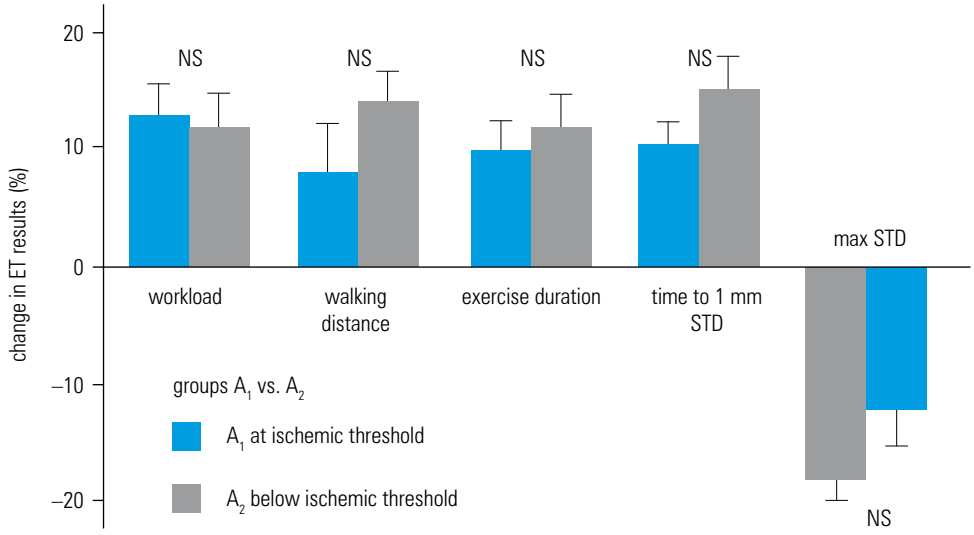

FIGURE 2 Changes in the results of exercise tests performed in subgroups A_1 and A_2 before and at the end of the training program; all values are mean ± standard deviation
Abbreviations: see TABLE 1 and FIGURE 1

TABLE 3 Echocardiographic indices of the left ventricular function before and at the end of the training program

Echocardiographic index	Group A (n = 18)			Group B (n = 12)		
	before	at the end	*P*	before	at the end	*P*
E/E' lateral, m/s	6.8 ±1.9	6.9 ±1.9	0.19	7.0 ±2.1	7.9 ±2.7	0.36
E/E' septal, m/s	7.7 ±1.9	8.3 ±1.4	0.56	8.6 ±1.4	8.5 ±1.6	0.19
E/E' posterior, m/s	6.8 ±1.9	7.3 ±1.8	0.19	7.5 ±2.8	7.8 ±2.7	0.30
E/E' anterior, m/s	8.2 ±1.8	8.3 ±1.96	0.18	8.9 ±2.1	9.3 ±1.8	0.89
LVEF, %	59.0 ±10.5	60.0 ±8.7	0.39	52.4 ±8,8	53.0 ±8.9	0.28

Data are expressed as mean ± standard deviation. There were no intergroup differences in the baseline values for these variables in either study group.

Abbreviations: E – early transmitral left ventricular filling velocity, E´ – early diastolic mitral annulus velocity, others – see TABLE 1

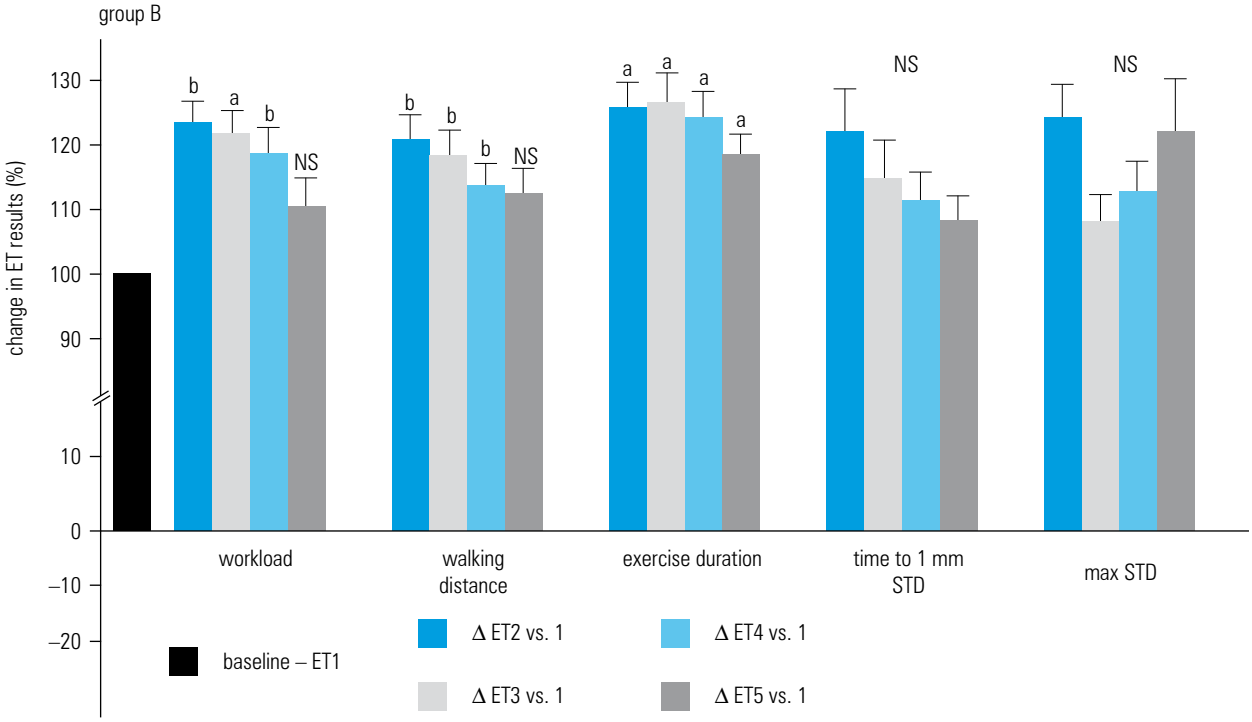

FIGURE 3 Changes in the results of exercise tests performed in group B; percent changes from the results of the baseline exercise test are plotted; see FIGURE 1 for more details; all values are mean ± standard deviation
a $P < 0.05$
b $P < 0.01$ vs. baseline
Abbreviations: see TABLE 1

Interestingly, in our patients the presence of warm-up ischemia prior to training resulted in further prolongation in time to 1-mm STD observed 72 hours after training. Furthermore, this favorable anti-ischemic effect was maintained for up to 10 days, independently of training intensity. Long-standing evidence supports the view that exercise training remains a key tool in the management of stable CAD patients.[19] Regular training has numeorus cardiac, metabolic, and psychological benefits.[4,6,20] However, it should also ensure patients' safety. Therefore, prescribing exercise to stable CAD patients is a problem clinicians regularly face. It is still unknown whether more intensive ischemia-inducing TPs would be superior to a guideline-driven approach, which recommends that training intensity in stable CAD patients should correspond to a HR that remains 10 bpm below the IT.[21,22] There is some evidence that only training at higher intensity produces improvements in physical capacity and lipid profile.[1] On the other hand, Noel et al.[23] showed that although prolonged and repeated ischemic training sessions up to 60 minutes were safe without evidence of myocardial injury, significant arrhythmias, or LV dysfunction, there were no measurable improvements in physical capacity and rate pressure product in an ischemic group compared with traditionally trained patients.

In our study, all trained patients improved their physical capacity, which was maintained for up to 1 month. However, the warm-up effect before their training was needed to induce less ischemia after an 8-week TP. Moreover, myocardial protection against ischemia was prolonged and appeared to be independent of the intensity of the training protocol. An attractive explanation would be that some kind of myocardial adaptation induced by warm-up ischemia accounted for delayed attenuation of myocardial ischemia, similarly to the delayed phase of ischemic preconditioning. Thus, the explanation of the mechanism of the warm-up phenomenon may be a tool for its practical exploitation. For example, some pharmacological agents may reproduce the warm-up effect without ischemia-provoking exercise. Moreover, it should be examined why this preconditioning-like effect is present only in some patients with stable CAD.

In summary, our findings suggest that the warm-up effect before exercise training appears to be the key to attenuate myocardial ischemia after training. These findings may be of practical relevance in physical activity counselling.

Conclusions The warm-up effect before exercise training appears to be necessary to attenuate myocardial ischemia after training. Training with repeated ischemic sessions has no additional beneficial effects on exercise tolerance of ischemia. The time-course of anti-ischemic effects of training resembles delayed attenuation of myocardial ischemia observed during ischemic preconditioning.

Limitations First, the results of this study can be applied to a specific group of patients with stable angina, positive ET, and preserved LV function. Therefore, the very rigorous selection criteria limited the number of patients included to the study. Moreover, women were not included because the reliability of the stress test in women is lower than that in men. Second, by using STD as an electrocardiographic marker of myocardial ischemia during ET, we eliminated those patients who had evidence of ST-segment shift at resting ECG. Moreover, irrespective of the presence or absence of MI, STD during ET has been accepted as a noninvasive tool for patient selection. Third, apart from the above study limitations, the nonrandomized design is another one that has to be considered.

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REFERENCES

1 O'Donovan G, Owen A, Bird SR et al. Changes in cardiorespiratory fitness and coronary heart disease risk factors following 24-wk of moderate- or high-intensity exercise of equal energy cost. J Appl Physiol. 2005; 98: 1619-1625.

2 Laslett LJ, Paumer L, Amsterdam EA. Increase in myocardial oxygen consumption indexes by exercise training at onset of ischemia in patients with coronary artery disease. Circulation. 1985; 71: 958-962.

3 Hofman-Bang C, Lisspers J, Nordlander R, et al. Two-year results of a controlled study of residential rehabilitation for patients treated with percutaneous transluminal coronary angioplasty: a randomized study of a multifactorial programme. Eur Heart J. 1999; 90: 1465-1474.

4 Maines TY, Lavie CJ, Milani RV, et al. Effect of cardiac rehabilitation and exercise programs on exercise capacity, coronary risk factors, behavior, and quality of life in patients with coronary artery disease. South Med J. 1997; 90: 43-49.

5 Bertelinek R, Oh P. Exercise training and the ischemic patient. Current Issues in Cardiac Rehabilitation and Prevention. 2006; 14: 17-20.

6 Fletcher G F, Balady GJ, Amsterdam EA, et al. Exercise Standards for testing and training: a statement for healthcare professionals from the American Heart Association. Circulation. 2001; 104: 1694-1740.

7 Bogaty P, Kingma JG, Robitaille NM, et al. Attenuation of myocardial ischemia with repeated exercise in subject with chronic angina: relation to myocardial contractility, intensity of exercise and the adenosine triphosphate-sensitive potassium channel. J Am Coll Cardiol. 1998; 32: 1665-1671.

8 Bogaty P, Poirier P, Boyer L, et al. What induces the warm-up ischemia/angina phenomenon: exercise or myocardial ischemia? Circulation. 2003, 107: 1858-1863.

9 Murry CE, Jennings RB, Reimer KA. Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium. Circulation. 1986; 74: 1124-1136.

10 Sumeray MS, Yellon DM: Myocardial preconditioning. What have we learned? Eur Heart J. 1997; 18 (Suppl A): A8-A14.

11 Bilińska M. [Ischemic preconditioning in human heart]. Kardiol Pol. 1999; 51: I-73.1. Polish.

12 Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification. Eur J Echocard. 2006; 7: 79-108.

13 Mulvagh S, Quinones MA, Kleiman NS, et al. Estimation of left ventricular end-diastolic pressure from Doppler transmitral flow velocity in cardiac patients independent of systolic performance. J Am Coll Cardiol. 1992; 20: 112-119.

14 Paulus WJ, Tschöpe C, Sanderson JE, et al. How to diagnose diastolic heart failure: a consensus statement on the diagnosis of heart failure with normal left ventricular ejection fraction by the Heart Failure and Echocardiography Association of the European Society of Cardiology. Eur Heart J. 2007; 28: 2539-2550.

15 Marber MS, Joy MD, Yellon DM. Is warm-up in angina ischemic preconditioning? Br Heart J. 1994; 72: 213-215.

16 Negrusz-Kawecka M, Mazurek W. [Opinions on the cardioprotective influence of ischemic preconditioning]. Pol Arch Med Wewn. 1999; 101: 75-80. Polish.

17 Kavazis AN. Exercise preconditioning of the myocardium. Sports Med. 2009; 39: 923-935.

18 Korzeniowska-Kubacka I. [Physical training as an effective way to protect the heart against ischaemia]. Kardiol Pol. 2011; 69 (Suppl 3): 75-79. Polish.

19 Gibbons RJ, Chatterjee K, Daley J, et al., ACC/AHA/ACP-ASIM guidelines for the management of patients with chronic stable angina: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guide-

lines (Committee on Management of Patients With Chronic Stable Angina). J Am Coll Cardiol. 1999; 33: 2092-2197.

20 Digenio AG, Noakes TD, Joughin H, Daly L. Effect of myocardial ischaemia on left ventricular function and adaptability to exercise training. Med Sci Sports Exerc. 1999; 31: 1094-1101.

21 Lennon SL, Quindry J, Hamilton KL, et al. Loss of exercise-induced cardioprotection after cessation of exercise. J Apply Physiol. 2004; 96: 1299-1305.

22 Ehsani AA, Heath GW, Hagberg JM, et al. Effects of 12 months of intense exercise training on ischemic ST-segment depression in patients with coronary artery disease. Circulation. 1981; 64: 1116-1124.

23 Nöel M, Jobin J, Marcoux A, et al. Can prolonged exercise-induced myocardial ischemia be innocuous? Eur Heart J. 2007; 28: 1559-1565.

Wpływ zjawiska *warm-up* na efekty treningu fizycznego u chorych na stabilną chorobę wieńcową

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SŁOWA KLUCZOWE

choroba wieńcowa,
trening fizyczny,
zjawisko rozgrzewki

STRESZCZENIE

WPROWADZENIE Dotychczas nie wiadomo, czy trening indukujący niedokrwienie przynosi więcej korzyści niż trening prowadzony według obowiązujących standardów poniżej progu niedokrwienia, u pacjentów ze stabilną chorobą.

CELE Celem badania była ocena wpływu efektu rozgrzewki (*warm-up*) stwierdzanego przed rozpoczęciem programu treningowego na efekty treningu prowadzonego poniżej lub na progu niedokrwienia u pacjentów ze stabilną chorobą wieńcową.

PACJENCI I METODY Trzydziestu mężczyzn w wieku 56 ±8 lat, po zawale serca, ze stabilną chorobą wieńcową i dodatnim testem wysiłkowym (*exercise test* – ET1) podzielono na 2 grupy: do grupy A włączono 18 chorych z występującym efektem *warm-up*, a do grupy B – 12 chorych bez tego efektu. Wszyscy chorzy odbyli 8-tygodniowy cykl treningów interwałowych. Jako limit tętna treningowego ustalono próg niedokrwienia. Kolejne testy wysiłkowe wykonywano bezpośrednio po zakończeniu treningu (ET2), w 3. dniu (ET3), 10. dniu (ET4) i miesiąc po zakończeniu treningu (ET5).

WYNIKI Po cyklu treningowym w grupie A nastąpiła istotna statystycznie poprawa wszystkich badanych parametrów z wyjątkiem maksymalnego obniżenia ST. Maksymalne obciążenie wzrosło o 28%, dystans marszu o 24%, czas trwania próby o 20%, a czas do obniżenia ST o 1 mm – o 28%. Redukcja maksymalnego obniżenia ST wyniosła 14% ($p = 0,13$). Korzystny efekt treningu na wysiłkowe niedokrwienie utrzymał się do 10 dni (ET4), a na wydolność fizyczną – do miesiąca (ET5) od zakończenia treningu. W grupie B, cykl treningowy nie wpłynął na czas do obniżenia ST o 1 mm, ale uzyskano istotną poprawę wydolności fizycznej, która utrzymała się do miesiąca (ET5).

WNIOSKI Efekt *warm-up* wydaje się warunkiem koniecznym do poprawy ukrwienia mięśnia sercowego pod wpływem treningu.

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