HEALTH IMPACT OF TRAINING INTENSITY IN OLDER INDIVIDUALS

Katarina T. Borer[1], **Germaine Cornelissen**[2], **Franz Halberg**[2] **and Courtney Hughes**[1]

[1]*Division of Kinesiology, University of Michigan, Ann Arbor, USA*
[2]*Chronobiology Center, University of Minnesota, Minneapolis, USA*

Conference paper
UDC 796.067.3:613.73-053.9

Introduction

As we age, our capacity for sustained and intense physical exertion declines. This partly represents a decline in the cardio-respiratory capacity epitomized by the relationship of the variables in the Fick equation: a lowering of the rate of oxygen consumption as a function of reduction in its two components, the cardiac output and tissue extraction of oxygen. The undesirable consequences of these changes include reduced capacity for heart rate acceleration (tachycardia) during exertion and increased risk of developing cardiovascular disease (CVD). CVD is the leading cause of death of older people in developed societies. Despite considerable interest in prevention and treatment of the decline in cardio-respiratory function, it is still not certain to what extent it is caused by the irreversible features of aging and hence likely more resistant to intervention, and to what extent it is caused by a lifestyle of inactivity and excessive consumption of energy-rich food, in which case it should be more readily modifiable. Inactivity and excessive intake of energy-rich food are considered to be the chief causes of a metabolic syndrome characterized by some combination of hypertension, abdominal obesity, hyper-lipidemia, insulin resistance, and hyper-coagulability of blood. These in turn are the risk factors for CVD and strokes (Kaplan, 1989; Opara & Levine, 1997). A decline in cardio-respiratory capacity with age is therefore of considerable interest as it interferes with a full range of physical activities in advanced years. It also is of considerable medical interest as it increases morbidity and importantly contributes to premature mortality.

Of the cardiovascular dependent variables in the Fick equation, ventilatory threshold (VT) as a measure of aerobic capacity (or oxygen consumption), and the heart rate (HR) component of cardiac output, are easy to measure. VT is a sensitive systemic index of the capacity for aerobic metabolism (Davis et al.,1976). As exercise intensity exceeds this capacity, increased production of lactic acid through anaerobic glycolysis triggers compensatory hyperventilation. Changes in aerobic capacity can therefore be monitored through measurements of VT. HR measurements are also indicative of an individual's cardio-respiratory fitness both at rest (when the relationship is inverse) and during a progressive physical challenge. In the latter case, the rate of HR acceleration to oxygen demands during progressive increase in exercise intensity is a measure of cardiac fitness. In addition, physical deconditioning is associated with decreased HR variability and may reflect excessive sympathetic and inadequate parasympathetic control of the heart function (Malliani, 1999). Decreased HR variability also is associated with increased risk of death (Adamopoulos et al., 1999).

Blood pressure (BP) is a variable of cardio-respiratory function that participates in oxygen delivery during exercise and also has a profound impact on cardiovascular health. Systolic blood pressure (BPsys) increases during exercise, while vasodilatation in the large contracting muscle mass prevents a change, or causes a decrease, in diastolic pressure (BPdia). Age-associated increases in BP can damage circulation to kidneys and other vital peripheral organs, cause cardiac hypertrophy, and increase the risk of a stroke. While exercise training can reduce resting BPsys and BPdia, its effectiveness has not been universal, and at high training intensities, deleterious effects on BP have sometimes been observed. This lack of consensus regarding the effects of exercise on BP is reflected in the American College of Sports Medicine position stand (1998) that advocates moderate exercise training intensities for BP reduction. In contrast to HR variability, BP variability is positively correlated with an increased risk of coronary heart disease and strokes (Kikuya et al., 2000). In addition to general BP variability, a circadian form of BP variability (Circadian-Hyper-Amplitude-Tension or CHAT) that consists of excessive diurnal elevations, and excessive nocturnal declines in BP, also confers an independent and significantly increased risk of strokes and nephropathy (Otsuka et al., 1996, 1997). The relation between the circadian amplitude and this risk appears to be non-linear (Cornelissen et al., 2000, 2001).

The aim of this study was to examine the extent to which a simple prototypic form of physical activity, walking, can improve the components of cardio-respiratory function in aging individuals. Postmenopausal women were selected for study because of their demographic prevalence, general paucity of information on the effects of training on their cardio-respiratory function, and their increased risk of CHD following estradiol decline. Walking was selected because it is the principal physical means of human locomotion and dynamically engages large muscle groups. It also is universally accessible without the necessity for specialized equipment or facilities. Exercise intensity was selected as a focus of these studies as it holds both scientific and medical interest. Exercise intensity is the key element for effective cardio-respiratory training as identified by Karvonen almost half a century ago (Karvonen et al., 1957). On the other hand, intense exercise may cause excessive BP elevation and can infrequently cause cardiac death or a stroke (Ciampricotti et al., 1989, 1990; Mittleman et al., 1993). If exercise is to be prescribed for cardio-respiratory health benefits, an understanding of the dose-response relationship between its intensity and the several components of cardio-respiratory function is a necessity.

Methods

Subjects were 42 generally healthy overweight to moderately obese postmenopausal women whose characteristics are shown in Table 1. They all agreed to participate in 15 weeks of training and signed an informed consent form approved by the University of Michigan School of Medicine Institutional Review Board.

Training consisted of walking 4.83 km/day five days a week for 15 weeks at an assigned intensity. Walking was carried out at a commercial mall, between 6:30 and 7:30 h, under supervision that included measurement of times and distances covered. Women were stratified by age, weight, body composition, and initial aerobic fitness and assigned to one of two exercise intensities, 95% of VT or 125% of VT. Individual VTs were established during a walking test on a level treadmill, during which the ventilatory rate was measured, as walking speeds increased by 0.64 km/min every 3 minutes. VT was expressed as the exercise intensity at which there was a distinct increase in the rate of minute ventilation (Figure 1).

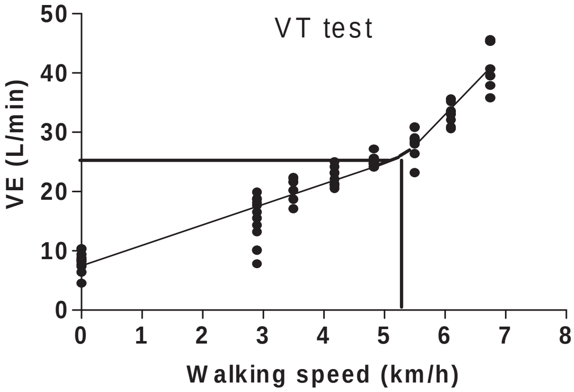

Figure 1. Ventilatory threshold (VT) test.

Dependent variables. Before the start, and after 15 weeks of training, the following measurements were taken. Body mass and stature were measured and used for the calculation of body mass index (BMI, kg/m^2). Skinfolds and circumferences were measured at standardized sites for the calculation of body fat (Jackson et al., 1980). VT was determined both for assignment to training intensity and as an indicator of aerobic capacity. HR was measured with a Polar tachometer during the treadmill walking test to determine the slope of tachycardia. Changes in HR and BP and their variabilities were assessed from HR and BP recordings collected during seven consecutive days

Table 1. Anthropometric characteristics of the 42 subjects before and after 15 weeks of training

Variable	Before training	After training	p
Age (years)	58 +/- 0.76*		
Body mass (kg)	77.5 +/- 1.7	76.9 +/- 2.0	0.09
Stature (m)	1.64 +/- 0.01		
BMI (kg/m^2)	28.7 +/- 0.58	28.5 +/- 0.72	0.09
Body fat (%)	38.69 +/- 0.77	37.62 +/- 0.93	0.0012

Mean +/- SEM. P values derived from Student's paired *t*-tests.

with the TM2421 ambulatory HR-BP monitors (A & D Company, Tokyo, Japan). During HR and BP measurements, both before and after the training, the subjects did not engage in any organized physical activity.

Statistical analyses. The circadian patterns of HR and BP changes, including the mean estimate statistic of rhythm (MESOR), the magnitude of circadian swings above and below the MESOR (double amplitudes), and the standard deviations of HRs and BPs were calculated after fitting the HR and BP data to 24-h and 12-h cosine curves (Halberg, 1969). Student's *t*-test for matched goups was used to assess changes in body mass, BMI, and body fatness as a result of training. Although the subjects were assigned to two distinct training speeds, their imperfect compliance resulted in two overlapping distribution curves of relative training intensities. A change in all cardio-respiratory dependent variables, between the values obtained after 15 weeks of training and the initial pre-training values, were subjected to regression analysis against relative training intensities. Probability of < 0.05 was used as the criterion of significant difference. A power test was used to estimate the numbers of subjects needed for inclusion in future studies to have an 80% chance of achieving statistical significance at a probability level of 5% (Cohen, 1992).

Results and discussion

Anthropometric effects of training. Training had no effect on the body mass or the BMI, but resulted in a significant loss of body fat (t=3.591, df=29, Table 1). The modest 2-3% reduction in body fat is in agreement with the limited effectiveness of aerobic training in producing body fat loss observed by others. Modest body fat loss unaccompanied by a change in body mass agrees with the findings of others that exercise protects against losses of, or produces increases in, lean body mass at the same time as it facilitates some body fat loss (Ballor & Poehlman, 1994).

Training intensity and changes in aerobic fitness. Training intensity had a very significant stimulatory effect on aerobic capacity assessed from increases in VT (Table 2 and Figure 2). The relationship between the two variables (y=0.4546x –41.02) indicates that the training produced a 4.5% increase in the VT for each 10 percent increase in training intensity. The power test prediction of a significant positive relationship between walking intensity and increases in VT at the power of 80% called for as few as 13 subjects.

Training intensity and changes in BP parameters. In contrast to the highly significant effects of training intensity on VT as a measure of capacity to utilize oxygen, training intensity had a less pronounced effect on several measures of BP function (Table 2). In decreasing the order of magnitude, the training intensity tended to decrease circadian variability (double amplitude) of BPsys (Table 2), BPdia MESOR (Figure 3), the SD measure of BPdia variability, and BPsys MESOR (Table 2). In all four instances, the effect was proportional to exercise intensity. In no case did the effect reach significance. The power test prediction for the number of subjects required to attain significance was, respectively, 70, 89, 153, and 202 (Table 2). Thus, training intensity in the ranges attainable during 15 weeks of walking had a numerical, albeit not statistically significant, dose-dependent lowering effect on the four variables. However, judging from the power test predictions, this effect was, respectively, about 5, 7, 12, and 15 times smaller than the effect of training intensity on aerobic

Table 2. Correlations between the intensity of training during 15 weeks of walking and changes in selected cardiovascular functions in postmenopausal women

Variable	Measure	Coefficient of correlation (r)	p	Number of subjects needed for significance
VT (km/h)		0.658	<0.001	13
Systolic blood pressure (mm Hg)	MESOR Double-amplitude SD	-0.175 -0.296 -0.004	0.425 0.143 0.849	202 70 4185
Diastolic blood pressure (mmHg)	MESOR Double-amplitude SD	-0.269 -0.032 0.200	0.184 0.875 0.316	89 6097 153
Heart rate (bpm)	MESOR Double-amplitude SD	-0.332 -0.036 -0.103	0.122 0.857 0.267	55 4689 582
Exercise-induced tachycardia (slope)		0.124	0.501	4512

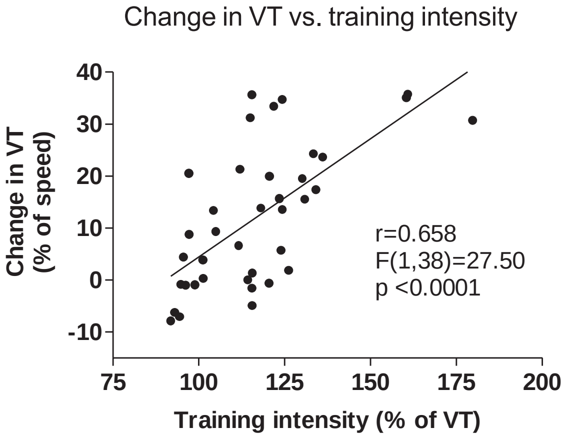

Figure 2. Change in ventilatory threshold as a function of training intensity.

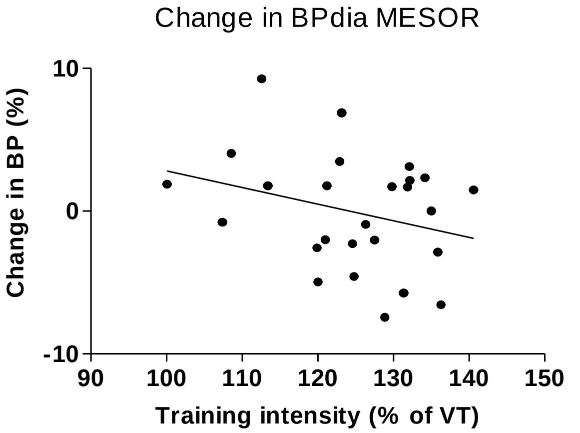

Figure 3. Change in BPdia MESOR as a function of training intensity.

capacity. Training intensity had no measurable effect on the SD of BPsys variability or on the double amplitude of the BPdia variability (Table 2).

Training intensity and changes in HR parameters. Training intensity tended to decrease HR MESOR (Table 2). Although training intensity had no significant effect on any parameter of HR function, its effect on HR MESOR was second largest (albeit four times less) in magnitude after the effect of training intensity on changes in VT. There was a slight trend toward a decrease in the SD of HR variability with increases in training intensity. Power test prediction, however, was for 600 subjects for this effect to reach statistical significance. Unlike the beneficial trends in BP changes, training intensity tended to influence the SD of HR variability in the direction associated with increased risk of cardiac death. Training intensity had no effect on the double-amplitude measure of HR variability or the slope of HR acceleration during the incremental treadmill walking test (Table 2).

Training and circadian BP overswinging (Circadian-Hyper-Amplitude-Tension or CHAT). One of the 42 subjects studied exhibited acceptable MESOR BP values, but Figure 4 (data from Borer et al., 2002) indicates the presence of CHAT with double amplitudes of BPsys in excess of the 90% prediction limits (upper and lower horizontal lines) for her age and gender. Her BPsys MESOR (middle horizontal line) was within the acceptable range before training, and 3 mmHg lower after training. In this subject, exercise training at 118.5% of her VT, also produced reductions in BPdia and HR MESORs, but had no effect on excessive circadian BPsys and BPdia double amplitudes.

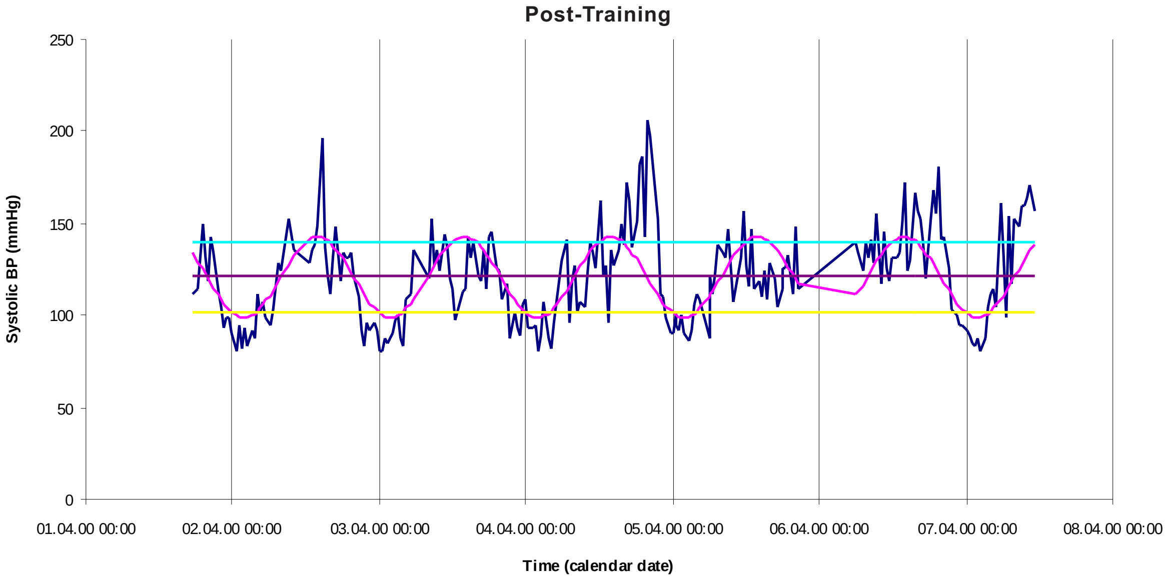

Figure 4. Persistence of circadian hypertension or CHAT after 15 weeks of training.

Conclusion

This study permits several conclusions regarding the effectiveness of walking as a means of cardio-respiratory conditioning for postmenopausal women. The overall conclusion is that 15 weeks of walking are sufficient for an increase in aerobic capacity, and that the magnitude of the effect will be, in agreement with Karvonen's observations (Karvonen et al., 1957), proportional to training intensity.

The second conclusion is that, under the conditions of this study, training intensity has pleiotropic effects on individual cardio-respiratory variables and their components. It significantly stimulated increases in aerobic capacity, but exerted insignificant lowering tendency on HR and BP parameters. Within each cardio-respiratory variable, only some components showed the effects of training intensity, while the others were unaffected. Thus, training intensity tended to decrease BPsys, BPdia, and HR MESORs, and usually only one measure of their variability (BPsys double amplitude, and BPdia and HR SDs). Although this study had insufficient power to establish definitively these relationships, it is reasonable to infer that, within the moderate range attainable by walking, training intensity could have a beneficial modifying effect on MESORS and mean values of BPsys, BPdia, and HR. These conclusions are in agreement with training studies performed at an intermediate range of intensities by others (Fagard, 2001; Kelley & Kelley, 1999; Kelley & Sharp Kelley, 2001).

In contrast to apparent modifiability by exercise training of BP and HR MESORs, the rate of HR acceleration during the progressive treadmill walking test, and some measures of HR and BP variability were unaffected by training intensity. The first variable reflects the well-known age-associated decline in maximal heart rate (Tanaka et al., 2001) and is most likely related to the change in contractile characteristics of the heart muscle fibers (Long et al., 1999) as well as age-associated reduction in their responsiveness to sympathetic stimulation (Esler et al., 1995; Seals et al., 1994). In some studies, higher levels of aerobic fitness are associated with greater HR variability (Davy et al., 1996, 1998; Gregoire et al., 1996). This would predict a positive influence of aerobic training on this variable in contrast to the trend toward reduced HR variability observed in the present study. The selective effect of training intensity on some parameters of BP (BPsys double amplitude, BPdia SD) and HR variability (HR SD), but not the others, requires further study and suggests that training intensity may affect the individual variables differently.

Finally, it is useful to acknowledge that BP is under multifactorial control. This is apparent when BP abnormalities are considered without and within the circadian context. Circadian and casual BP values may independently conform or diverge from their respective norms, as exemplified by the individual described by Borer at associates (2002) who was MESOR normortensive but exhibited excessive circadian double-amplitude swings. Her CHAT conferred an independent increased risk of a stroke in spite of her normal average blood pressure. Training lowered her already acceptable BPsys, BPdia, and HR MESORs, but had no effect on, or slightly exacerbated, her CHAT. This suggests that different mechanisms coordinate BP and HR MESORs on one hand, and their circadian swings on the other. Much more research is needed for a better understanding of the effect of exercise training on these cardio-respiratory variables. The use of ambulatory HR-BP monitors over several days, allows assessment of MESORs as well as circadian changes (Halberg et al., 2002) and thus represents a particularly useful approach to study these variables in response to exercise training.

References

Adamopoulos, S., Kemp, G, J., Thompson, C. H., Arnolda, L., Brunotte, F., Stratton, J. R., Radda, G. K., Rajagopalan, B., Kremastinos, D. T., & Coats, A. J. (1999). The time course of haemodynamic, autonomic and skeletal muscle metabolic abnormalities following first extensive myocardial infarction in man. *Journal of Molecular and Clinical Cardiology, **31***, 1913-1926.

American College of Sports Medicine Position Stand (1998). Recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. *Medicine & Science in Sports & Exercise*, ***30***, 975-991.

Ballor, D. L., & Poehlman, E. T. (1994). Exercise-training enhances fat-free mass preservation during diet-induced weight loss: a meta-analytical finding. *International Journal of Obesity*, ***18***, 35-40.

Borer, K. T., Cornelissen, G., Halberg, F., Brook, R., Rajagopalan, S., & Fay, W. (2002). Circadian blood pressure overswinging in a physically-fit normotensive Afro-American woman. *American Journal of Hypertension* (in press).

Ciampricotti, R., el-Gamal, M., Relik, T., Taverne, R., Panis, J., de Swart, J., van Gelder B., & Relik-van Wely, L. (1990). Clinical characteristics and coronary angiographic findings of patients with unstable angina, acute myocardial infarction, and survivors of sudden ischemic death occurring during and after sport. *American Heart Journal*, ***120***, 1267-1278.

Ciampricotti, R., & el Gamal, M. I. (1989). Unstable angina, myocardial infarction and sudden death after an exercise stress test. *International Journal of Cardiology*, ***24*,** 211-218.

Cohen, J. (1992). A power primer. *Psychological Bulletin*, ***112***, 155-159.

Cornelissen, G., Otsuka, K., Chen, C.-H., Kumagai, Y., Watanabe, Y., Halberg, F., Siegelova, J., & Dusek, J. (2000). Nonlinear relation of the circadian blood pressure amplitude to cardiovascular disease risk. *Scripta Medica (Brno), **73***, 85-94.

Cornelissen, G., Schwartzkopf, O., Halberg, F., Otsuka, K., & Watanabe, Y. (2001). 7-day ambulatory monitoring for adults with hypertension and diabetes. *Journal of Kidney Diseases, **37***, 878.

Davis, J. A., Vodak, P., Wilmore, J. H., Vodak, J., & Kurtz, P. (1976). Anaerobic threshold and maximal aerobic power for three modes of exercise. *Journal of Applied Physiology*, ***41***, 544-550.

Davy, K. P., DeSouza, C. A., Jones, P. P., & Seals, D. R. (1998). Elevated heart rate variability in physically active young and older adult women. *Clinical Science*, ***94***, 579-584.

Davy, K. P., Miniclier, N. L., Taylor, J. A., Stevenson, E. T., & Seals, D. R. (1996). Elevated heart rate variability in physically active postmenopausal women: a cardioprotective effect? *American Journal of Physiology*, ***271***, H455-H460.

Esler, M. D., Thompson, J. M., Kaye, D. M., Turner, A. G., Jennings, G. L., Cox, H. S., Lambert, G. W., & Seals, D. R. (1995). Effects of aging on the responsiveness of the human cardiac sympathetic nerves to stressors. *Circulation*, ***91***, 351-358.

Fagard, R. H. (2001). Exercise characteristics and the blood pressure response to dynamic physical training. *Medicine & Science in Sports & Exercise*, ***33*** (6 Suppl), S484-S492; Discussion S493-S494.

Gregoire, J., Tuck, S., Yamamoto, Y., & Hughson, R. L. (1996). Heart rate variability at rest and exercise: influence of age, gender, and physical training. *Canadian Journal of Applied Physiology*, ***21***, 455-470.

Halberg, F. (1969). Chronobiology. *Annual Reviews of Physiology, **31***, 675-725.

Halberg, F., Cornelissen, G., Wall, D., Otsuka, K., Halberg, J., Katinas, G., Watanabe, Y., Halhuber, M., Muller Bohn, T., Delmore, P., Siegelova, J., Homolka, P., Fiser, B., Dusek, J., Sanchez de la Pena, S., Maggioni, C., Delyukov, A., Gorgo, Y., Gubin, D., Caradente, F., Schaffer, E., Rhodus, N., Borer, K., Sonkowsky, R. P., & Schwartzkopf, O. (2002). Engineering and governmental challenge: 7-day/24-hour chronobiologic blood pressure and heart rate screening: Part I. *Biomedical Instrumentation & Technology*, ***36***, 89-122.

Jackson, A. S., Pollock, M. L., & Ward, A. (1980). Generalized equations for predicting body density of women. *Medicine & Science in Sports & Exercise, **12***, 175-181.

Kaplan, N. M. (1989). The deadly quartet. Upper-body obesity, glucose intolerance, hypertriglyceridemia, and hypertension. *Archives of Internal Medicine*, ***149***, 1514-1520.

Karvonen, M. J., Kentala, E., & Mustala, O. (1957). The effects of training heart rate: A longitudinal study. *Annales Medicinae Experimentalis et Biologiae Fenniae*, ***35***: 307-315.

Kelley, G. A., & Kelley, K. S. (1999). Aerobic exercise and resting blood pressure in women: a meta-analytic review of controlled clinical trials. *Journal of Women's Health & Gender-Based Medicine*, ***8***, 787-803.

Kelley, G. A., & Sharpe Kelley, K. (2001). Aerobic exercise and resting blood pressure in older adults: a meta-analytic review of randomized controlled trials. *Journals of Gerontology Series A-Biological Sciences & Medical Sciences*, ***56***, M298-303.

Kikuya, M., Hozawa, A., Ohokubo, T., Tsuji, I., Michimata, M., Matsubara, M., Ota, M., Nagai, K., Araki, T., Satoh, H., Ito, S., Hisamichi, S., & Imai, Y. (2000). Prognostic significance of blood pressure and heart rate variabilities: the Ohasama study. *Hypertension,* ***36***, 901-906.

Long, X., Boluyt, M. O., O'Neill, L., Zheng, J. S., Wu, G., Nitta, Y. K., Crow, M. T., & Lakatta, E. G. (1999). Myocardial retinoid X receptor, thyroid hormone receptor, and myosin heavy chain gene expression in the rat during adult aging. *Journals of Gerontology Series A-Biological Sciences & Medical Sciences,* ***54***, B23-27.

Malliani, A. (1999). The pattern of sympathovagal balance explored in the frequency domain. *News in Physiological Sciences*, ***14***, 111-117.

Mittleman, M. A., Maclure, M., Tofler, G. H., Sherwood, J. B., Goldberg, R. J., & Muller, J. E. (1993). Triggering of acute myocardial infarction by heavy physical exertion. Protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study Investigators. *New England Journal of Medicine*, ***329***, 1677-1683.

Opara, J. U., & J. H. Levine (1997). The deadly quartet - the insulin resistance syndrome. *Southern Medical Journal*, ***90***, 1162-1168.

Otsuka, K., Cornelissen, G., & Halberg, F. (1996). Predictive value of blood pressure dipping and swinging with regard to vascular disease risk. *Clinical Drug Investigation,* ***11***, 20-31.

Otsuka, K., Cornelissen, G., Halberg, F., & Oehlert, G. (1997). Excessive circadian amplitude of blood pressure increases risk of ischemic stroke and nephropathy. *Journal of Medical Engineering Technology,* ***21***, 23-30.

Seals, D. R., Taylor, J. A., Ng, A. V., & Esler, M. D. (1994). Exercise and aging: autonomic control of the circulation. *Medicine & Science in Sports & Exercise*, ***26***, 568-576.

Tanaka, H., Monahan, K. D., & Seals, D. R. (2001). Age-predicted maximal heart rate revisited. *Journal of the American College of Cardiology*, ***37***, 153-156.

Correspodence to:
Prof. Katarina T. Borer, Ph.D.
Department of Kinesiology University of Michigan
401 Washtenow Avenue
48109-2214 Ann Arbor, MI, USA
Phone: +1 (734) 647-2703
Fax: +1 (734) 647-1925
E-mail: katarina@umich.edu

UTJECAJ TRENAŽNOG INTENZITETA NA ZDRAVLJE STARIJIH OSOBA

Sažetak

Uvod

Kako starimo, naš se kapacitet za dugotrajno i intenzivno tjelesno vježbanje smanjuje. Ta pojava djelomično ocrtava smanjenje kardiorespiratornoga kapaciteta i to na račun snižavanja stope potrošnje kisika kao funkcije dviju komponenata: srčanog učinka i tkivne potrošnje kisika. Neželjene posljedice tih promjena uključuju tahikardiju za vrijeme napora i povećani rizik od razvoja kardiovaskularnih bolesti. Usprkos povećanom interesu za prevenciju i liječenje narušenih kardio-respiratornih funkcija, još uvijek nije potpuno jasno koliko je to stanje uzrokovano ireverzibilnim promjenama starenja, koje su otporne na intervencije, a koliko neaktivnim životnim stilom ili prevelikom konzumacijom energetski bogatih namirnica, na što se može djelovati. Upravo se neaktivnost i prekomjerna konzumacija energetski bogate hrane smatraju glavnim uzrocima metaboličkog sindroma obilježenog kombinacijom povišenoga krvnoga tlaka, abdominalnom gojaznošću, povećanom količinom masnoće u krvi, inzulinskom otpornošću i povećanom koagulacijom krvi. Navedene karakteristike su i faktori rizika kardiovaskularnih bolesti i srčanog udara (Kaplan, 1989; Opara i Levine, 1997). Neke zavisne varijable koje se odnose na kardiovaskularne funkcije lako je mjeriti, npr. Fickova jednadžba, ventilacijski prag (VP) (što je mjera aerobne sposobnosti) i frekvencija srca (FS). Osim toga, krvni tlak (RR) je varijabla kardiorespiratornih funkcija koja sudjeluje u prijenosu kisika za vrijeme vježbanja i također značajno utječe na kardiovaskularno zdravlje. Povećanje krvnog tlaka, koje se javlja s dobi, može oštetiti cirkulaciju prema bubrezima i ostalim vitalnim perifernim organima, uzrokovati srčanu hipertrofiju i povećati rizik od srčanog udara. Iako tjelesno vježbanje može pridonijeti smanjenju krvnog tlaka u mirovanju, njegov utjecaj nije univerzalan. Naime, u nekim su slučajevima zabilježeni i nepovoljni učinci vježbanja (trening jačeg intenziteta). Cilj je ovog istraživanja utvrditi u kojem opsegu jednostavan prototip tjelesnog vježbanja - hodanje, može djelovati na poboljšanje kardio-respiratornih funkcija u starijih osoba. Ako se tjelesno vježbanje preporučuje za dobrobit pojedinca, nužno je ispitati odnose između količine tjelesnog vježbanja, intenziteta i nekih komponenata kardio-respiratornih funkcija.

Metoda

Uzorak ispitanika činile su 42 gojazne do umjereno gojazne žene u postmenopauzi. Sve su ispitanice dobrovoljno sudjelovale u 15-tjednom trenažnom programu koji je osmislio i odobrio Ocjenjivački odbor Medicinskog fakulteta Sveučilišta u Michiganu. Trenažni postupak sastojao se od hodanja u jutarnjim satima po 4,83 km dnevno, pet dana u tjednu, u ukupnom trajanju od 15 tjedana. Za vrijeme hodanja trener je mjerio vrijeme i prijeđenu udaljenost. Ispitanice su podijeljene u u dvije skupine prema dobi, težini, tjelesnoj građi i inicijalnoj razini aerobnih sposobnosti, koje su vježbale intenzitom od 95% ili 125% ventilacijskog praga. Razina VP bila je prethodno utvrđena testom hodanja na pokretnom sagu. Prije početka i na kraju trenažnog programa, ispitanice su izmjerene s obzirom na ispitivane zavisne varijable: indeks tjelesne mase (*body mass index*, BMI), postotak tjelesne masti, VP i FS koja je mjerena Polar tahometrom za vrijeme hodanja na traci. Promjene u srčanoj frekvenciji i krvnom tlaku procjenjivane su na temelju njihova praćenja pomoću TM2421 ambulantnog FS-RR monitora tijekom sedam dana (A&D Company, Tokyo, Japan). Ispitanice nisu sudjelovale ni u kakvom dodatnom programu vježbanja.

Rezultati

Dnevni uzorak promjena FS i krvnog tlaka, uključujući i prosječnu procjenu srčanog ritma (MESOR), veličinu dnevnih otklona ispod i iznad prosječnog ritma (dvostruka amplituda), kao i standardne devijacije FS i RR izračunate su nakon prilagođavanja FS i RR 24- i 12-satnoj kosinusnoj krivulji (Halberg, 1969). Pomoću studentovog *t*-testa izračunate su promjene u veličinama parametara zavisnih varijabli. Utjecaj treninga na antopometrijske varijable nije se odrazio na veličinu BMI-a, dok je došlo do statistički značajnog smanjenja tjelesne masti ($t=3.591$, $p<0.001$). Umjereni gubitak tjelesne masti (2-3%), koji nije popraćen smanjenjem tjelesne mase, sukladan je nalazima prethodnih istraživanja koja govore o paralelnom povećanju bezmasne tjelesne mase (Ballor i Poehlman, 1994). Intenzitet treninga djelovao je stimulativno na aerobni kapacitet, i to preko povećanja VP-a. Trening je proizveo 4.5%-tno povećanje VP-a za svakih 10% povećanja trenažnog intenziteta. S druge strane, trenažni je intenzitet proizveo manje izraženi učinak na mjere funkcija RR-a. Trenažni intenzitet djelo-

vao je i na smanjenje sistoličkog i dijastoličkog krvnog tlaka (RRsis, RRdijast), srčane frekvencije (FS) i srčanog ritma (MESOR), a učinak je bio proporcionalan intenzitetu vježbanja. Međutim, niti jedna od navedenih promjena nije bila statistički značajna. Kod jedne ispitanice, koja je vježbala sa 118.5% svog plućnoga kapaciteta, utvrđeno je smanjenje vrijednosti RRsis i MESOR (3mm Hg niže, mjereno pomoću Circadian Hyper Amplitude Tension, CHAT) nakon treninga. Međutim, nisu zabilježeni promjene krajnjih cirkadijanih RRsis i RRdijast dvostrukih amplituda.

Zaključak

Ovo istraživanje omogućuje donošenje ograničenih zaključaka o učinkovitosti hodanja radi poboljšanja kardio-respiratornih funkcija u postmenopauzalnih žena. Opći zaključak jest da je 15-tjedni trenažni postupak dovoljan za povećanje aerobnoga kapaciteta, a da će veličina poboljšanja, sukladno Karvonenovoj observaciji (Karvonen, 1957), proporcionalno ovisiti o trenažnom intenzitetu. Primijenjeni program vježbanja dovodi do značajnog povećanja aerobnoga kapaciteta, a dobiveni trend smanjenja parametara FS i RR nije statistički značajan. Unutar mjerenih kardio-respiratornih varijabli, samo su se neke komponente (RRsis, RRdijast, FS, MESOR) promijenile pod utjecajem trenažnog intenziteta. Također je došlo do neznačajnog smanjenja mjera njihove varijabilnosti. Iako rezultati ovog istraživanja nemaju dostatnu snagu da bismo definitivno mogli utvrditi ove odnose, razumno je reći da umjerena trenažna aktivnost (hodanje) može imati povoljne modifikacijske učinke na MESOR i prosječne vrijednosti varijabli RRsis, RRdijast i FS, što je sukladno nalazima nekih prethodnih istraživanja. Intenzitet treninga nije utjecao na stopu ubrzanja FS za vrijeme progresivnog testa hodanja na traci niti na promjenu varijabilnosti mjera FS i RR. Konačno, može se zaključiti kako je RR pod kontrolom većeg broja faktora, što je vidljivo kada se RR abnormalnosti razmatraju izvan cirkadijanog konteksta. Nalazi dobiveni mjerenjem CHAT na primjeru jedne ispitanice (Borer i sur, 2002) pokazuju povećani rizik od srčanog udara usprkos normalnim prosječnim vrijednostima krvnog tlaka. Taj primjer ukazuje na različite mehanizme koji koordiniraju RR, FS i MESOR, s jedne, i cirkadijane promjene, s druge strane. Za bolje razumijevanje učinaka tjelesnog vježbanja na te kardio-respiratorne varijable potrebna su daljnja istraživanja. Koristan prilog tome bila bi upotreba prijenosnih monitora FS-RR koji bilježe srčane ritmove, kao i cirkadijane promjene (Halberg, sur. 2002).