

K. NAZAR¹, A. GAŚSIOROWSKA¹, T. MIKULSKI¹, G. CYBULSKI¹,
W. NIEWIADOMSKI¹, J. SMORAWIŃSKI², K. KRZEMIŃSKI¹, A.W. ZIEMBA¹,
A. DORSZ², H. KACIUBA-UŚCIELKO¹

EFFECT OF 6-WEEK ENDURANCE TRAINING ON HEMODYNAMIC AND NEUROHORMONAL RESPONSES TO LOWER BODY NEGATIVE PRESSURE (LBNP) IN HEALTHY YOUNG MEN

1. Department of Applied Physiology, Medical Research Centre,
Polish Academy of Sciences, Warsaw, Poland

2. Department of Sport Medicine, Academy of Physical Education, Poznan, Poland

Endurance training is considered as a factor impairing orthostatic tolerance although an improvement and lack of effect have been also reported. The mechanisms of the changes and their relation to initial tolerance of orthostasis are not clear. In the present study, effect of moderate running training on hemodynamic and neurohormonal changes during LBNP, a laboratory test simulating orthostasis, was investigated in subjects with high (HT) and low (LT) tolerance of LBNP. Twenty four male, healthy subjects were submitted to graded LBNP (-15, -30 and -50 mmHg) before and after training. During each test heart rate (HR), stroke volume (SV) and blood pressure, plasma catecholamines, ACTH, adrenomedullin, atrial natriuretic peptide, and renin activity were determined. Basing on initial test, 13 subjects who withstood LBNP at -50 mmHg for 10 min were allocated into HT group and 11 subjects who earlier showed presyncopal symptoms to LT group. Training improved LBNP tolerance in six LT subjects. This was associated with attenuated rate of HR increase and SV decline (before training, at -30 mmHg Δ HR was 21 ± 4 beats/min and Δ SV – -36 ± 8 ml while after training the respective values were 8 ± 4 beats/min and -11 ± 6 ml). No differences in hemodynamic response were found in HT subjects and those from LT group whose LBNP tolerance was unchanged. In neither group training affected neurohormonal changes except inhibition of plasma ACTH rise in subjects with improvement of LBNP tolerance. It is concluded that some subjects with low orthostatic tolerance may benefit from moderate training due to improvement of cardiac function regulation.

Key words: *orthostatic tolerance, training, heart rate, blood pressure, stroke volume, catecholamines, plasma renin activity, ACTH, adrenomedullin, atrial natriuretic peptide*

INTRODUCTION

Several studies have demonstrated that endurance athletes show propensity towards the lower orthostatic tolerance than untrained people (see 1,2). Levine *et al.* (3) demonstrated that endurance-trained athletes show greater stroke volume (SV) decrease than untrained subjects during lower body negative pressure (LBNP), which is a laboratory technique that simulates orthostasis. The authors suggested that athletes' heart has greater ventricular compliance and steeper volume-pressure curve resulting from training-induced remodeling of the heart. The other mechanisms that are considered as being responsible for lower tolerance of gravitational stress in endurance athletes include attenuated carotid baroreflex responsiveness under condition of progressive decrease of central blood volume (4,5), diminished reactivity of blood vessels to the sympathetic stimulation (6) and increased leg venous compliance (2). The influence of the latter factor on the orthostatic tolerance is probably of minor importance (7). The study of Hildebrandt *et al.* (8) indicated that endurance-trained subjects had higher rate of capillary filtration in the calves during an orthostatic tilt test than the untrained individuals. This mechanism could also play a role in the intolerance of gravitational stimuli, depending on the preexisting fluid state.

On the other hand, there are data indicating that aerobic fitness does not contribute to prediction of orthostatic intolerance (9-12). Moreover, Shwartz (13) demonstrated that endurance runners had better orthostatic tolerance than unfit individuals. Some authors reported also that improving aerobic capacity of moderately fit individuals was associated with higher blood pressure during standing (14) and better tolerance of LBNP (15), head-up tilting (16) or the combination of tilt with LBNP (17,18). Correlation between changes in orthostatic tolerance and plasma volume ascertained by Convertino (15) and Mtinangi and Hainsworth (17, 18) strongly suggests that the beneficial effect of training results from plasma volume expansion. However, the data on regulatory mechanisms associated with the training-induced improvement of the orthostatic tolerance are equivocal. Both the increase (15) and a decrease in the baroreflex sensitivity (17,18) were reported. Convertino (15) found an enhancement in cardiovascular response to LBNP while Mtinangi and Hainsworth (17, 18) failed to find differences in blood pressure during combined orthostatic test performed after training.

The effect of training on neurohormonal responses to orthostatic stimuli is rather poorly recognized. Convertino (15) did not find significant alterations in plasma concentrations of norepinephrine and vasopressin or plasma renin activity during LBNP after 10 weeks of endurance training in healthy subjects, however, this author determined these variables only at the point of syncope. On the other hand, Winker *et al.* (16) demonstrated significantly smaller increase in plasma norepinephrine level during 75° tilt test in subjects with idiopathic orthostatic intolerance after 12 week of training.

Data obtained by Mtinangi and Hainsworth (17) suggested that the training-induced improvement of orthostatic tolerance in healthy subjects is greater in those with poor initial tolerance. In order to test this hypothesis in the present study the effect of moderate endurance training on LBNP tolerance was determined in young men who are allocated to the high and low tolerant groups according to the pre-training test (19). Our second aim was to obtain a deeper insight of the effect of training on hemodynamic and neurohormonal changes occurring in the response to LBNP.

MATERIAL AND METHODS

Subjects.

Twenty four healthy male volunteers (age: $20.8 \pm (SD)0.9$ yrs, body mass: $74.2 \pm (SD)7.1$ kg, height: $176.9 \pm (SD)4.3$ cm, maximal oxygen uptake: $47 \pm (SD)4$ ml·kg⁻¹·min⁻¹) took part in this study after giving an informed consent. All of them were the students of the Military Academy, lived in the Students' Hostel, had similar diet and daily activities. They were physically active but not specifically trained. The study protocol was approved by Ethical Committee of the Medical Academy in Poznan, Poland.

Study protocol. The students were submitted to LBNP test twice: before and after six weeks of endurance training. The training program included five sessions per week. Each daily session (60-90 min) consisted of 10 min jogging at a heart rate (HR) of 120-130 beats/min, 30 min of constant rate running at a HR corresponding to 60-70% of the predetermined VO₂max, interval running with the maximal speed for 150-200 m., and 30 min of swimming or playing soccer.

Three days before both LBNP tests they underwent the incremental exercise test on bicycle ergometer until volitional exhaustion in order to determine their maximal oxygen uptake (VO₂max). Exercise load was increased by 50 W every three minutes starting from 50 W.

In the evening before LBNP test the subjects reported to the laboratory where they spent the night. In the morning, after an overnight fast they were carried on the stretcher in the supine position to the LBNP chamber, which was sealed at the level of iliac crest. Thirty min after inserting catheter to the antecubital vein and instrumentation two baseline circulatory measurements were made and blood sample for hormone and hematocrit (HTC) determinations was taken at ambient pressure. Then the subjects were submitted to serial LBNP: 10 min at -15 mmHg, 10 min at -30 mmHg and 10 min at -50 mmHg or until onset presyncope signs or symptoms, and after 10 min of the recovery period at ambient pressure. The presyncope symptoms and signs included lightheadedness, nausea, sweating, narrowing of vision and rapid drop of systolic blood pressure by more than 20 mmHg or bradycardia. Before and every 3 min during LBNP and the recovery period blood pressure (BP), heart rate (HR), stroke volume (SV), cardiac output (CO) were measured. Blood samples for epinephrine (E), norepinephrine (NE), and ACTH concentrations and plasma renin activity (PRA) were taken at the end of the second (-30 mmHg), the third (-50 mmHg) stage of LBNP or immediately after onset of presyncope symptoms as well as at the end of the recovery period. Hematocrit and adrenomedullin (ADM) and atrial natriuretic peptide (ANP) concentrations were determined only before and at the end of final LBNP stage.

Methods. For LBNP the chamber with the pressure control system was used (ITAM, Zabrze, Poland). It allows to change pressure within approx. 15 s. Heart rate was monitored and recorded by the Sport Tester (PE 3000, Polar Electro, Finland). Blood pressure (BP) was measured on brachial artery by electronic sphygmomanometer. Stroke volume (SV) and cardiac output (CO)

were determined by impedance cardiography (ICG) using a monitoring device designed in the Medical Research Centre, Polish Academy of Sciences by Cybulski et al. (20). The measurement is based on the tetrapolar technique: the sinusoidal alternating current (95 kHz) is applied via the pair of electrodes placed on the chest, the voltage signal is collected from other electrodes and demodulated. The ECG and the first derivative of the impedance signal are sampled at the rate of 200 Hz. The system allows for the off-line, beat-to-beat automatic evaluation of stroke volume (SV), and HR and cardiac output (CO). Validity of SV measurements was determined using echocardiography ($r=0.90$, $n=21$, $p<0.001$) (21). Mean blood pressure was calculated as diastolic BP plus 0.33 of difference between systolic and diastolic BP then the total peripheral resistance (TPR) was calculated dividing mean BP by CO. Changes in plasma volume during LBNP were calculated from differences in blood hematocrit. Mean hematocrit values were obtained by multiplying venous blood hematocrit by 0.8723.

Blood samples for catecholamine determination were taken to the chilled polyethylene tubes containing EGTA and reduced glutathione while for other hormones the tubes with EDTA with aprotinin (Trasylool, 500 KIU/ml blood) were used. All samples were centrifuged within 30 min at 3000 rpm at 4°C, and stored at -70°C until further processed.

Plasma [E] and [NE] were measured using high pressure liquid chromatography. Other hormones were determined by radioimmunoassay using CIS bio international (France) kits for plasma [ACTH] and [ANP], Phoenix Laboratories.(Belmont, CA, USA) reagent set for [ADM] and Immunotech, Angiotensin I kit (Prague, Czech Republic) for PRA.

Data analysis and statistics. After the first LBNP test the subjects were divided into two groups: high tolerance group (HT) which included 13 students who completed the test and low tolerance group (LT) which consisted of 11 persons who showed presyncopal signs or symptoms at -30 mmHg or within the first 3 min of the final LBNP stage. The comparison of data obtained in these two groups before training has been presented elsewhere (19). To evaluate the effect of training in both groups two way analysis of variance for repeated measures and *post hoc* a paired Student's *t* test were used. As the level of significance $p<0.05$ was accepted. The data are presented as means with standard errors (SEM) unless otherwise stated.

RESULTS

Training caused an increase in $VO_2\text{max}$ from 46.1 ± 1.4 to 52.5 ± 2.1 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ($p<0.001$) in the group with high LBNP tolerance (HT) and from 48.0 ± 0.9 to 51.1 ± 2.4 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ($p<0.01$) in the group with low LBNP tolerance (LT). After training 12 subjects from group HT completed the LBNP test while in one person the test was terminated at -30 mmHg because of presyncopal symptoms. In LT group in 6 subjects LBNP was improved after training and in 5 persons it remained unchanged. There was no difference in training-induced increases in $VO_2\text{max}$ between the subjects who showed improvement of LBNP tolerance and those whose tolerance was not affected.

Effect of training on cardiovascular responses to LBNP

Analysis of variance showed that before training in both groups LBNP caused a significant increase in HR ($p<0.001$) and TPR ($p<0.001$) and a decrease in SV,

CO ($p<0.001$), systolic BP ($p<0.001$) and plasma volume ($p<0.01$). Diastolic BP decreased during LBNP only in LT group ($p<0.001$).

Cardiovascular responses to LBNP before and after training are presented in Figs. 1 and 2. In HT group training resulted only in the tendency toward an increase of SV ($p=0.065$) and CO ($p=0.069$) values without changes in the time-course of their responses to LBNP. Heart rate, systolic and diastolic BP and TPR in this group were not modified. In LT group analysis of values obtained at 0, -15 and -30 mmHg

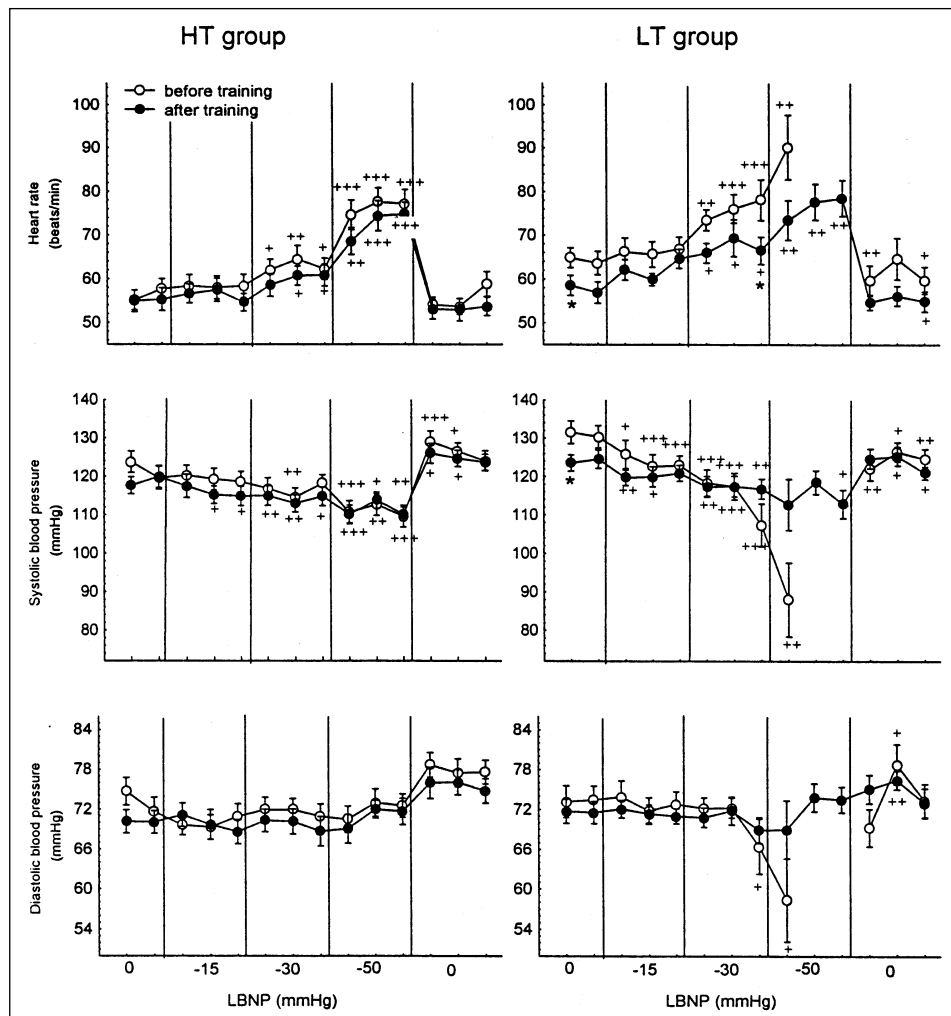


Fig. 1. Time courses of changes in heart rate, systolic and diastolic blood pressure during LBNP before and after training; crosses denote significant effect of LBNP : + $p<0.05$, ++ $p<0.01$, +++ $p<0.001$; asterisks denote significant differences between values obtained before and after training: * $p<0.05$.

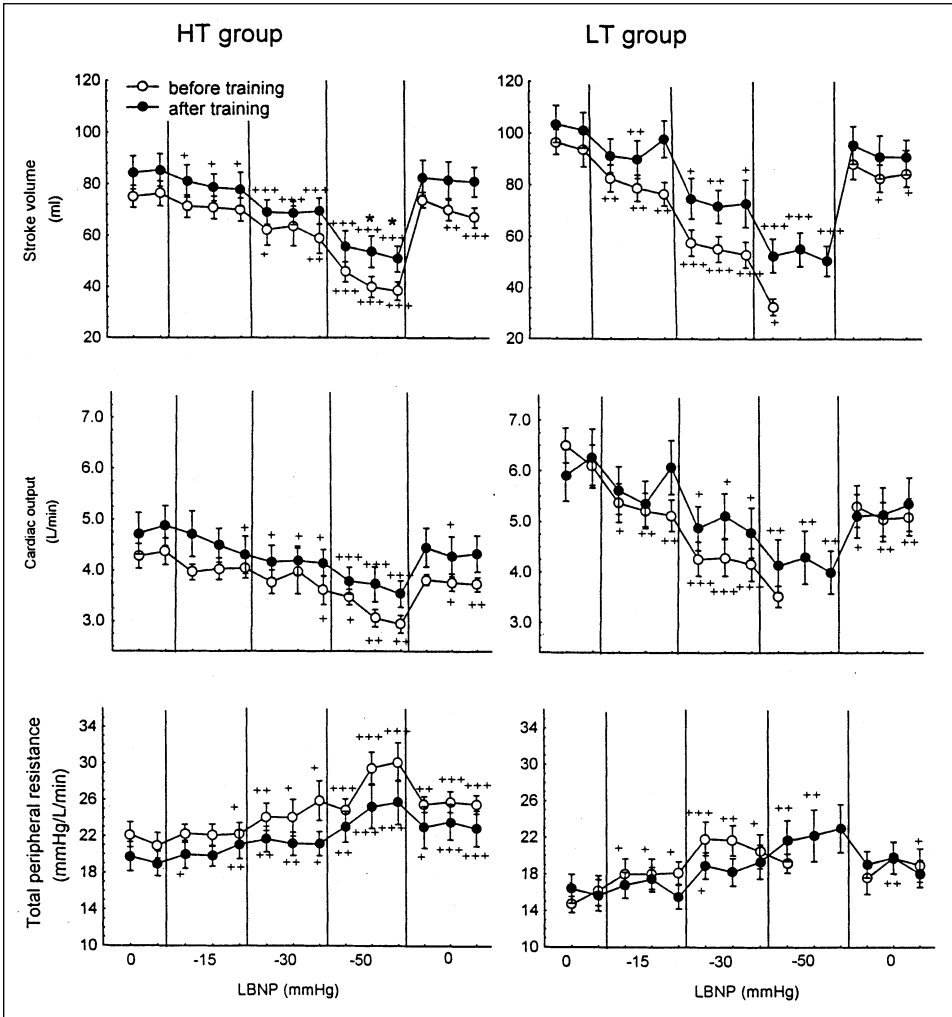


Fig. 2. Time courses of changes in stroke volume, cardiac output and total peripheral resistance during LBNP before and after training; crosses denote significant effect of LBNP : + p<0.05, ++ p<0.01, +++ p<0.001.

by ANOVA revealed significant reduction of HR ($p<0.05$) and tendency toward an increase of SV ($p=0.080$) after training. In contrast to HT group in LT subjects interaction of the effects of training and LBNP on cardiovascular indices was found to be significant. There was attenuation of HR ($p<0.05$), SV ($p<0.001$), CO ($p<0.001$), systolic BP ($p<0.001$), diastolic BP ($p<0.001$) and TPR ($p<0.05$) responses. Neither in LT nor in HT group training affected significantly the decreases in plasma volume associated with LBNP the respective values are 7.3 ± 1.8 and 7.4 ± 2.9 % before training and 7.0 ± 2.1 and 10.0 ± 2.2 after training.

In five subjects from LT group whose LBNP tolerance was not affected by training cardiovascular indices before and after training did not differ significantly. Comparison of this subgroup with six subjects who showed improved tolerance by training did not reveal any differences before training whilst after training during LBNP at -30 mmHg their diastolic BP was lower ($p<0.05$) and there was a tendency towards smaller SV ($p=0.08$).

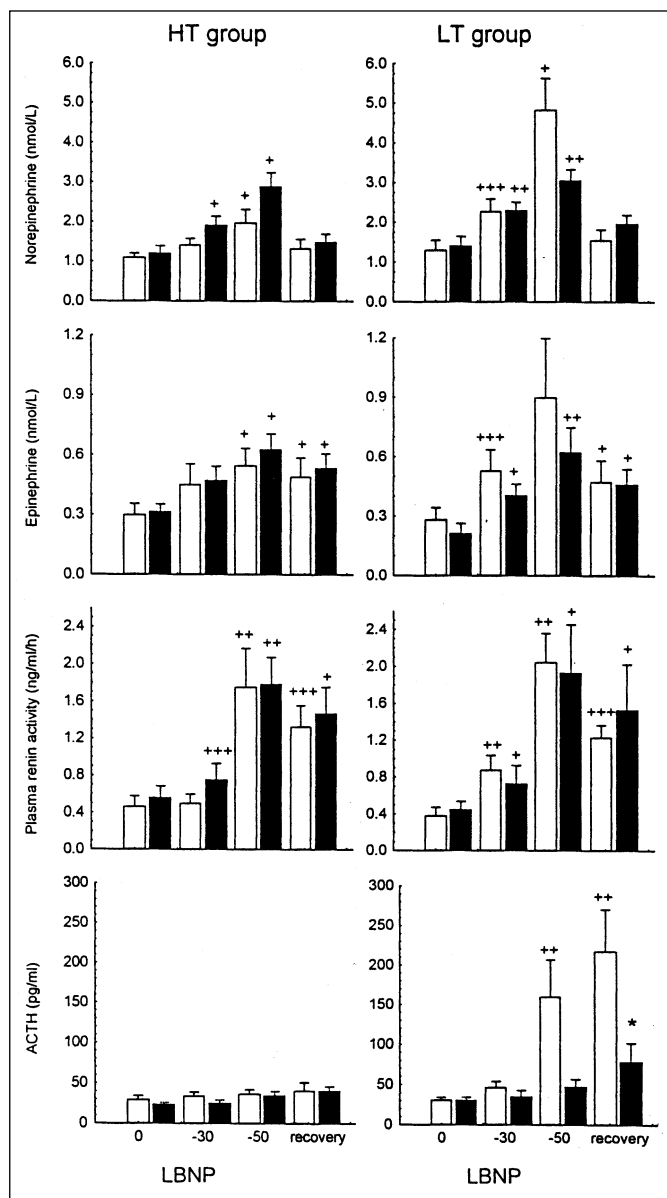


Fig. 3. Changes in plasma concentration of catecholamines and ACTH and plasma renin activity during LBNP before (white bars) and after (black bars) training; crosses denote significant effect of LBNP: $^+p<0.05$, $^{++}p<0.01$, $^{+++}p<0.001$; asterisks denote significant differences between values obtained before and after training: $*p<0.05$.

Effect of training on neurohormonal responses to LBNP

Before training in both groups plasma levels of NE ($p < 0.001$), E ($p < 0.001$), PRA ($p < 0.001$) and ADM ($p < 0.01$) increased while plasma ANP ($p < 0.01$) concentration decreased during LBNP. Plasma ACTH significantly rose ($p < 0.01$) in the final stages of LBNP test only in LT group. Training did not affect significantly neurohormonal responses to LBNP except that it reduced plasma ACTH level in LT group ($p < 0.05$) (Fig. 3-4).

In five subjects from LT group whose LBNP tolerance was not affected by training plasma concentrations of hormones before and after training did not differ significantly. Comparison of this subgroup with six subjects who showed improved tolerance by training did not reveal any differences before training whilst after training the plasma level of ACTH during recovery after LBNP was significantly higher.

DISCUSSION

The present data confirmed the previous studies (9,11,12,14-18) showing that moderate training does not impair orthostatic tolerance in most of healthy men and causes an improvement in some subjects with low tolerance. Since after training $VO_2\text{max}$ of our subjects was below $55 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ our data support the concept of Convertino (15) that endurance training does not compromise and may improve orthostatic tolerance when aerobic capacity does not exceed 55-60

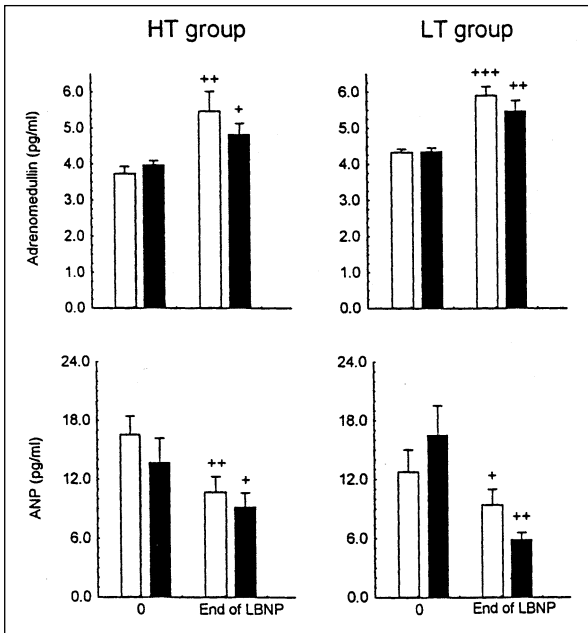


Fig. 4. Changes in plasma concentration of adrenomedullin and atrial natriuretic peptide (ANP) during LBNP before (white bars) and after (black bars) training; crosses denote significant effect of LBNP : + $p < 0.05$, ++ $p < 0.01$.

ml·kg⁻¹·min⁻¹. However, we found an improvement of LBNP tolerance only in 6 from 11 subjects. Basing on the measured indices, we are unable to provide the explanation why there was a great numbers of "nonrespondents". It should be noted, that the beneficial effect of training on the orthostatic tolerance reported by other authors also did not occur in all subjects examined. For example, Winker et al. (16) found this effect in 10 from 16 subjects. The authors suggested existence of some genetic cause of orthostatic intolerance which cannot be affected by training.

In the previous investigation performed with the same subjects before training we attempted to determine which of the cardiovascular and neurohormonal changes occurring at submaximal LBNP could be considered as predictive for low tolerance of central hypovolemia induced by this stimulus (19). It appeared that the most pronounced difference between subjects with low and high tolerance was the steeper decrease in stroke volume in the former which suggested inadequacy in cardiac function regulation. After training in the HT group there were tendencies towards higher SV and CO but there were no differences in time-course of cardiovascular responses to LBNP. This data were similar to those of Lightfoot et al. (10) and Convertino (15) who did not find any changes in cardiovascular responses to submaximal level of LBNP after 10 weeks of endurance training in young healthy men. However, in subjects from LT group who improved their tolerance of LBNP after training the rate of HR increase and the decreases of SV, CO, and BP were attenuated. As a result, significant interaction of the effects of training and LBNP was ascertained by ANOVA in the whole LT group including also the subjects whose tolerance of LBNP and cardiovascular responses were not altered by training. As it was emphasized by several authors (15-18), the improvement of orthostatic tolerance by training may be related to plasma volume expansion resulting in the increase in SV. Winker et al. (16) who investigated heart rate variability as an index of parasympathetic-sympathetic balance in subjects with idiopathic orthostatic intolerance subjected to training suggested the important role of enhanced parasympathetic tone leading to decreased HR response to baroreceptor activation and subsequent increase in SV. However, the improvement of hemodynamic function of the heart due to increased contractility by training (22) could be also considered as a factor mitigating SV decrease during LBNP.

The role of increasing peripheral resistance during LBNP in determining tolerance of this stimulus is unclear. From one hand it attenuates a drop in blood pressure but on the other hand it decreases SV. Before training total peripheral resistance was lower in LT than in HT group (19). Training did not reduce this difference until the highest level of LBNP (-50 mmHg) where similar values were obtained in both groups. However, in LT group the increase in TPR at lower levels of LBNP was less pronounced after than before training. The latter finding is consistent with the data of Winker et al (16) who showed an improvement in orthostatic tolerance after training in spite of decreased TPR.

Our study failed to demonstrate significant effect of training on the plasma volume decreases during LBNP. Thus, the data did not confirm the training-induced enhancement of the rate of capillary filtration suggested by Hildebrandt et al.(8).

Analysis of neurohormonal indices in HT and LT groups before training performed in the previous paper demonstrated (19) that there were no significant differences between HT and LT groups in the baseline plasma catecholamine and ACTH concentrations or plasma renin activity (PRA) while plasma level of adrenomedullin (ADM) was significantly higher in LT than in HT group. The LBNP-induced changes in plasma norepinephrine concentration and PRA were higher in LT than in HT group and similar tendency was noted in the case of epinephrine. The increases in plasma ADM were similar in both groups. The significant increase in plasma ACTH was found in the final stage of LBNP and during recovery only in LT groups. In the present study the plasma atrial natriuretic peptide (ANP) was additionally measured since this hormone plays a role in the adjustment to gravitational stimuli and its secretion is diminished during LBNP (see 23). It appeared that neither the baseline level of this hormone nor the magnitude of its decrease during LBNP discriminated the low and high tolerant groups. The main finding of the present study is that training did not modify significantly the neurohormonal response to LBNP except reduction in plasma ACTH increase in LT group. The rise of this hormone concentration occurred at the final stages of the test and only in those who did not improve their LBNP tolerance after training. This effect indicates that the rise of ACTH secretion is connected with the stress of imminent syncope.

In conclusion, our data showed that moderate endurance training does not compromise tolerance of central hypovolemia induced by LBNP, moreover, some young subjects with low tolerance of this stimulus may benefit from training. This is associated with less pronounced increase of HR and attenuated rate of SV and CO decline during the test without alteration in the vasoactive hormone responses.

Acknowledgments: The study was partly supported by the Polish State Committee for Scientific Research, grant no: 6 PO5D 012 20. The authors wish to express their gratitude to Dr C. Młynarczyk for elaboration of training program and supervised its accomplishment and to Mrs. B. Kurek, Mrs. L. Wiśnik and Mrs. M. Cisowska -Wienclaw for their excellent technical assistance.

REFERENCES

1. Geelen G, Greenleaf JE. Orthostasis: exercise and exercise training. *Exerc Sport Sci Rev* 1993; 21:201-230.
2. Raven PB, Pawelczyk JA. Chronic endurance exercise training: a condition of inadequate blood pressure regulation and reduced tolerance to LBNP. *Med. Sci Sports Exerc* 1993; 25: 713-721.

3. Levine BD, Lane LD, Buckey JC, Friedman DB, Blomquist CG. Left ventricular pressure-volume and Frank-Starling relations in endurance athletes. Implications for orthostatic tolerance and orthostatic performance. *Circulation* 1991; 84: 1016 -1023.
4. Smith SA, Querry RG, Fadel PJ, Welch-O'Connor RM, Olivencia-Yurvati A, Shi X, Raven PB. Differential baroreflex control of heart rate in sedentary and aerobically fit individuals. *Med Sci Sports Exerc* 2000; 32:1419-1330.
5. Ogoh S, Volianitis S, Nissen P, Wray DW, Secher NH, Raven PB. Carotid baroreflex responsiveness to head-up tilt- induced central hypovolaemia. *J Physiol* 2003; 551: 601-608.
6. Fadel PJ, Stromstad M, Hansen J, Sander N, Horn K, Ogoh S, Smith ML, Secher NH, Raven PB. Arterial baroreflex control of sympathetic nerve activity during hypotension: effect of fitness. *Am J Physiol: Heart Circ Physiol* 2001; 280:H2524-H2532.
7. Hernandez JP, Franke WD. Age- and fitness-related differences in limb venous compliance do not affect tolerance to maximal lower body negative pressure in men and women. *J Appl Physiol* 2004; 97: 925-929.
8. Hildebrandt W, Schutze H, Stegemann J. Higher capillary filtration rate in the calves of endurance-trained subjects during orthostatic stress. *Aviat Space Environ Med* 1993; 64: 380-385.
9. Convertino VA, Satcher TM, Goldwater DJ, Alford WR. Aerobic fitness does not contribute to prediction of orthostatic intolerance. *Med Sci Sports Exerc* 1986; 18: 551-556.
10. Lightfoot JT, Claytor RP, Torok DJ, Journell TW, Fortney SM. Ten weeks of aerobic training does not affect lower body negative pressure responses. *J Appl Physiol* 1989; 67: 894-901.
11. Hernandez JP, Franke WD. Effects of a 6-mo endurance-training program on venous compliance and maximal lower body negative pressure in older men and women. *J Appl Physiol* 2005; 99:1070-1077.
12. Hernandez JP, Karandikar A, Franke WD. Effects of age and fitness on tolerance to lower body negative pressure. *J Gerontol A Biol Sci Med Sci* 2005; 60:782-786.
13. Shvartz E. Endurance fitness and orthostatic tolerance. *Aviat Space Environ Med* 1996; 67: 935-939.
14. Cybulski G, Krzeminski K, Niewiadomski W, Nazar K. The influence of endurance training on the transient haemodynamic response to orthostatic manoeuvre. *J Physiol Pharmacol* 1999; 50:275-286.
15. Convertino VA. Endurance exercise training conditions of enhanced hemodynamic response and tolerance of LBNP. *Med Sci Sports Exerc* 1993; 25: 705-712.
16. Winker R, Barth A, Bidmon D, Ponocny I, Weber M, Mayr O, Robertson D, Diedrich A, Maier R, Pilger A, Haber P, Rudiger HW. Endurance exercise training in orthostatic intolerance: a randomized, controlled trial. *Hypertension* 2005; 45: 391-398.
17. Mtinangi BL, Hainsworth R. Effects of moderate exercise training on plasma volume, baroreceptor sensitivity and orthostatic tolerance in healthy subjects. *Exp Physiol* 1999; 84:121-130.
18. Mtinangi BL, Hainsworth R. Increased orthostatic tolerance following moderate exercise training in patients with unexplained syncope. *Heart* 1998; 80: 596-600.
19. Gąsiorowska A., Nazar K., Mikulski T., Cybulski G., Niewiadomski W., Smorawiński J., Krzemiński K., Porta S., Kaciuba-Uściłko H. Hemodynamic and neuroendocrine predictors of lower body negative pressure (LBNP) intolerance in healthy young men. *J Physiol Pharmacol* 2005; 56: 179-193.
20. Cybulski G, Ksiazkiewicz A, Lukasik W, Niewiadomski W, Palko T. Central hemodynamics and ECG ambulatory monitoring device with signals recording on PCMCIA memory cards. *Med Biol Eng Comp* 34, (suppl.1): 22-34, 1996.
21. Cybulski G, Miskiewicz Z, Szulc J, Torbicki A, Pasierski T. A comparison between the automatized impedance cardiography and pulsed - wave Doppler echocardiography methods

- for measurements of stroke volume (SV) and systolic time intervals (STI). *J. Physiol. Pharmacol* 1993; 44: 251-258.
22. Slordahl SA, Madslie VO, Stoylen A, Kjos A, Helgerud J, Wisloff U. Atrioventricular plane displacement in untrained and trained females. *Med Sci Sports Exerc* 2004; 36:1871-1875.
 23. Norsk P. Gravitational stress and volume regulation. *Clin Physiol* 1992; 12: 505-526.

Received: September 9, 2005

Accepted: April 28, 2006

Author's address: Krystyna Nazar, Department of Applied Physiology, Medical Research Centre, Polish Academy of Sciences, 5 Pawińskiego str., 02-106 Warsaw, Poland. Tel/fax (22) 6684554. E-mail: nazar@cmdik.pan.pl