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Heart rate variability in women exposed to very cold air (−110 °C) during whole-body cryotherapy

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Abstract

Heart rate monitoring was used to measure heart rate variability (HRV) at thermoneutral conditions (Ta 24 °C) in healthy women resting in supine position before and after acute and after repeated (3 times a week during a 3-month period) whole-body cryotherapies (WBC), at −110 °C. The observed acute cooling-related increase in high frequency power (HFP) of RR-intervals indicates an increase in cardiac parasympathetic modulation. After 3 months of repeated WBC the increase in parasympathetic tone was attenuated, which may be interpreted as an adaptation of autonomic function. The repeated WBC exposures-related increase in resting low frequency power (LFP) of RR-intervals during the 3 months resembles the response observed related to exercise training.

Keywords: Whole-body cryotherapy; Heart rate variability; Adaptation of the autonomic nervous system; Women

1. Introduction

Acute cold exposure induces tachycardia and peripheral vasoconstriction with concomitant increase in blood pressure. These responses are under the control of the sympathetic and parasympathetic nervous systems (Finley et al., 1979; Heath and Downey, 1990). Earlier works on cardiovascular responses to cold have been done by immersing extremities into the cold water (cold pressor tests) (Janský et al., 2003; for review see LeBlanc et al., 1975), by exposing face to wind or by applying ice packs to the face (LeBlanc et al., 1976). Facial cooling increases blood pressure and results in bradycardia in resting subjects, whereas during a cold pressor test both heart rate and blood pressure increase (LeBlanc et al., 1975). Cardiovascular responses to whole-body cold stress have been also studied intensively (Janský et al., 1996).

Previously, the effect of autonomic regulations on cardiovascular function has mostly been characterized by changes in heart rate and blood pressure. In recent works, heart rate variability (HRV) has been used as an indirect indicator of the activity of the autonomic nervous system. To our knowledge, no data are available on HRV after the whole-body cold stress.

Whole-body cryotherapy (WBC) is a mode of cold therapy in which patients are exposed to very cold air (−110 °C) for 1–3 min in minimal clothing. It is used to alleviate inflammation and pain in cases of arthritis (Fricke, 1989), and osteoarthritis (Metzger et al., 2000) and is used as pain reliever in cases of fibromyalgia (Samborski et al., 1992).

As rheumatoid arthritis is known to be associated with increased rates of cardiovascular diseases (Solomon et al., 2003; Turesson et al., 2004), it is important to ensure the safety of WBC. In our previous study, the WBC increased blood pressure significantly, but the increase was considered as safe for persons without cardiac problems (Westerlund et al., 2004).

In the present study we examined HRV changes in response to WBC. Although the study was a part of a project undertaken to ensure the safety of WBC for patients and personnel, it also allowed us to look on how an extreme cold air exposure influences cardiac autonomic

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regulation. The study was designed to evaluate changes in HRV induced by an acute WBC (−110 °C) in healthy women and to find out whether or not the repeated WBC causes adaptation in this response.

2. Methods

2.1. Subjects

Ten female subjects were reached by an announcement in a local newspaper and they agreed to participate after having been informed of the study requirements. All the subjects were healthy without medication. They did not use oral contraceptives. Their age (mean ± SD) was 38 ± 3 years, height 167 ± 7 cm, weight 68 ± 14 kg, BMI 24 ± 3 kg/m^2, systolic blood pressure 126 ± 12 mmHg and diastolic blood pressure 79 ± 10 mmHg. The subjects were moderately physically active and they were advised to maintain the same physical activity level during the experiment as before that. The experimental protocol and procedures were approved by the Ethics Committee of the Päijät-Häme District.

2.2. Experimental design

WBC exposures took place in a specially built, temperature-controlled unit (Zimmer Elektromedizin, Germany), which consists of three rooms with different temperatures (−10, −60 and −110 °C). The two first exposures were made in the pre-rooms only (temperatures −10 and −60 °C) and the third exposure was made in the therapy-room (−110 °C). Each exposure lasted 2 min. During the WBC, subjects passed through the first two rooms before coming into the therapy-room. Air in the therapy room was dry and clear. The subjects wore a bathing suit, surgical mask, cap, gloves, socks and shoes. While in the therapy-room, the subjects were advised to move slightly their fingers and legs and to avoid holding their breath.

The subjects were not allowed to practise winter swimming for 3 months before and during the experiment. To study the long-term effect of cooling, subjects were exposed to the −110 °C temperature three times a week during 3 months.

2.3. Measurement of HRV

The subjects were requested not to eat, drink or smoke for 4 h before the heart rate measurements. Before the measurement they first rested in room temperature (24 °C) in the supine position for 10 min. Then they sat for 5 min before being exposed the WBC. After the WBC, the subjects rested in the supine position for 30 min at thermoneutral conditions.

Both before (PRE WBC) and after (POST WBC) the WBC, a 5-min supine rest HRV after 5 min lying was analysed. Additionally, from the 2 min recording during the WBC, a 1 min HRV (DURING WBC) in standing position was analysed. Maximal heart rate areas were not included into analysis. The measurements were conducted after the first exposure to WBC, and then monthly for the following 3 months (at the month 1, 2 and 3).

R-R-intervals were measured continuously by beat-to-beat with a heart rate monitor (Polar S 810) (Polar Electro, Kempele, Finland). Data transfer was done with the Polar IR Interface to the Polar Precision Performance SW 3.0 analysis software. HRV was evaluated in time and frequency domains, and the parameters were the following: R-R interval (RRI), standard deviation (SD), the square root of the mean-squared differences of RRI (RMSSD), low frequency power (LFP) (0.04–0.15 Hz), high frequency power (HFP) (0.15–0.40 Hz) and LFP/HFP-ratio.

To study the acute effect of the WBC on HRV, the values of heart rate variables at PRE WBC and POST WBC were compared. To evaluate adaptation to WBC, differences between PRE and POST WBC the heart rate variables at the start of the experiment and after repeated WBC (3 times a week for 3 months = 'cold training') were compared. To evaluate the 'cold training' effect on supine resting HRV, the PRE WBC heart rate variables measured at the start of the experiment and at the 3-month time point were compared. In addition, DURING WBC HRV at the start of the experiment and at the 3-month time point was compared.

2.4. Statistics

The results were expressed as means and standard deviations (SD). The normality of variables was evaluated by the Shapiro-Francia test with Monte Carlo p-value. However, as variable values had wide dispersion, bootstrap estimation was used to derive a 95% confidence interval; confidence intervals for the means were obtained by bias corrected bootstrapping (5000 replications) (Efron and Tibshirani, 1993). Statistical comparison of changes in outcome measurements was performed by using permutation test and Hommel's adjustments were performed to correct significance levels for the multiple test.

3. Results

Mean heart rates before and during WBC at the start of the experiment were 74 (± 11) and 91 (± 13) beats/min, respectively. After the 3 months period, the corresponding values were 73 (± 11) and 83 (± 10) beats/min, respectively.

No significant change in BMI (24.0 ± 3.4 kg/m^2 versus 23.8 ± 3.1 kg/m^2) measured at the start of the experiment and at the 3-month time point was observed.

In an acute experiment the RRI, the RMSSD and the HFP of RRI increased significantly from PRE WBC to POST WBC, but the LFP of RRI and the LFP/HFP-ratio did not change (Table 1). After the 3 month 'cold training', only the RRI and the RMSSD increased significantly from PRE WBC to POST WBC (Table 1). Initially, the RRI increased by 15.4% as a response to acute WBC, while after the 3 months it increased by 10.7%, only.

Table 1
Changes in HRV induced by an acute cold exposure (−110°) at first time and at 3 months

HRV	First time		3 months	
	Baseline mean (SD)	Change from baseline after the exposure mean (95% CI[a])	Baseline mean (SD)	Change from baseline after the exposure mean (95% CI[a])
RRI (ms)	852 (147)	131 (103 to 160)***	890 (171)	95 (52 to 138)**
SD (ms)	53 (17)	25 (12 to 34)*	64 (12)	16 (7 to 25)
RMSSD (ms)	36 (16)	19 (12 to 26)**	46 (17)	13 (7 to 18)**
LFP (ms^2)	972 (505)	804 (292 to 1331)	1479 (583)	302 (−232 to 837)
LFP (%)	68 (17)	−4 (−11 to 4)	67 (18)	−3 (−14 to 8)
HFP (ms^2)	528 (388)	572 (304 to 928)*	773 (622)	339 (52 to 625)
HFP (%)	32 (17)	8 (0 to 13)	33 (18)	3 (−8 to 14)
LFP/HFP	340 (276)	−134 (−264 to −20)	313 (234)	−102 (−229 to 23)

RRI, R-R-interval; SD, standard deviation; RMSSD, the square root of the mean-squared differences of RRI; LFP, low frequency power; HFP, high frequency power
*$p<0.05$, **$p<0.01$, ***$p<0.001$.
[a]Ninty-five percent confidence interval obtained by bias corrected bootstrapping (100 000 replications).

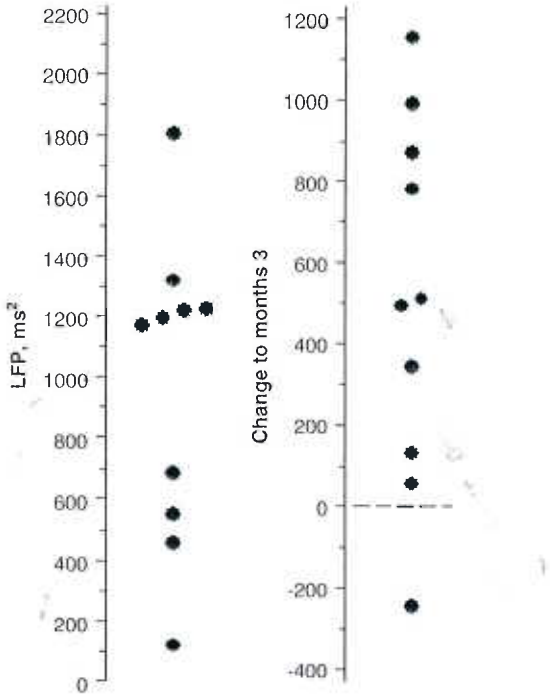

Fig. 1. Resting LFP measured before WBC and changes of LFP after repeated WBC at 3 month.

Repeated WBC exposures—for the 3 months—increased the PRE WBC LFP ($p<0.05$) (Fig. 1), but the PRE WBC RRI, the RMSSD, the HFP and the LFP/HFP-ratio did not change. Also HRV during the WBC showed no significant change during the 3 months (Table 2).

Table 2
HRV during WBC exposure and changes of HRV after repeated WBC at 3 month

HRV	Baseline mean (SD)	Change from baseline to 3 months mean (95% CI[a])	p-value[b]
RRI (ms)	676 (102)	82 (24 to 149)	0.25
SD (ms)	101 (37)	4 (−17 to 24)	0.81
RMSSD (ms)	43 (15)	9 (0 to 17)	0.36
LFP (ms^2)	1479 (878)	305 (−215 to 878)	0.55
LFP (%)	66 (11)	−3 (−12 to 9)	0.81
HFP (ms^2)	803 (547)	391 (−53 to 944)	0.41
HFP (%)	34 (11)	3 (−9 to 13)	0.80
LFP/HFP	243 (173)	11 (−144 to 166)	0.90

RRI, R-R-interval; SD, standard deviation; RMSSD, the square root of the mean-squared differences of RRI; LFP, low frequency power; HFP, high frequency power.
[a]Ninty-five percent confidence interval obtained by bias corrected bootstrapping (10 000 replications).
[b]p value adjusted by using Hommel's method.

4. Discussion

4.1. Acute effect of the WBC

Our data demonstrated an acute increase in supine resting RRI, resting RMSSD and resting HFP, the indices of cardiac parasympathetic modulation, as a response to extreme whole-body cold stress.

Since there are no previous data on HRV after the whole-body cold stress, our results can be compared with that obtained in exercise stress studies. Arai et al. (1989) found a marked reduction in the absolute HFP during maximal bicycle exercise test with a return to the normal level within few minutes after the exercise. In addition, Pober et al. (2004) has shown that a single bout of moderate exercise changes in the balance of the autonomic nervous system by increasing parasympathetic activity. This is generally considered as a healthy situation. Thus, our finding that the parasympathetic tone increases as a response to WBC, mimics the response induced by physical exercise stress. If the mechanism behind the increase in HFP of RRI after physical training is assumed to be similar

to that after WBC, the WBC could be considered as beneficial for human health.

Physiological explanations for the increased parasympathetic tone induced by the WBC could be due to cold stimulation of the face. Increased activity of the peripheral sympathetic system was also observed after face cold stimulation, however (LeBlanc et al., 1976) the combined activation of both component of the autonomous system might induce peripheral vasoconstriction and then bradycardia via baroreflexes (Friedman et al., 1996; Heath and Downey, 1990). Our finding that the parasympathetic tone increases as a response to WBC could be interpreted as a vagal rebound effect, which also occurs after the strong exercise stress (Arai et al., 1989; Hautala et al., 2001). The mechanism of the vagal rebound effect is unknown.

4.2. Cold training

The WBC-related acute increases in resting RRI and resting HFP and RMSSD appeared to be less prominent after the 3 months period of 'cold training'. In addition, the resting LFP measured before the WBC was found to be increased. No other changes in HRV during the 3-month cold training were observed.

Thermal sensation and comfort values have shown habituation after repeated WBC (Smolander et al., 2004). However, no adaptation of blood pressure was found after repeated WBC (Westerlund et al., 2004). In accordance with our results, LeBlanc et al. (1975) have found adaptation of heart rate in the cold face test in mailmen (working outdoors 6 h a day, 5 days a week). The enhanced bradycardia and the diminished fall in skin temperature of the cheek were found at the end of the winter (May) compared to the fall (October). In addition, Kauppinen and Vuori (1988) have reported lower mean resting heart rates in winter swimmers compared to controls. From exercise stress, the identical studies are missing.

Resting supine LFP of RRI before the WBC exposure seemed to increase after repeated cold exposures. This finding is in agreement with the finding of Iwasaki et al. (2003). They have found the increase in LFP with moderate exercise training program (3–4 times/week) from the baseline to 3- and 6-month time points (Iwasaki et al., 2003). On the other hand, in some randomised longitudinal training studies, a significant effect of aerobic training on daytime HRV in 24-h recording has been found to be due to increase in vagal modulation of cardiac activity (Ståhle et al., 1999; Tulppo et al., 2003). Cold adaptation may mimic exercise training effects, because the both stresses increase acutely the level of stress hormones and sympathetic activity (Huttunen et al., 2001; McArdle et al., 1996). The increase in LFP may reflect either increase in cardiac sympathetic modulation (Pagani et al., 1986), or increase in cardiac parasympathetic modulation, only (Hedman et al., 1992; Uusitalo et al., 1996) or increase in both of them (Akselrod et al., 1985). Pomeranz et al. (1985) demonstrated a 75% reduction in LFP of RRI both after the β-adrenergic (propranolol) or after vagal (atropine) blockade. Thus, it seems that LFP of RRI is dependent on both cardiac sympathetic and vagal activity. Also Goldstein et al. (1994) have pointed out that LFP is mediated by combined sympathetic and parasympathetic action at rest. The predominance of the sympathetic activity during stressful conditions may occur, while at rest the HRV may be mainly vagally modulated (Uusitalo et al., 1996). In the present study, the measurements were performed during supine rest, which allows to conclude that the LFP could mainly be dependent on vagal activity. However, the physiological mechanism of vagal regulation of HFP and LFP of RRI could be different, because HFP did not change significantly and even tended to increase.

Our study has a few limitations. We did not measure breathing frequency, which is known to modulate the HFP. Change in breathing frequency could be seen in the HFP, when the breathing frequency corresponds to 12–15 breaths/min or more. However, Stemper et al. (2002) have indicated that the cold face test (0–1 °C, 60 s) induced bradycardia is not due to respiratory or baroreflex influences, but seems to result from central vagal activation even during changed breathing frequency after cold exposure. Further, we did not consider the effect of different phases of menstrual cycles. Sympathetic nervous activity has been suggested to be higher during the luteal than the follicular phase measured both at rest and during exercise (Sato et al., 1995). Menstrual cycle phases were not controlled, because the repeated measurements were made 4 weeks in-between and so individually at about the same time of the cycle each. As a consequence, the effect of menstrual cycle was probably minimal at the individual level. Finally, maximal heart rate area DURING WBC was not included for the analysis, because of the technical reasons considering HRV analysis. HRV during the WBC exposure showed no changes from the baseline to the 3 months time point. Uusitalo et al. (2004) speculated that the effect of physical activity on cardiac autonomic modulation could the most sensitively be seen during some intervention or activity, which is not with the present finding of the effect of cold training. However, it should be noticed, that WBC lasted only for 2 min and the analysed time was only 1 min. The recommended time for the short-term HRV recordings is minimally 5 min (Task Force, 1996).

As a conclusion, the WBC seems to have a positive effect on cardiac parasympathetic modulation mimicing the effect of physical exercise training. Although we have no information about the maximal heart rate response during the WBC, we are inclined to suggest that the WBC is safe and even beneficial for the autonomic functions of healthy people.

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