Influence of age and moderate intensity exercise training on heart rate variability in young and mature adults

Anthony S. Leicht[a,*], Graham D. Allen[a,b], Andrew J. Hoey[a]

[a]*Department of Biological and Physical Sciences, and* [b]*Centre for the Assessment of Human Performance, University of Southern Queensland, Toowoomba, QLD 4350, Australia*

*Corresponding author and address:

Dr Anthony Leicht

Institute of Sport and Exercise Science

James Cook University

Townsville QLD 4811

Australia

Phone: +61 7 4781 4576

Fax: +61 7 4781 6688

E-mail: Anthony.Leicht@jcu.edu.au

Abstract

The purpose of this study was to examine the influence of age on the changes in heart rate variability (HRV) following moderate intensity exercise training, and elucidate further the mechanism of training-induced bradycardia and cardioprotection. Electrocardiograms were recorded from 12 young (18-24 years) and 12 mature (29-43 years) human subjects during supine rest and submaximal moderate exercise. Recordings were obtained prior to, midway and following 16 weeks of aerobic exercise training designed to improve cardiorespiratory fitness and health. Training resulted in bradycardia during rest and all submaximal exercise workloads ($p<0.05$). Total HRV during rest and exercise was significantly increased for the mature subjects ($p<0.05$) following training while other measures of HRV were not significantly changed for either group. It was concluded that training of moderate intensity was insufficient to induce changes in the autonomic control of heart rate for young to mature subjects. The lack of HRV changes may suggest the existence of a vagal critical point, below which training induced increases in vagal activity may be forthcoming and above which, changes in vagal activity may be negligible. Training-induced bradycardia and the cardioprotective effect of regular aerobic exercise may result from factors other than an increased vagal activity.

Keywords: autonomic nervous system, bradycardia; spectral analysis, physical activity, aging

Introduction

It has been well established that regular aerobic exercise results in a lowering of heart rate (HR) for humans (Blomqvist and Saltin, 1983; Scheuer and Tipton, 1977; Seals and Chase, 1989) and animals (Lin and Horvath, 1972), although the mechanism for the training-induced lower HR (bradycardia) has been controversial (Scheuer and Tipton, 1977). Studies using heart rate variability (HRV), a non-invasive measure of cardiac autonomic control of HR (Pagani et al., 1986; Task Force, 1996), have provided additional information concerning the influence of regular exercise training on the neural control of HR and training-induced bradycardia. These studies have examined three major frequency components: low frequency (LF) 0.041-0.15 Hz, reflecting modulations of the sympathetic and parasympathetic nervous systems; high frequency (HF) 0.15-0.40 Hz, reflecting modulations solely of the parasympathetic nervous system; and total power (TP) 0-0.40 Hz reflecting primarily the influence of the parasympathetic nervous system (Pagani et al., 1986, Task Force, 1996).

Cross-sectional and longitudinal studies have demonstrated greater resting HRV and parasympathetic (vagal) activity post-training (Al-Ani et al., 1996; Dixon et al., 1992; Smith et al., 1989) while others have reported similar HRV and vagal activity between the trained and untrained (Bonaduce et al., 1998; Boutcher and Stein, 1995) despite training-induced bradycardia. The influence of age may account for the inconsistent reports of HRV changes, post-training. Levy et al. (1992) reported a greater training-induced increase in cardiac vagal tone in older persons than young adults while others (Gregoire et al., 1996) reported that young subjects (<30 years) failed to increase HRV following exercise training although they exhibited

similar training-induced adaptations to older subjects. Therefore, training-induced HRV increases may be more likely for mature adults, than young adults, due to their lower HRV and vagal levels (Pagani et al., 1986; Seals and Chase, 1989). More importantly, training-induced HRV increases may improve the cardiovascular health of mature adults as regular exercise training has been associated with lower cardiovascular morbidity/mortality in the general population and those with known cardiovascular risk factors (Farrell et al., 1998).

Given the relationship between regular exercise training and decreased risk of cardiovascular morbidity/mortality and possible increased vagal activity, further studies examining the influence of age on HRV and exercise training are necessary. The inconsistent reports of training-induced changes in HRV/cardiac vagal activity during rest and exercise, and the cross sectional design of most studies questions the relationship between enhanced HRV/vagal activity and training-induced bradycardia. Therefore, the current study was designed to examine the influence of age on the change in supine rest and upright exercise measures of HRV following a moderate exercise training programme designed to improve cardiorespiratory fitness and health, and to examine the training-induced changes in the autonomic control of HR (HRV) as possible contributors to the training-induced bradycardia and cardioprotection. We hypothesised that regular moderate intensity exercise training would enhance HRV/cardiac vagal activity (HF and TP) in both populations but to a greater extent for mature subjects with lower initial HRV/cardiac vagal activity. Further, we hypothesised that enhanced HRV/cardiac vagal activity (HF and TP) would contribute to the mechanism of training-induced bradycardia and cardioprotection during rest and upright exercise.

Methods

SUBJECTS

Twelve young (Y) (5 men, 7 women; 18-24 years) and 12 mature (M) (7 men, 5 women; 29-43 years) untrained, healthy adults from the local community volunteered for this study. Subjects had not exercised regularly for at least three months prior to commencement of the study and completed pre-screening questionnaires to confirm their healthy status. All subjects were non-smokers, not taking any medications or drugs, familiar with the testing equipment and procedures used in the study and provided informed consent prior to participation. The study was approved by the Human Ethics Committee of the University of Southern Queensland.

EXPERIMENTAL PROCEDURE

Electrocardiographic (ECG) recordings were obtained from subjects, prior to (pre-training), midway (mid-training) and following (post-training) a 16-week training programme of aerobic exercise. In line with guidelines for developing and maintaining cardiorespiratory fitness in healthy adults (ACSM, 1998), subjects performed any aerobic exercise of their choice, 3-4 bouts/week, ≥30 minutes/bout, at an intensity of 70% age-predicted maximum heart rate [MHR]). Examples of exercise type included cycling, running, swimming, rowing and aerobic dance and was variable to increase subject retention rate. Compliance with training was confirmed every 4 weeks by a researcher (ASL) who examined training diaries for exercise type, duration, intensity and frequency. Exercise intensity was determined by age-predicted MHR in accordance with ACSM guidelines (ACSM, 1998) and laboratory policy. More recently, the predictiveness of this MHR equation has been shown to be very good (± 6 bpm) for actual MHR in subjects of similar

age to the current study (Tanaka at al., 2001).

On each recording occasion, subjects arrived at the laboratory (20-24°C) at least 12 hours post-prandial (Widerlov et al., 1999) and 24-36 hours post-exercise (Furlan et al., 1993). All recordings were obtained at the same time of the day for each subject between 6 am and 12 noon. Females were recorded from between day 5 and 12 of the menstrual cycle to control for known hormonal influences on autonomic activity (Sato et al., 1995). Repeat recordings were conducted at a similar stage of the menstrual cycle for each female. The recording of HRV during a time of low hormonal influence for females was conducted to minimise any potential gender differences due to hormonal influences. Males and females were incorporated in the current study as several studies have reported no gender HRV differences (Arai et al., 1989; Sinnreich et al., 1998). Further, a statistical comparison between males and females per group in the current study was conducted with no significant differences identified. Subsequently, data from males and females were pooled together for each group for subsequent analysis.

Upon arrival, body mass was recorded and silver/silver chloride ECG monitoring electrodes (3M Pty Ltd, St Paul, Minnesota) were placed on the subject for the recording of modified V_1 and V_5 leads. A Marquette Holter Monitor 8500 (Marquette Electronics Inc., Milwaukee, Wisconsin) was connected to the electrodes via a 5-cable lead. The Holter ECG recordings were stored on an audiocassette tape (TDK AD60, TDK U.S.A Corporation, Port Washington, New York) for later HRV analysis. Each subject rested in the supine position for 46 minutes, the final 16 minutes being used to determine resting HRV. A 30-minute period was utilised to ensure the stabilisation of resting HR. Throughout the resting period subjects lay awake on a bench in a quiet

environment with minimal noise and body movement and were visually monitored to ensure wakefulness. Subjects breathed spontaneously during the study as breathing rate has been reported to be similar during rest and moderate exercise for the trained and untrained state (Dixon et al., 1992; Furlan et al., 1993) while controlled (metronome) breathing has been reported to be a mild stressor and alter HR (Patwardhan et al., 1995). More recently, others have stated that the control of breathing rate is unnecessary for HRV studies over the typical breathing rates (7.5-20 breaths/minute), after demonstrating minimal changes in HRV and vagal activity during spontaneous and controlled breathing (Bloomfield et al., 2001; Patwardhan et al., 2001).

Physical exercise (walking) was performed by subjects on a motor driven treadmill (Quinton Model 24-72, Quinton Instrument Company, Bothell, Washington) after the recording of resting HRV. The speed and inclination of the treadmill were altered to obtain different exercise workloads. Pre-training, subjects undertook three exercise workloads that corresponded to the lowest treadmill workload (2 kilometres per hour at 0% incline) (Ex1) and workloads that elevated HR to 50% (Ex2) and 60% (Ex3) of age-predicted MHR. During the mid- and post-training recording sessions, subjects experienced the same three absolute exercise workloads. Each workload lasted approximately 10 minutes and included the recording of exercise HRV during steady state (last 6 minutes).

HEART RATE VARIABILITY ANALYSIS

The Holter ECG recordings were analysed on a commercially available Holter analysis system (Marquette 8000 Laser Holter System, version 5.7 software, Marquette Electronics Inc., Milwaukee, Wisconsin). Each QRS complex type was automatically labelled as normal, ectopic

or artefact, but then reviewed manually to confirm correct labelling. Subsequently, the ECG recordings were analysed for HRV using a certified Marquette HRV programme (version 002A software, Marquette Electronics Inc., Milwaukee, Wisconsin). Only RR intervals between successive normal beats (normal to normal RR) were included in the calculation of HRV. Each interval excluded due to ectopy or artefact was replaced by holding the previous normal RR interval level throughout the time interval to the next valid RR interval. Recordings with greater than 1% ectopy (Kamath and Fallen, 1995) or artefact ridden were excluded from analysis.

Frequency domain measurements of HRV were determined by spectral analysis using Fast Fourier Transform (FFT). Each individual amplitude spectrum (computed over 2 minutes of data) represented a 256-point Radix 2 FFT without overlap. A Hanning window was applied to minimise spectral leakage and the time series function consisted of sampling the RR intervals every 469 ms. Each point of the amplitude spectrum was squared to obtain the power spectral density plot or power spectrum. Three frequency components were examined in each power spectrum: LF 0.041-0.15 Hz, reflecting modulations of the sympathetic and parasympathetic nervous systems; HF 0.15-0.40 Hz, reflecting modulations solely of the parasympathetic nervous system; and TP 0-0.40 Hz (Pagani et al., 1986, Task Force, 1996). Each component was determined by the area (power) of its relevant frequency band and expressed in absolute units (ms^2) and normalised units (nu). Normalised units were calculated by dividing the absolute power of a given component by the TP minus the very low frequency (VLF) component (0-0.041 Hz) (Pagani et al., 1986). The LF/HF ratio (absolute units), an index of sympathovagal balance (Pagani et al., 1986), was also determined for each power spectrum.

STATISTICAL ANALYSIS

Data were expressed as mean ± S.E.M. Statistical comparisons were conducted using the statistical package SPSS (SPSS Inc., Chicago, Illinois). Significant differences due to age and training were determined by a two way repeated measures ANOVA. Further identification of these differences was determined by Fisher's Least Significance Difference and independent t-test. Data violating the assumptions of these parametric procedures were compared using the Friedman's χ^2 test and Nemenyi's procedure (Hatch and Lazaraton, 1991).

Pre- and post-training relationships between variables were determined by Spearman's Rank-Order correlation. Correlation coefficients only greater than ± 0.5 were considered. A $p<0.05$ (2-tailed) was deemed to be statistically significant.

Results

SUBJECTS

Despite a significant mean age difference (19.9 ± 0.5 years vs 34.1 ± 1.2 years, $p<0.001$), body mass was similar for the Y and M subjects (68.3 ± 2.7 kg vs 76.6 ± 3.9 kg, NS), pre-training and remained unchanged for both groups throughout the study.

There was a significant training effect on HR during rest and all exercise workloads ($p<0.05$, power ≥0.99). Heart rates during rest and exercise were significantly lower following 8 weeks of training (mid-training) and remained significantly lower at the conclusion of the study (Figure 1,

Figure 2).

EXERCISE TRAINING AND HRV

There was no significant training effect on HRV except for LF (ms^2) and TP (ms^2) during Ex1 and TP (ms^2) during Ex2 ($p<0.05$, power 0.62, 0.58, 0.62 respectively). Post hoc analyses indicated that none of the pre-training HRV measures during rest and exercise for Y subjects were significantly altered by the training programme (Table 1). All mid- and post-training HRV measures during rest and exercise for M subjects were similar to pre-training values except for a significantly greater post-training LF(ms^2) during Ex1 and a significantly greater post-training TP (ms^2) during Ex1 and Ex2 (Table 2).

COMPARISON BETWEEN YOUNG AND MATURE SUBJECTS

There were no significant group (Y vs M) HRV differences except for HR during Ex2 and Ex3, HF (ms^2) and HF (nu) during rest, and LF/HF during Ex3 ($p<0.05$, power 0.99, 1.00, 0.58, 0.51, 0.63 respectively). Post hoc analyses indicated that pre-training, M subjects exhibited a significantly lower HF (ms^2) during rest and lower LF/HF during Ex3 (Table 2). Mid-training, HF (ms^2) and HF (nu) during rest were significantly lower for M subjects compared with Y subjects (Table 2). All other HRV measures were similar between Y and M subjects pre-, mid- and post-training.

Despite the above-mentioned significant differences between groups for HR and HRV, analyses confirmed similar HR and HRV responses to training for each group ($p>0.05$) (ie mid-pre and

post-pre).

RELATIONSHIPS BETWEEN AGE AND HRV

Prior to training, age was significantly and negatively correlated ($\rho = -0.54$, $p<0.01$) with resting HF (ms^2). This relationship was not evident post-training. No other relationships between age and HRV were evident in the current study.

Discussion

The current study demonstrated that: 1) cardiac vagal activity, measured as the HF component of HRV, was unchanged following 16 weeks of moderate intensity exercise training in Y and M subjects; 2) training-induced increases of total HRV were dependent upon age or possible initial HRV level; 3) training–induced bradycardia occurred without significant changes in cardiac autonomic control of HR (HRV); and 4) factors other than an increased vagal activity may contribute to the mechanism of training-induced bradycardia and cardioprotection.

CHRONIC EFFECTS OF EXERCISE ON HRV

During rest and most of the exercise workloads, Y and M subjects exhibited similar absolute measures of HRV (LF, HF) regardless of training status. This lack of training-induced HRV change was similar to that previously reported in longitudinal (Bonaduce et al., 1998; Boutcher and Stein, 1995) and cross-sectional studies (Gregoire et al., 1996; Macor et al., 1996). The results indicated that the neural modulation of HR and vagal activity were unchanged by the

short-term exercise training programme used in the present study. This is in contrast to reports of significantly greater vagal activity following regular exercise training in longitudinal (Al-Ani et al., 1996; Seals and Chase, 1989) and cross-sectional human studies (Dixon et al., 1992; Goldsmith et al., 1992; Smith et al., 1989). Factors such as the intensity and duration of the training programmes and the incorporation of subjects with long histories (>16 weeks) of regular exercise training in cross-sectional studies make comparisons between studies difficult. Further, the possible existence of 'responders' and 'non-responders' to exercise training may also confound comparisons between studies. Several studies have reported significantly greater HRV and vagal tone following regular training in only some of their subjects (Al-Ani et al., 1996; Coats et al., 1992); these 'responders' could not be distinguished from the 'non-responders' based upon either disease severity, drug usage or pre-training HRV. In the current study, 7 of the 12 Y subjects and 3 of the 12 M subjects did not significantly increase at least two of the resting HRV components (LF, HF and TP) as a consequence of training; such 'non-responders' could not be differentiated from the other subjects based upon age, gender, body mass or resting HR and HRV measures. Collectively, these results suggest the existence of 'responders' and 'non-responders', with the latter demonstrating an inability to increase HRV with training. Training may bring about little or no changes in subjects with high levels of vagal activity and HRV, while others with lower HRV and vagal activity may increase vagal activity to a much greater extent. Consistent reports of training-induced HRV increases for cardiac patients (Coats et al., 1992; Kiilavuori et al., 1995), greater HRV increases in more sedentary subjects (Schuit et al., 1999), the close positive relationship reported between aerobic capacity, vagal activity and HRV (Tulppo et al., 1998) further suggest the existence of a vagal level resistant to training (ie vagal critical point, VCP). The exact level of vagal activity for the proposed VCP is presently

unknown. It could be suggested that the VCP is located at a percentage of one's genetically determined maximum vagal activity rather than an absolute level of vagal activity as evident by the inconsistent identification of responders/non-responders to exercise training in the current and other studies (Al-Ani et al., 1996; Coats et al., 1992). Recently, Goldberger et al. (2001) reported a negative quadratic relationship between HRV/parasympathetic activity and HR in which HRV (ie HF) plateaued between 43 and 60 bpm (approximately 48 bpm) for subjects of similar age to that in the current study. The existence of a VCP could account for the lack of training-induced HRV (HF) change in the current study with subjects exhibiting levels of vagal activity resistant to training. Taken together, the results of Goldberger et al. (2001) and the current study suggest the existence of a VCP in which HRV at a HR between 43 and 60 bpm may not be modified with moderate intensity exercise training.

Although the existence of a VCP could account for the lack of HRV change in the current study, the stimulus provided by the exercise training programme may have been insufficient to produce significant central cardiac changes (Ehsani et al., 1982). Recently, others have suggested that intense or prolonged training was necessary for significant HRV changes (Melanson and Freedson, 2001; Schuit et al., 1999). Therefore, the moderate intensity exercise training regime of the present study may have not provided sufficient stimulus to bring about significant central cardiac adaptations and changes in HRV. Further studies examining the influences of high intensity exercise training on HRV are warranted.

Despite a lack of significant change in the LF and HF components of HRV during rest and most of the exercise workloads, the M subjects demonstrated an increase in total HRV (TP) following

16 weeks of exercise training. This confirms previous results of a greater resting total HRV in longitudinal (Seals and Chase, 1989) and cross-sectional studies (Goldsmith et al., 1992). The current data revealed that the HF and LF components accounted for approximately 40% of the increase in TP with the remaining 60% of the increase possibly attributed to a significant increase in the VLF component. The VLF component was not examined in the present study, as the physiological meaning of this component is poorly understood. However, several studies have documented the influence of the renin-angiotensin system (RAS) and vagal activity in the modulation of very low HR oscillations (Akselrod et al., 1981; Taylor et al., 1998). These and other studies examining cardiac angiotensin receptors (Saito et al., 1987) suggest that RAS activity may produce a direct (sino-atrial node) and indirect (vagus nerve) inhibitory effect on the heart, thereby reducing HRV. A negative correlation between plasma angiotensinogen and HRV (Busjahn et al., 1998) provides additional support for an inhibitory effect of the RAS on vagal activity and HRV. In the current study, plasma levels of Angiotensin I and II and RAS activity via ACE inhibition were not determined. Therefore, the hypothesised increase in the VLF component for M subjects following exercise training cannot be attributed to altered RAS activity at this stage. Further studies are needed to investigate fully the influence of Angiotensin and the RAS on the VLF band and its possible association with exercise training.

INFLUENCE OF AGE AND EXERCISE TRAINING ON HRV

Pre-training, the M subjects demonstrated lower resting HRV and vagal activity than the Y subjects in agreement with the well-published inverse relationship between HRV and age (De Meersman, 1993; Pagani et al., 1986). However, like previous studies (Levy et al., 1992), no significant relationship between age and post-training HRV was evident in the current study, as

HRV was similar between groups. As previously discussed it may be inferred that the M subjects, with lower HRV, were able to increase their vagal modulation of HR to a greater extent than the Y subjects with higher levels of vagal activity (ie VCP). Additionally, moderate intensity exercise training may increase the responsiveness of HRV to vagal activity in older subjects similar to that of younger subject (Goldberger et al., 2001). Therefore, exercise training may be more beneficial for older adults than younger adults (<30 years) for increasing vagal activity and reducing the likelihood of cardiovascular disease.

MECHANISM OF TRAINING-INDUCED BRADYCARDIA AND CARDIOPROTECTION

The mechanism by which regular exercise training induces bradycardia has been a highly debated topic for many years (Blomqvist and Saltin, 1983; Scheuer and Tipton, 1977) with several mechanisms proposed such as an enhanced cardiac vagal activity (Al-Ani et al., 1996; Dixon et al., 1992), decreased sympathe ic activity (Lin and Horvath, 1972), lower intrinsic rate (Katona et al., 1982; Smith et al., 1989) or a combination of these (Smith et al., 1989). In the current study, HR during rest and exercise was significantly reduced for subjects following training without significant changes in HRV and vagal activity. The reduction in HR was similar to that reported with training-induced increases of VO_{2max} (Seals and Chase, 1989) and attests to the effectiveness of and subject compliance with the training programme. The results of the current study indicated that an enhanced vagal activity, as measured by HRV, was not essential for training-induced bradycardia. Other possible mechanisms, such as a lower intrinsic HR (Katona et al., 1982; Smith et al., 1989) therefore may contribute to training-induced bradycardia. The exact mechanism by which training may reduce the intrinsic HR is unknown; however, it may be related to changes in ionic concentrations (Raab, 1969) or to the mechanical stretching of the

right atrium and sino-atrial node (Blomqvist and Saltin, 1983; Katona et al., 1982). Therefore, a combination of various factors may contribute to the training-induced bradycardia.

In the current study, vagal activity (HRV) was not increased in any of the subject groups following exercise training and suggested that enhanced vagal activity may not contribute to training-induced cardioprotection. As previously discussed the lack of vagal change following exercise training may have resulted from the initial high level of vagal activity of subjects or an insufficient training stimulus. Additionally, exercise training and its associated cardioprotective effects may only become evident when vagal activity falls below the proposed VCP. The reported decrease of vagal activity with age (De Meersman, 1993; Pagani et al., 1986), the greater incidence of cardiovascular events in older subjects (Schuit et al., 1999) and the beneficial effects of regular exercise training in retarding age-associated declines in autonomic control of HR (De Meersman, 1993; Levy et al., 1992) support this notion. The results of the current study do not refute the association between vagal activity and cardioprotection following exercise training; however, they may suggest that in young to mature healthy adults with high levels of vagal activity (ie levels above the proposed VCP), cardioprotection and vagal activity may be resistant to regular exercise training.

Although several studies have inferred an association between vagal activity and training–induced cardioprotection, the cardioprotective effect of regular exercise may also be related to the change in intrinsic HR following training. To our knowledge, no study has examined the intrinsic HR change following regular exercise training and its possible association with cardioprotection. This could be largely due to the contradictory reports of a lower intrinsic HR

following exercise training (Shi et al., 1995; Smith et al., 1989) and the invasive procedures needed to record intrinsic HR. Whatever the reason, the possible lower intrinsic HR following regular exercise training may contribute to the cardioprotective effects of regular exercise, possibly by inducing bradycardia.

In conclusion, the current study demonstrated that for young and mature adults, short-term regular aerobic exercise training reduced HR during supine rest and upright exercise with no significant changes in the LF and HF components of HRV. Mature aged subjects exhibited a significantly greater total HRV following 16 weeks of moderate intensity exercise training possibly via increased vagal and/or RAS activities at very low frequencies (<0.04 Hz). Factors other than an increased cardiac vagal activity (ie non-autonomic) may contribute to the mechanism of training-induced bradycardia and cardioprotection.

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Acknowledgements

The authors would like to thank Henry Eastment for his statistical assistance. We are also indebted to the Cardiac Investigations Unit at Prince Charles Hospital, Brisbane, Australia and Marquette Electronics Incorporated for their assistance with and access to the Marquette Holter Analysis System used in the current study.

Figure captions

Figure 1. Average heart rate for young subjects during rest and submaximal exercise pre-, mid- and post-training. * $p<0.05$, ** $p<0.01$, *** $p<0.001$ different from pre-training.

Figure 2. Average heart rate for mature subjects during rest and submaximal exercise pre-, mid- and post-training. * $p<0.05$, ** $p<0.01$, *** $p<0.001$ different from pre-training, †† $p<0.01$, ††† $p<0.001$ different from young subjects.

Table 1 Heart rate and heart rate variability measures for young (Y) subjects during rest and the exercise workloads (Ex1, Ex2, Ex3), pre-, mid- and post-training.

		Rest	Ex1	Ex2	Ex3[#]
LF *(ms^2)*	**Pre-T**	3585 ± 1117	1150 ± 211	374 ± 51	52 ± 10
	Mid-T	3595 ± 777	1526 ± 268	496 ± 92	83 ± 14
	Post-T	3554 ± 969	1548 ± 446	555 ± 180	77 ± 19
LF *(nu)*	**Pre-T**	43.65 ± 4.56	77.63 ± 3.74	82.23 ± 2.01	77.58 ± 2.69
	Mid-T	43.55 ± 4.66	77.91 ± 2.71	78.50 ± 2.66	77.07 ± 2.40
	Post-T	45.93 ± 4.74	77.62 ± 3.70	80.00 ± 2.60	78.87 ± 4.22
HF *(ms^2)*	**Pre-T**	3940 ± 704	471 ± 171	77 ± 14	12 ± 2
	Mid-T	4009 ± 843	489 ± 125	126 ± 32	24 ± 5
	Post-T	3405 ± 798	701 ± 311	128 ± 35	14 ± 2
HF *(nu)*	**Pre-T**	58.88 ± 4.44	23.93 ± 3.94	19.23 ± 2.13	23.24 ± 2.66
	Mid-T	59.59 ± 4.09	23.86 ± 3.00	22.60 ± 2.72	23.96 ± 2.51
	Post-T	55.84 ± 4.72	24.16 ± 3.99	21.14 ± 2.71	21.88 ± 4.24
TP *(ms^2)*	**Pre-T**	10470 ± 2349	2737 ± 529	1062 ± 105	489 ± 26
	Mid-T	11102 ± 1716	3334 ± 533	1516 ± 205	622 ± 40
	Post-T	10589 ± 2223	3444 ± 868	1404 ± 226	556 ± 37
LF/HF	**Pre-T**	1.06 ± 0.20	5.36 ± 0.95	6.04 ± 0.94	4.72 ± 0.74
	Mid-T	1.08 ± 0.20	4.97 ± 0.85	5.10 ± 0.92	4.39 ± 0.82
	Post-T	1.22 ± 0.32	5.06 ± 0.85	5.37 ± 0.75	5.97 ± 1.17

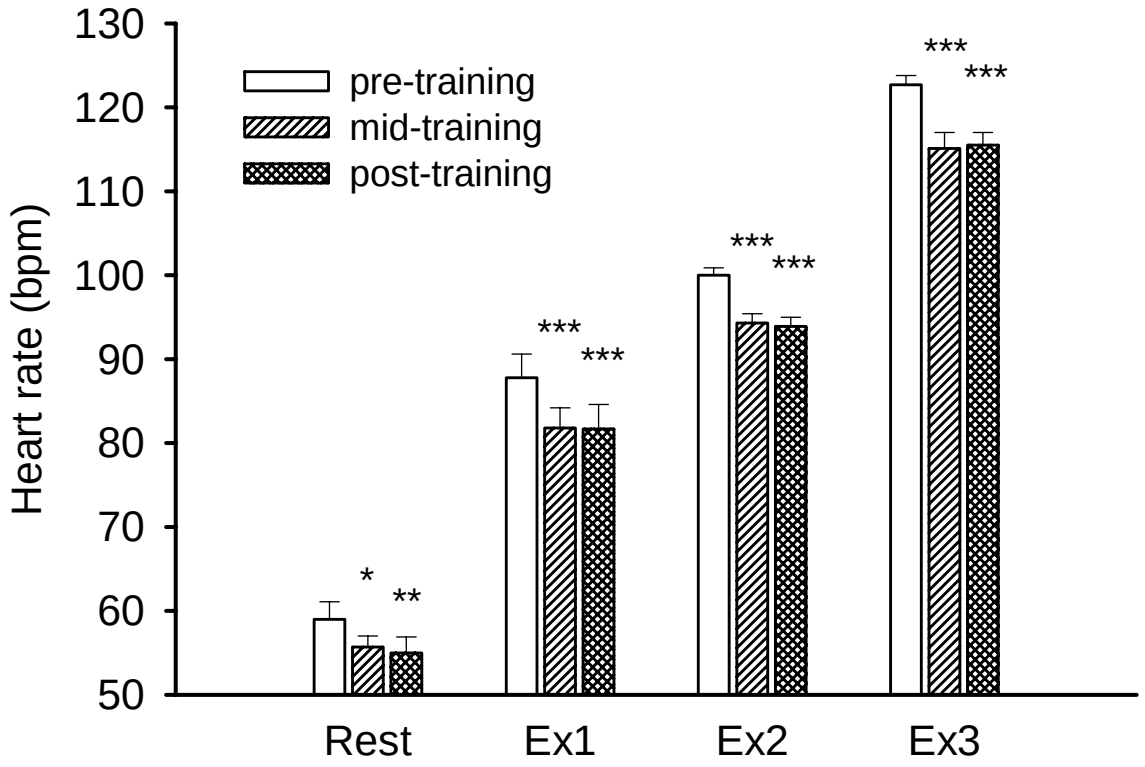
Values are mean ± SEM (n=12; [#] n=11); Pre-T – pre-training; Mid-T – mid-training; Post-T – post-training; LF – low frequency; HF – high frequency; TP – total power; nu – normalised units

Table 2 Heart rate and heart rate variability measures for mature (M) subjects during rest and the exercise workloads (Ex1, Ex2, Ex3), pre-, mid- and post-training.

		Rest	Ex1	Ex2	Ex3[#]
LF *(ms^2)*	**Pre-T**	1500 ± 327	1074 ± 249	408 ± 85	125 ± 36
	Mid-T	2120 ± 419	1412 ± 307	470 ± 114	160 ± 43
	Post-T	2182 ± 522	1629 ± 337[*]	603 ± 182	307 ± 203
LF *(nu)*	**Pre-T**	54.55 ± 4.21	75.07 ± 3.81	74.47 ± 3.38	68.24 ± 3.60
	Mid-T	55.28 ± 4.59	76.27 ± 4.41	73.99 ± 4.62	72.83 ± 3.02
	Post-T	54.93 ± 3.80	72.71 ± 5.90	70.49 ± 4.98	70.47 ± 4.50
HF *(ms^2)*	**Pre-T**	1518 ± 702[†]	358 ± 84	181 ± 69	54 ± 18
	Mid-T	1624 ± 416[†]	461 ± 108	194 ± 67	68 ± 24
	Post-T	1827 ± 630	646 ± 168	323 ± 120	200 ± 159
HF *(nu)*	**Pre-T**	47.55 ± 4.10	26.63 ± 3.99	27.68 ± 3.68	32.90 ± 3.62
	Mid-T	46.63 ± 4.54[†]	25.28 ± 4.46	27.28 ± 4.69	28.23 ± 3.12
	Post-T	47.51 ± 3.82	28.89 ± 6.07	30.83 ± 5.04	30.67 ± 4.57
TP *(ms^2)*	**Pre-T**	5133 ± 1179	2686 ± 472	1273 ± 197	711 ± 60
	Mid-T	7255 ± 1073	3215 ± 480	1526 ± 204	798 ± 118
	Post-T	7635 ± 1632	3947 ± 642[*]	1795 ± 326[*]	1071 ± 418
LF/HF	**Pre-T**	1.55 ± 0.23	4.34 ± 0.80	3.94 ± 0.63	2.79 ± 0.51[†]
	Mid-T	1.71 ± 0.32	4.93 ± 1.16	4.23 ± 0.74	3.26 ± 0.39
	Post-T	1.79 ± 0.33	5.07 ± 1.20	4.05 ± 0.89	3.26 ± 0.46

Values are mean ± SEM (n=12; [#] n=10); Pre-T – pre-training; Mid-T – mid-training; Post-T – post-training; LF – low frequency; HF – high frequency; TP – total power; nu – normalised units

[*] *$p<0.05$ different from Pre-T;* [†] *$p<0.05$ different from young (Y) subjects*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Heart rate (bpm)
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mid-training
post-training
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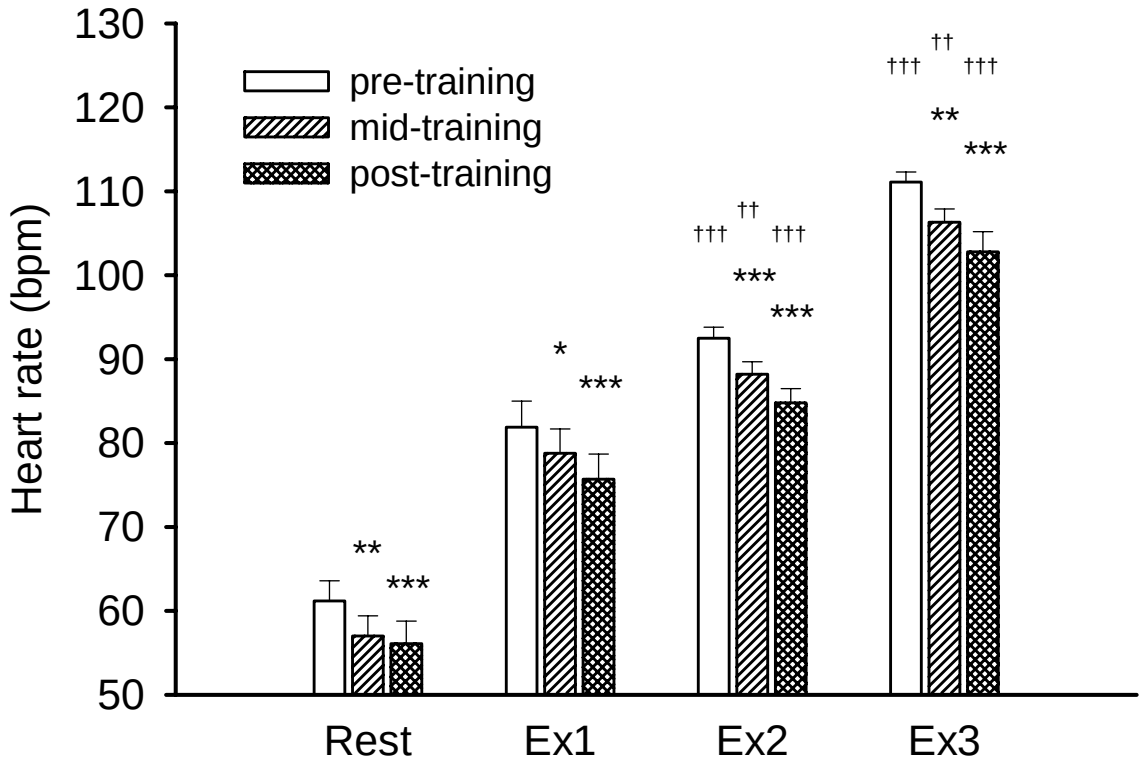
Rest
Ex1
Ex2
Ex3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Rest
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Ex2
Ex3