Influence of Age and Moderate-Intensity Exercise Training on Heart Rate Variability in Young and Mature Adults

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Abstract/Résumé

The purpose of this study was to examine the influence of age and moderate-intensity exercise training on heart rate variability (HRV), and to elucidate further the mechanism of training-induced bradycardia and cardioprotection. Electrocardiograms were recorded from 12 young (18–24 yrs) and 12 mature (29–43 yrs) individuals 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 augmented estimated $\dot{V}O_2$max and bradycardia during rest and submaximal exercise. Total and low frequency components of HRV during exercise were significantly increased for the mature subjects following training whereas 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 significant HRV changes may suggest the existence of a vagal critical point, below which training-induced increases in vagal modulation may be forthcoming, and above which changes in vagal modulation may be negligible.

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Training-induced bradycardia and the cardioprotective effect of regular aerobic exercise may result from factors other than an increased vagal modulation.

Le but de cette étude est d'analyser l'effet de l'âge et de l'entraînement physique modéré sur la variabilité de la fréquence cardiaque (HRV), et de comprendre plus à fond le mécanisme de réduction de la fréquence cardiaque (bradycardie) et de la cardioprotection à la suite d'un entraînement. Les électrocardiogrammes de 12 jeunes adultes (18 à 24 ans) et de 12 individus d'âge mûr (29 à 43 ans) sont enregistrés au cours d'un repos couché et d'un exercice sous-maximal modéré. Les observations sont faites avant, à mi-chemin, et après 16 semaines d'entraînement aérobie conçu pour améliorer la performance cardiorespiratoire et la santé. L'entraînement donne l'effet escompté: une amélioration du $\dot{V}O_2max$ prédit et une bradycardie au repos et à l'effort sous-maximal. Les composantes totales et à basse fréquence de l'HRV pendant l'exercice sont plus importantes dans le groupe d'âge mûr, mais toutes les autres mesures de l'HRV ne sont pas modifiées par l'entraînement, peu importe le groupe. En conclusion, un entraînement modéré ne suffit pas à modifier le contrôle autonome de la fréquence cardiaque chez des adultes de 18 à 43 ans. L'absence de modification substantielle de l'HRV laisse sous-entendre la présence d'un point critique, au-dessous duquel l'augmentation de la modulation vagale par l'entraînement serait imminente, et au-dessus duquel l'effet serait négligeable. Il appert que la bradycardie et la cardioprotection consécutives à la pratique régulière d'activités aérobies dépendent probablement de facteurs autres que l'augmentation de la modulation vagale.

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 a subject of controversy (Scheuer and Tipton, 1977). Studies using heart rate variability (HRV), a noninvasive measure of cardiac autonomic control of HR (Pagani et al., 1986; Task Force, 1996), have provided additional information as to 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) modulation posttraining (Al-Ani et al., 1996; Dixon et al., 1992; Smith et al., 1989), while others have reported similar HRV and vagal modulation between trained and untrained subjects (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 at posttraining. Levy et al. (1992) reported a greater training-induced increase in cardiac vagal modulation in older persons than in young adults, while Gregoire et al. (1996) reported that young subjects (<30 yrs) failed to increase HRV following exercise training although they exhibited training-induced adaptations similar to those of older sub-

jects. Therefore, training-induced increases in HRV may be more likely for mature vs. young adults, due to their lower HRV and vagal modulation (Pagani et al., 1986; Seals and Chase, 1989). More important, training-induced increases in HRV may improve the cardiovascular health of mature adults, as regular exercise training has been associated with lower cardiovascular morbidity/mortality in the general population as well as in persons 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 modulation, further studies are needed which examine the influence of age on HRV and exercise training. The inconsistent reports of training-induced changes in HRV/cardiac vagal modulation during rest and exercise, and the cross-sectional design of most studies, raises the question of the relationship between enhanced HRV/vagal modulation and training-induced bradycardia.

Therefore the current study was designed to examine the influence of age and a moderate exercise training programme, designed to improve cardiorespiratory fitness and health, on supine rest and upright exercise measures of HRV, and to examine the training-induced changes in the autonomic control of HR (HRV) as possible contributors to the training-induced bradycardia and cardioprotection. It was hypothesised that regular moderate-intensity exercise training would enhance HRV/cardiac vagal modulation (HF and TP) in both populations, but to a greater extent for mature subjects with lower initial HRV/cardiac vagal modulation. Further, it was hypothesised that enhanced HRV/cardiac vagal modulation (HF and TP) would contribute to the mechanism of training-induced bradycardia and cardioprotection during rest and upright exercise.

Methods

SUBJECTS

Twelve young (5 M, 7 F; ages 18–24 yrs) and 12 mature (7 M, 5 F; ages 29–43 yrs) untrained healthy adults from the local community volunteered for this study. They had not exercised regularly for at least 3 months prior to the study; they completed prescreening questionnaires to confirm their healthy status. All subjects were nonsmokers, were not taking any medications or drugs, were 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.

Based on minimum sample size calculations (Thomas and Nelson, 1996), using data from previous studies of trained and untrained subjects similar in age to those in the current study (Bonaduce et al., 1998; Furlan et al., 1993; Goldsmith et al., 1992), we recruited 12 subjects for the current study to ensure similar effect size (≥ 0.7) and statistical power (>0.8) to these previous studies.

EXPERIMENTAL PROCEDURE

Electrocardiographic (ECG) recordings were obtained from the subjects prior to (pretraining), midway through (midtraining), and following (posttraining) a 16-week training programme of aerobic exercise. In line with guidelines for develop-

ing and maintaining cardiorespiratory fitness in healthy adults (American College of Sports Medicine [ACSM], 1998), subjects performed any aerobic exercise of their choice, three to four bouts per week, ≥30 min/bout, at an intensity of 70% age-predicted maximum heart rate (MHR).

All exercise was unsupervised by researchers and, to increase subject retention rate, included cycling, running, swimming, rowing, aerobic dance, or similar aerobic activities. A researcher interviewed the subjects every 4 weeks to confirm compliance with training and to examine training diaries for exercise type, duration, intensity, and frequency. Adequate compliance was determined as the completion of aerobic exercise on an average of at least 3 bouts per week and at least 30 min per bout, at an average intensity of ≥70% MHR. Exercise intensity was determined by age-predicted MHR in accordance with ACSM guidelines (1998) and recorded by subjects during each exercise bout (pulse or HR monitor). Recently the predictability of this MHR equation has been shown to be very good (±6 bpm) for actual MHR in subjects similar in age to those in the current study (Tanaka et al., 2001).

On each recording occasion, subjects arrived at the laboratory (20–24 °C) at least 12 hrs postprandial (Widerlov et al., 1999) and 24–36 hrs postexercise (Furlan et al., 1993). All recordings were obtained at the same time of day for each subject between 6 a.m. and noon. Women were recorded from between Day 5 and Day 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. The recording of HRV for women was conducted during a time of low hormonal influence (i.e., follicular phase) to minimise any potential gender differences due to hormonal influences. Both genders were incorporated in the current study, as several studies have reported minimal HRV differences between men and premenopausal women of similar age (Arai et al., 1989; Evans et al., 2001; Stein et al., 1997). Further, we conducted a statistical comparison between genders per group and found no significant differences. Consequently, data from men and women per group were pooled together for subsequent analysis.

When subjects arrived at the lab, body mass was recorded and silver/silver chloride ECG monitoring electrodes (3M Pty Ltd, St. Paul, MN) were placed on them for the recording of modified V_1 and V_5 leads. A Marquette holter monitor 8500 (Marquette Electronics, Milwaukee, WI) was connected to the electrodes via a 5-cable lead. The holter ECG recordings were stored on an audiotape (TDK AD60, TDK USA Corp., Port Washington, NY) for later HRV analysis. Each subject rested supine for 46 minutes, the final 16 min being used to determine resting HRV. A 30-min period was utilised to ensure the stabilisation of resting HR. Throughout the resting period the subjects lay awake on a bench in a quiet environment with minimal noise and body movement, and they 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). Controlled (metronome) breathing, on the other hand, has been reported to be a mild stressor which can 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/min), after demonstrating minimal changes in HRV and va-

gal modulation during spontaneous and controlled breathing (Bloomfield et al., 2001; Patwardhan et al., 2001).

Subjects performed physical exercise (walking) on a motor-driven treadmill (Quinton model 24-72, Quinton Instrument Co., Bothell, WA) after the recording of resting HRV. The speed and inclination of the treadmill were altered to obtain different exercise work rates. At pretraining, subjects undertook three exercise work rates that corresponded to the lowest treadmill work rate (2 km per hour at 0% incline) (Ex1), and work rates that elevated HR to 50% (Ex2) and 60% (Ex3) of age-predicted MHR. During the mid- and posttraining recording sessions, subjects experienced the same three absolute exercise work rates. Each work rate lasted approximately 10 min and included the recording of exercise HRV during steady state (last 6 min).

To confirm the effectiveness of the training programme, we calculated estimated $\dot{V}O_2$max from the treadmill exercise work rates and corresponding HR using the ACSM walking equations (ACSM, 2000) and graph extrapolation (HR vs. estimated $\dot{V}O_2$) to age-predicted MHR.

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). Each QRS complex type was automatically labelled as normal, ectopic, or artefact, but then was 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). 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 more than 1% ectopy (Kamath and Fallen, 1995) or those that were artefact ridden were excluded from analysis.

Frequency domain measurements of HRV were determined by spectral analysis using fast fourier transform (FFT). Each amplitude spectrum (computed over 2 min 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, index of sympathetic and parasympathetic modulations; HF 0.15–0.40 Hz, index of parasympathetic modulation; and TP 0–0.40 Hz, reflecting primarily parasympathetic modulation (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/Hz) and normalised units (n.u.).

Normalised units were calculated as the absolute power of a given component divided by TP minus the very low frequency (VLF) component (0–0.041 Hz) (Pagani et al., 1986). The LF/HF ratio (absolute units) was also determined for each power spectrum and, along with the HRV n.u. measures, was utilized as an index of sympathovagal balance (Pagani et al., 1986) (i.e., enhanced sympathetic

modulation = greater LF/HF, greater LF n.u., reduced HF n.u.; enhanced parasympathetic modulation = reduced LF/HF, reduced LF n.u., increased HF n.u.).

STATISTICAL ANALYSIS

Data were expressed as mean ± *SEM*. Statistical comparisons were conducted using the statistical package SPSS (SPSS Inc., Chicago). Significant differences for each experimental condition and variable (e.g., resting HR, Ex1, LF n.u.) were determined by a two-way ANOVA (group vs. time) with repeated measures for time (i.e., pre- vs. mid- vs. posttraining). Further identification of these differences was determined by Fisher's least significance difference (LSD) test 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 posttraining relationships between variables were determined by Spearman's rank-order correlation. Only correlation coefficients 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 yrs vs. 34.1 ± 1.2 yrs, $p < 0.001$), body mass was similar for the young (Y) and mature (M) subjects (68.3 ± 2.7 kg vs. 76.6 ± 3.9 kg, n.s.) at pretraining and remained unchanged for both groups throughout the study.

Heart rates during rest and all exercise work rates were significantly reduced by training (time main effect, $p < 0.001$). Post hoc analyses indicated that HR during each experimental condition was significantly lower at midtraining and remained significantly lower at the end of the study for Y (Figure 1) and M subjects (Figure 2). Estimated $\dot{V}O_2$max was significantly increased following training (time main effect, $p < 0.001$) and of similar magnitude for Y and M subjects (9.3 ± 3.4 vs. 16.2 ± 8.2%, $p > 0.05$).

EXERCISE TRAINING AND HRV

No measure of HRV was significantly changed by training except for Ex1 LF (ms^2/Hz), Ex1 TP (ms^2/Hz), and Ex2 TP (ms^2/Hz) (time main effect, $p < 0.05$; Tables 1 and 2). Post hoc analyses indicated that posttraining Ex1 LF (ms^2/Hz), Ex1 TP (ms^2/Hz), and Ex2 TP (ms^2/Hz) were significantly greater compared to pretraining values for M subjects only (Table 2).

COMPARISON BETWEEN YOUNG AND MATURE SUBJECTS

Heart rates during rest and exercise did not differ significantly between Y and M subjects except during Ex2 and Ex3 (group main effect, $p < 0.001$). Mature subjects exhibited a significantly lower HR during Ex2 and Ex3 compared to Y subjects at pre-, mid-, and posttraining (Figure 2).

No measure of HRV was significantly different between groups except for HF (ms^2/Hz) and HF (n.u.) during rest, and LF/HF during Ex3 (group main effect,

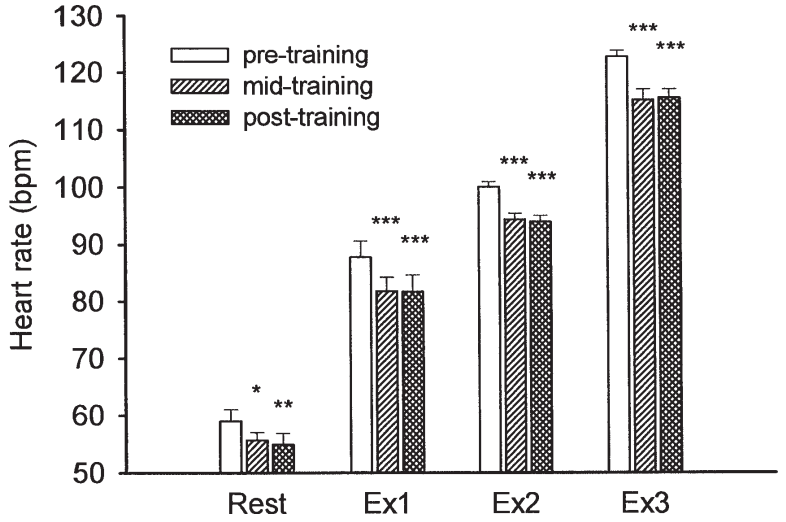

Figure 1. Average (± *SEM*) heart rate for young subjects during rest and submaximal exercise at pre-, mid-, and posttraining. Significantly different from pretraining: *p < 0.05; **p < 0.01; ***p < 0.001.

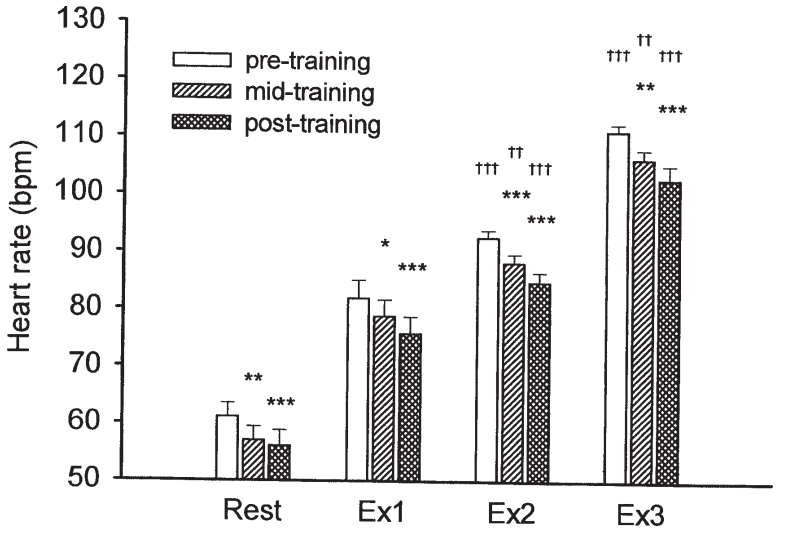

Figure 2. Average (± *SEM*) heart rate for mature subjects during rest and submaximal exercise at pre-, mid, and posttraining. Significantly different from pretraining: *p < 0.05; **p < 0.01; ***p < 0.001. Significantly different from young subjects: $^{\dagger\dagger}p$ < 0.01; $^{\dagger\dagger\dagger}p$ < 0.001.

Table 1 Heart Rate Variability Measures for Young (Y) Subjects During Rest and the Exercise Work Rates (Ex1, Ex2, Ex3) at Pre-, Mid- and Posttraining

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

Note: Values are mean ± *SEM* (n = 12; [#]n = 11); LF = low frequency; HF = high frequency; TP = total power; n.u. – normalised units.

Table 2 Heart Rate Variability Measures for Mature (M) Subjects During Rest and the Exercise Work Rates (Ex1, Ex2, Ex3) at Pre-, Mid- and Posttraining

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

Note: Values are mean ± *SEM* (n = 12; [#]n = 10); LF = low frequency; HF = high frequency; TP = total power; n.u. – normalised units. *Different from pretraining, $p < 0.05$; [†]Different from young (Y) subjects, $p < 0.05$.

$p < 0.05$). Post hoc analyses indicated that at pretraining, M subjects exhibited a significantly lower HF (ms^2/Hz) during rest and lower Ex3 LF/HF (Table 2). At midtraining, HF (ms^2/Hz) and HF (n.u.) during rest were significantly lower for M subjects compared with Y subjects (Table 2). All other HRV measures were similar between both groups at pre-, mid-, and posttraining.

Despite the above-mentioned significant differences between groups for HR and HRV, analyses confirmed similar HR and HRV responses to training for each group, $p > 0.05$ (i.e., 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/Hz). This relationship was not evident at posttraining. No other relationships between age and HRV were evident in the current study.

Discussion

The current study demonstrated the following: (1) cardiac vagal modulation, 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). (4) Factors other than an increased vagal modulation may contribute to the mechanism of training-induced bradycardia and cardioprotection.

CHRONIC EFFECTS OF EXERCISE ON HRV

During rest and most of the exercise work rates, both groups 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 control of HR and vagal modulation were unchanged by the exercise-training programme used in the present study. The lack of training-induced HRV change does not seem to be a result of HRV measurement instability, as previous studies have documented stable and reproducible HRV under controlled conditions of rest and exercise (Amara and Wolfe, 1998) and rest over several months (Sinnreich et al., 1998).

The present results of similar HRV following exercise training are in contrast to reports of significantly greater vagal modulation 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 a long history (>16 weeks) of regular exercise training in cross-sectional studies make comparisons between studies difficult. Furthermore, 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 modulation 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 on either disease severity, drug usage, or pretraining 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 on age, gender, body mass, or resting HR and HRV measures.

Collectively, these results suggest there are responders and non-responders, with the latter demonstrating an inability to increase HRV with training. Training may bring about little if any changes in subjects with high levels of vagal modulation and HRV, while others with lower HRV and vagal modulation may increase the latter 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), and the close positive relationship reported between aerobic capacity, vagal modulation, and HRV (Tulppo et al., 1998) further suggest the existence of a vagal modulation level that is resistant to training (i.e., vagal critical point, VCP). The exact level of vagal modulation for the proposed VCP is unknown. It could be suggested that the VCP is located at a percentage of one's genetically determined maximum vagal modulation, rather than an absolute level of vagal modulation, as evidenced by the inconsistent identification of responders/nonresponders to exercise training in the current study 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 modulation and HR in which HRV (i.e., HF) plateaued between 43 and 60 bpm (approx. 48 bpm) for subjects similar in age to those 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 modulation 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 exercise training of moderate intensity.

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 needed to produce significant HRV changes (Melanson and Freedson, 2001; Schuit et al., 1999). Therefore, the moderate-intensity exercise training regimen of the present study may have not provided sufficient stimulus to elicit significant central cardiac adaptations and changes in HRV. Further studies are warranted which examine the influences of high-intensity exercise training on HRV.

Despite a lack of significant change in the LF and HF components of HRV during rest and most of the exercise work rates, the M subjects demonstrated an increased total HRV (TP) following 16 weeks of exercise training. This confirms previous results of a greater 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 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 parasympathetic nervous system 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 modulation 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 fully investigate 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

At pretraining, the M subjects demonstrated lower resting HRV and vagal modulation 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 posttraining HRV was evident in the current study, as HRV was similar between groups. As previously discussed, we may infer that the M subjects, with lower HRV, were able to increase their vagal modulation of HR to a greater extent than the Y subjects, who had higher levels of vagal modulation (i.e., VCP). Additionally, moderate-intensity exercise training may increase the responsiveness of HRV to vagal modulation in older subjects similar to that of younger subjects (Goldberger et al., 2001). Therefore, exercise training may be more beneficial for older adults than younger adults (<30 yrs) for increasing vagal modulation 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 highly debated for many years (Blomqvist and Saltin, 1983; Scheuer and Tipton, 1977). Several mechanisms have been proposed such as an enhanced cardiac vagal modulation (Al-Ani et al., 1996; Dixon et al., 1992), decreased sympathetic 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 modulation. These HR reductions and increased estimated $\dot{V}O_2$max for Y and M subjects were similar to those reported previously (Melanson and Freedson, 2001; Seals and Chase, 1989) and attest to the effectiveness of and subject compliance with the training programme.

The results of the current study indicated that an enhanced vagal modulation, 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 might 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 could contribute to the training-induced bradycardia.

In the current study, vagal modulation (HF) was not increased in either group following exercise training, which suggests that enhanced vagal modulation may not contribute to training-induced cardioprotection. As previously discussed, the lack of vagal change following exercise training may have resulted from the subjects' initial high level of vagal modulation or an insufficient training stimulus. Additionally, exercise training and its associated cardioprotective effects may only become evident when vagal modulation falls below the proposed VCP. The reported decrease of vagal modulation 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 modulation and cardioprotection following exercise training. However, they may suggest that in young to mature healthy adults with high levels of vagal modulation (i.e., levels above the proposed VCP), cardioprotection and vagal modulation may be resistant to regular exercise training.

Although several studies have inferred an association between vagal modulation 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 due largely 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, regular aerobic exercise training reduced HR during supine rest and upright exercise with minimal changes in the LF and HF components of HRV. Mature subjects exhibited greater total HRV following 16 weeks of moderate-intensity exercise training, possibly via increased vagal and/or RAS modulations at very low frequencies (<0.04 Hz). Training stimulus and the existence of a vagal critical point (i.e., vagal modulation level resistant to training) were proposed as possible influences to training-induced HRV changes. Factors other than an increased cardiac vagal modulation (i.e., nonautonomic) may contribute to the mechanism of training-induced bradycardia and cardioprotection.

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