

Effects of aerobic exercise training and yoga on the baroreflex in healthy elderly persons

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Abstract. It is unclear whether the age-associated reduction in baroreflex sensitivity is modifiable by exercise training. The effects of aerobic exercise training and yoga, a non-aerobic control intervention, on the baroreflex of elderly persons was determined. Baroreflex sensitivity was quantified by the α -index, at high frequency (HF; 0.15–0.35 Hz, reflecting parasympathetic activity) and mid-frequency (MF; 0.05–0.15 Hz, reflecting sympathetic activity as well), derived from spectral and cross-spectral analysis of spontaneous fluctuations in heart rate and blood pressure. Twenty-six (10 women) sedentary, healthy, normotensive elderly (mean 68 years, range 62–81 years) subjects were studied. Fourteen (4 women) of the sedentary elderly subjects completed 6 weeks of aerobic training, while the other 12 (6 women) subjects completed 6 weeks of yoga. Heart rate decreased following yoga (69 ± 8 vs. 61 ± 7 min⁻¹, $P < 0.05$) but not aerobic training (66 ± 8 vs. 63 ± 9 min⁻¹, $P = 0.29$). VO_2 max increased by 11% following yoga ($P < 0.01$) and by 24% following aerobic training ($P < 0.01$). No significant change in α_{MF} (6.5 ± 3.5 vs. 6.2 ± 3.0 ms mmHg⁻¹, $P = 0.69$) or α_{HF} (8.5 ± 4.7 vs. 8.9 ± 3.5 ms mmHg⁻¹, $P = 0.65$) occurred after aerobic training. Following yoga, α_{HF} (8.0 ± 3.6 vs. 11.5 ± 5.2 ms mmHg⁻¹, $P < 0.01$) but not α_{MF} (6.5 ± 3.0 vs. 7.6 ± 2.8 ms mmHg⁻¹, $P = 0.29$) increased. Short-duration aerobic training does not modify the α -index at α_{MF} or α_{HF} in healthy normotensive elderly subjects. α_{HF} but not α_{MF} increased following yoga, suggesting that these parameters are measuring distinct aspects of the baroreflex that are separately modifiable.

Keywords. Ageing, baroreflex, exercise, yoga.

Introduction

The arterial baroreflex is a rapidly acting system that modulates blood pressure. Arterial pressure changes are detected by baroreceptors comprising nerve endings in the wall of the carotid sinuses and aortic arch. This information is integrated in the central nervous system,

which then sends efferent signals via parasympathetic and sympathetic pathways to control heart rate changes on a second-to-second basis [1]. In normal subjects, increased stretching of the baroreceptors initiates increased afferent nerve outflow to the solitary tract nucleus. This results in activation of the vagal nucleus and inhibition of neurones of the vasomotor centre, increasing vagal tone to the heart and decreasing sympathetic outflow to the heart and blood vessels. The opposite happens if pressure falls in the vessels housing the baroreceptors. Baroreceptors in hypertensive subjects appear to be 'reset' so that higher systemic blood pressures are tolerated, without causing a reduction in sympathetic activity, which would reduce peripheral resistance, cardiac contractility and so return blood pressure to normotensive levels. It has been suggested that the mechanism responsible for the age-associated changes in baroreflex sensitivity are similar to those occurring in younger, hypertensive individuals [1,2].

Baroreflex sensitivity decreases progressively with ageing in man [2–4] and is reduced in hypertension [3,5]. Studies have shown that hypertensive subjects undertaking aerobic exercise training can partially reverse this decline [6,7]. Several studies of the effect of aerobic exercise on BRS in young, normotensive subjects have produced conflicting results with decreased [7–9], no change [10,11] or increased [12] baroreflex sensitivity all being reported. A study of 10 middle-aged individuals found no change in baroreflex sensitivity after 12 weeks of aerobic exercise training [13], but few other data exist to indicate whether the age-associated decline in baroreflex sensitivity can be altered by exercise training in healthy, sedentary, elderly, normotensive individuals.

Heart rate response to a phenylephrine-induced rise in blood pressure has been the standard method [14] for measuring baroreflex sensitivity in man. In this technique, the slope of the pulse interval against the systolic blood pressure (ms R-R interval mmHg⁻¹) is taken as a measure of baroreflex sensitivity or gain. Latterly, the spontaneous variability of heart rate and blood pressure [15,16] have been used to determine baroreflex gain described by the α -index. To determine the α -index, R-R interval and systolic pressure spectra are calculated.

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These estimate the amplitude of fluctuations as a function of frequency. The ratio of the changes in the R-R interval to changes in systolic pressure obtained from the spectra gives α , an estimate of the baroreflex gain (ms mmHg^{-1}) as a function of frequency. Some studies suggest that the mid-frequency (MF; 0.05–0.15 Hz) component of the α -index is a quantitative index of the sympathetic activity controlling heart rate and vasomotor activity, and the high-frequency (0.15–0.35 Hz) respiratory component of the α -index reflects parasympathetic activity [17]. Good agreement between α estimated by spectral analysis and baroreflex sensitivity measured by the phenylephrine pressor method has been found [15,18,19]. Comparison of Finapres and intra-arterial blood pressure recordings of systolic blood pressure variability indicates that the Finapres device over-estimates mid-frequency component variability [18].

In a previous cross-sectional study, we observed that α_{HF} in endurance-trained athletes was greater than in a group of elderly, sedentary subjects, suggesting that long-term aerobic training may modify baroreflex sensitivity [20]. As cross-sectional studies of the effect of exercise on age-associated changes are subject to many confounding influences, we undertook an intervention study in sedentary, older subjects to determine whether exercise training directly modifies baroreflex sensitivity or is an indirect association caused by other factors. The aim of the present study was to determine the effects of aerobic exercise training on baroreflex sensitivity as measured by the α -index in healthy, sedentary, elderly, normotensive individuals. Yoga training was used as a non-aerobic control intervention.

Methods

Subject details and study design

Healthy, sedentary, elderly subjects were recruited from the local community by newspaper advertisement as part of a larger study. From 91 initial responses, 36 were excluded by telephone conversation because of a significant past medical condition, being a current or recent smoker, being on regular medication or lack of time. A further 15 were excluded following screening because of anaemia (3), ECG abnormalities (5), aortic stenosis (2), mitral incompetence (1), fasting cholesterol > 6.5 (3) or family commitments (1). All subjects had a normal history, examination, full blood count, random blood glucose, hepatic, renal and thyroid function, urinalysis, fasting lipids, resting and exercise ECG to maximum heart rate. The study was approved by the Newcastle joint ethics committee.

The 40 healthy, sedentary, elderly subjects who passed the screening procedures were enrolled in the exercise training programme. Subjects were randomized into two groups. One group of 20 (seven women) underwent 6 weeks of exercise bicycle-based, aerobic training. The second group of 20 (10 women) undertook 6 weeks of yoga training, a non-aerobic intervention (twice weekly for 1.5 h). Repeat studies were performed within 1 week

of completion of training on a non-exercise or yoga training day. Subjects were instructed to make no changes to their usual diet.

The screening exercise ECG allowed subjects to become familiar with the equipment and provided a guide to the workloads required for the subsequent exercise test, in which VO_2 max was determined. In the screening exercise test, to acclimatize them to the equipment, subjects were seated on a bicycle ergometer (Siemens, Ergomed) with a nose clip applied and breathing through the mouthpiece for 5 min before commencing cycling. Resting data were recorded during the last 2 min of that period. Ventilation minute volumes were measured using a rotating vane anemometer. Gas was sampled from a mixing chamber and analysed for CO_2 (infra-red, PK Morgan) and oxygen (paramagnetic, PK Morgan). Data from the gas analysers and ventilation monitor were processed by an IBM computer. Subjects cycled for 1 min at zero workload to warm up, after which the workload was increased by 15 W min^{-1} until exhaustion or predetermined exclusion criteria were met. These criteria were chest pain or discomfort consistent with angina, 0.2 mV or more ST segment depression, dysrhythmias more severe than occasional atrial or ventricular ectopic beats and abnormal blood pressure responses ($> 30 \text{ mmHg}$ rise in systolic BP or $> 15 \text{ mmHg}$ fall in diastolic BP) to exercise.

Training interventions

Aerobic training comprised a 10-min warm-up period followed by 20 min cycling at workload to increase the heart rate to 70–80% of maximum heart rate measured during the maximal exercise test. Subjects wore heart rate wrist monitors (Polar Electro, Finland) so that heart rates could be monitored constantly during the training periods, and bicycle resistance levels were adjusted to keep the subject's heart rate in the desired range. Training ended with a 10-min warm down.

Yoga comprised a warm-up period, followed by a series of static yoga postures and breathing exercises, ending with a 20-min relaxation period that involved subjects relaxing in the horizontal position (Hatha yoga). None of the subjects had performed yoga previously. The yoga postures used were selected specifically to be of a stretching nature with no repetitive muscular contraction to ensure that minimal physical exertion occurred.

Determination of VO_2 max

VO_2 max was determined at baseline and after training interventions on the bicycle ergometer described above. Subjects were seated on the ergometer with a nose clip on, breathing through the mouthpiece for 5 min, and resting data were recorded for the last 2 min of that period. Subjects cycled for 1 min at zero workload to warm up before the workload was smoothly increased over a 2-min period to 75% of the VO_2 max measured at the initial screening exercise test. Workload was further increased by 10 W min^{-1} until the subject was exhausted

or until a plateau of VO_2 max with increasing workload occurred. Oxygen uptake was measured every 30 s, and data collected in the last 30 s of each exercise workload were used for analysis. A maximal exercise test was defined as the achievement of two of the following three criteria: plateauing of VO_2 max with increasing intensity of exercise, respiratory exchange ratio of 1.1 or greater or maximum heart rate within 5 beats of the age-predicted maximum heart rate [21].

Blood pressure, α -index and heart rate variability determination

Data from 14 (four women) subjects in the aerobic-trained group were available for analysis. Three (two women) were unable to attend both measurement sessions, two (one woman) did not complete the study and one subject was excluded because of poor signal quality. Data from 12 (six women) subjects in the yoga-trained group were available for analysis. Four (two women) were unable to attend both measurement sessions, two (two women) did not complete the study and two subjects were excluded because of poor signal quality. α -Index, resting blood pressure and heart rate were measured on a non-training day separately from the determination of VO_2 max.

Subjects were studied following an overnight fast, and refrained from caffeine and alcohol for 24 h before each study day. For each subject, the α -index was estimated in the mid-frequency (MF; 0.05–0.15 Hz) and high-frequency (HF; 0.15–0.35 Hz) range before and after training interventions. The α -index and peak power values of heart rate variability were measured using methods described in detail previously [22]. Subjects rested supine in a temperature-controlled quiet room for 20 min before recordings were made over a further 20-min period. Subjects refrained from talking throughout the 40 min. In each subject, successive measurements following training interventions were made at similar times of the day. The surface ECG was recorded from a standard lead II, while the arterial pressure was measured using the Penaz technique (Finapres, Ohmeda) applied to the second finger of the right hand. The ECG and blood pressure signals were both digitized at 250 Hz (DAS-16 analogue-to-digital converter) and stored on computer (IBM) for subsequent offline analysis. Blood pressure and heart rate were determined from the mean of the second 20-min Finapres recording taken on each occasion.

Data analysis

The group means and standard deviations of heart rate variability and the α -index at HF and MF were used for comparison. Data are expressed as means $\pm$ SD. Paired data were compared using Student's *t*-test, and $P < 0.05$ was considered significant.

Results

Baseline results before the training interventions have been reported previously in a cross-sectional study examining baroreflex function in young, elderly and endurance-trained elderly athletes [19]. There were no significant differences in the baseline characteristics of the aerobic and yoga groups: age (66 ± 4 vs. 68 ± 5 years), weight (79 ± 10 vs. 75 ± 14 kg), height (171 ± 8 vs. 169 ± 8 cm), body mass index (26.8 ± 3.6 vs. 26.1 ± 2.8 kg m⁻²), resting heart rate (66.2 ± 15 vs. 71.5 ± 13 bpm), systolic blood pressure (137 ± 22 vs. 131 ± 24 mmHg).

VO_2 max

There was a $13 \pm 11\%$ (19.4 ± 6.1 vs. 22.0 ± 6.9 mL kg⁻¹ min⁻¹, $P < 0.01$) increase in VO_2 max after yoga, but a larger $25 \pm 16\%$ (21.7 ± 3.9 vs. 25.2 ± 16.4 mL kg⁻¹ min⁻¹, $P < 0.01$) increase after aerobic exercise training ($P < 0.05$ for comparison of changes).

Resting heart rate and blood pressure

Heart rate decreased following yoga (69 ± 8 vs. 61 ± 7 min⁻¹, $P < 0.05$) but not aerobic training (66 ± 8 vs. 63 ± 9 min⁻¹, $P = 0.29$). Systolic blood pressure did not change significantly following yoga (131 ± 24 vs. 127 ± 23 mmHg, $P > 0.05$) or aerobic exercise (132 ± 23 vs. 126 mmHg, $P > 0.05$).

Heart rate variability

No significant change occurred in HRV at either frequency (MF 2134 ± 1037 vs. 1847 ± 737 units, $P = 0.19$; HF 1577 ± 1518 vs. 1443 ± 905 units, $P = 0.55$) following aerobic training. Following yoga, there was a significant increase in the peak power values of heart rate variability in the MF (2158 ± 1455 vs. 3295 ± 1335 units, $P < 0.01$) but not HF (1485 ± 1139 vs. 1916 ± 970 units, $P = 0.87$) components (Fig. 1a and b).

α -Index

No significant changes in baroreflex sensitivity occurred in either α_{MF} (6.5 ± 3.5 vs. 6.2 ± 3.0 ms mmHg⁻¹, $P = 0.69$) or α_{HF} (8.5 ± 4.7 vs. 8.9 ± 3.5 ms mmHg⁻¹, $P = 0.65$) following aerobic training. Following yoga, there was a significant increase in α_{HF} (8.0 ± 3.6 vs. 11.5 ± 5.2 ms mmHg⁻¹, $P < 0.01$) but not α_{MF} (6.5 ± 3.5 vs. 7.6 ± 2.8 ms mmHg⁻¹, $P = 0.29$). (Fig. 2a and b).

Discussion

The main findings of this study are that 6 weeks of aerobic exercise training, associated with a significant training effect, did not modify baroreflex sensitivity as measured by the α -index in healthy, elderly, sedentary, normotensive subjects. This is in agreement with two

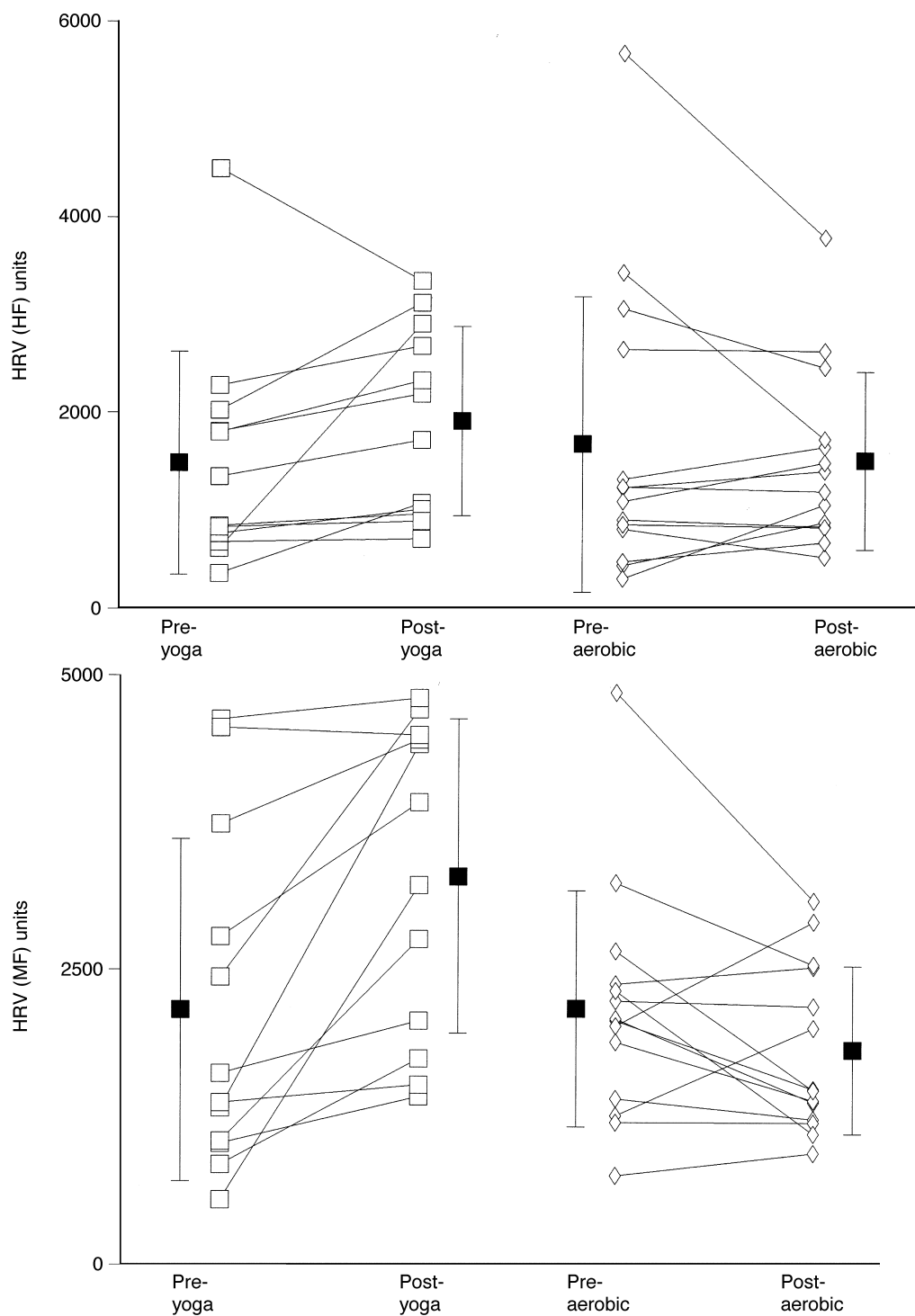


Figure 1. Heart rate variability in 26 healthy, sedentary, elderly subjects before and after yoga ($\square$, $n = 12$) and aerobic exercise training ($\diamond$, $n = 14$) for (a) high frequency (HF; 0.15–0.35 Hz) and (b) mid-frequency (MF; 0.05–0.15 Hz).

previous studies of younger subjects, which examined the effects of aerobic training on baroreflex sensitivity using other methods of baroreflex measurement [10,11]. In contrast, a 6-month study of aerobic exercise training in younger, hypertensive individuals resulted in an increase in the α -index at both frequencies [15]. Our

study also found a significant increase in α_{HF} following yoga. To our knowledge, no studies have previously investigated the effects of yoga on baroreflex sensitivity.

A 12% increase in VO_2 max occurred after yoga. Some of this increase may be caused by a low-grade aerobic training effect of the stretching exercises, which could

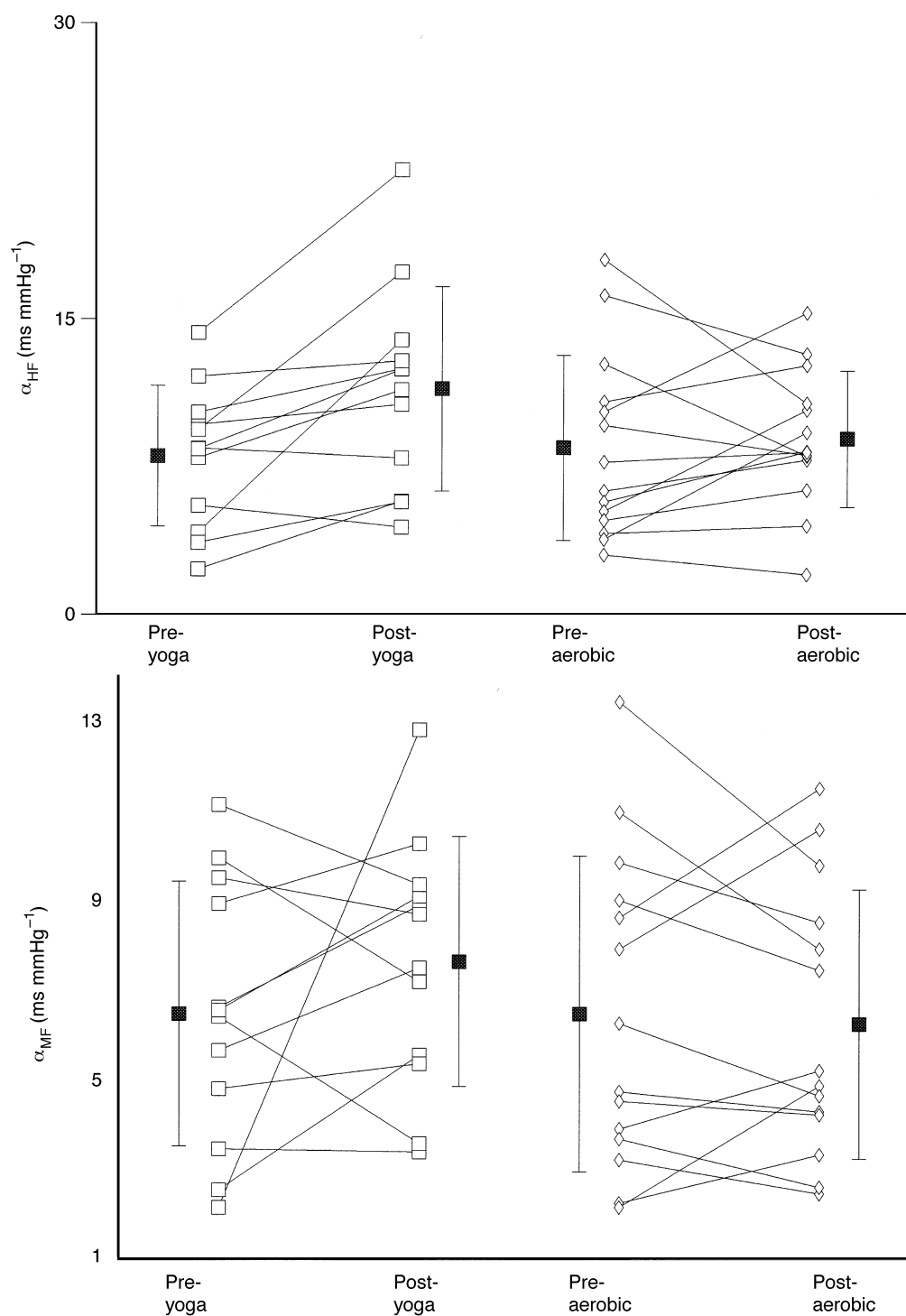


Figure 2. Baroreflex sensitivity as determined by the α -index in 26 healthy, sedentary, elderly subjects before and after yoga ($\square$, $n=12$) and aerobic exercise training ($\diamond$, $n=14$) for (a) high frequency (HF; 0.15–0.35 Hz) and (b) mid-frequency (MF; 0.05–0.15 Hz).

include some muscular contraction, or the increased physical activity associated with travelling to the yoga sessions, but it is more likely to be an habituation effect. The 25% increase in VO_2 max with aerobic exercise training is at the upper end of the effects from this type of training that have been observed in previous studies

using similar training regimens [15,23,24]. Given the changes in the control group, a component of this increase may have resulted from habituation, although all subjects were familiar with a graded bicycle exercise test from the screening ECG test and, even allowing for this, a significant training effect was evident.

In a previous study, we observed that, in contrast to the lack of effect of short-duration aerobic training on α_{HF} in this study, α_{HF} in endurance-trained athletes was higher than in a group of elderly, sedentary subjects [19]. This suggests that long-term aerobic training, unlike short-duration training, may modify baroreflex sensitivity, or that older individuals undertaking endurance training have existing increased baroreflex sensitivity for other reasons. In middle-aged, hypertensive subjects, exercise training increased baroreflex sensitivity [15]. In that study, the changes were thought to be caused by simultaneous reduction of sympathetic influence and an increase in vagal modulation. Therefore, at rest in hypertensive subjects, a readjustment of sympathovagal balance with an increase in the gain of BRS and enhanced vagal modulation appears to be the major adaptive change in autonomic regulation induced by exercise training. The effect of aerobic exercise training on baroreflex sensitivity in older hypertensive subjects would be of interest. No change occurred in HRV at either MF or HF, suggesting that 6 weeks of exercise training does not affect either vagal modulation or sympathetic influences. The duration of training in our study may have been insufficient to modify HRV, as the previous study, which demonstrated a change in hypertensive subjects, used a 6-month training period. Similarly, the intensity of training may not have been great enough, although this would appear to be unlikely, given the observed increases in $\dot{V}O_2$ max. The mechanisms causing a reduction of baroreflex sensitivity with ageing and hypertension may differ, and changes associated with hypertension but not ageing may be modifiable by exercise.

Previous studies have demonstrated a reduction in resting heart rate with short-duration exercise training, but this did not occur in our study, despite a significant training effect, as evidenced by the increase in $\dot{V}O_2$ max. This may relate to the duration of training, or reflect a type II statistical effect [21]. The reduction in heart rate following yoga has been observed in previous studies [26] and is likely to be related to increased vagal activity, as demonstrated by an increase in the HF peak power of HRV. There was no reduction in HRV at MF, suggesting a change in sympathetic withdrawal. Changes in vagal tone may have been induced by the relaxation and breathing techniques used in yoga training, which have been purported to affect autonomic balance [26,27]. The lack of a significant aerobic training effect with yoga suggests that the decrease in heart rate following yoga is not caused by improved cardiovascular fitness. An increase in α_{HF} but not α_{MF} occurred following yoga, suggesting that α_{HF} and α_{MF} are independent variables that can be modified separately. This is likely to be because of an increase in vagal tone, as evidenced by an increase in HRV at HF following yoga and may be induced by relaxation techniques used in the practice of yoga. A controlled study of yoga against a non-active control intervention would be required to determine whether this effect is directly related to yoga itself.

In conclusion, short-duration aerobic training does not modify baroreflex sensitivity as measured by the α -index

in healthy, normotensive, elderly subjects. Yoga training increases the α_{HF} but not the α_{MF} component of the baroreflex and the HF but not the MF component of HRV. These findings suggest that age-related changes in baroreflex sensitivity in normotensive, elderly subjects are unrelated to exercise status. The reported effect of exercise training on reversing the reduction in baroreflex sensitivity associated with hypertension in younger subjects suggests that differing mechanisms may be responsible for the reduction in baroreflex sensitivity observed in hypertension and ageing.

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