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Early dependency on sympathetic drive during dynamic exercise in the elderly

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Summary I investigated age-associated differences in the cardiovascular autonomic responses to dynamic exercise in 12 healthy young (age, 21 ± 1 yr) and 10 healthy elderly (age, 72 ± 5 yr) men. Subjects performed symptom-limited supine ramp ergometer exercise. I assessed autonomic activity through the amplitude of the high-frequency (0.15 to 0.85 Hz) component of the R-R interval fluctuation (RRHF), an index of cardiac vagal activity, and the amplitude of the low-frequency (0.04 to 0.15 Hz) component of mean blood pressure fluctuation (BPLF), an index of vasomotor sympathetic activity. The peak exercise workload and the peak increase in heart rate were lower in the elderly men than in the young men. In the elderly men compared with the young men, the amplitude of RRHF was lower at rest (14 ± 6 vs 42 ± 11 ms, $P<0.001$), showed a smaller absolute decrease during exercise (11 ± 6 vs 39 ± 13 ms, $P<0.05$) and withdrew at a lower intensity (50 vs 80%) of peak exercise. At the intensity, similar low values of the RRHF amplitude were found in the two groups (3 ± 1 ms). The resting value of the amplitude of BPLF and its peak increase at the peak exercise were similar in both groups; however, the BPLF amplitude started to increase at a lower exercise intensity in the elderly men than in the young men (40 vs 60% of peak exercise). These findings indicate that in elderly men compared with young men, both withdrawal of cardiac vagal activity and augmentation of sympathetic activity occur earlier during dynamic exercise with increasing workload, suggesting greater dependency on sympathetic mechanisms to generate cardiovascular responses to dynamic exercise in elderly men.

Key Words: *heart rate variability, blood pressure variability, aging, complex demodulation, human.*

Introduction

Healthy aging is associated with a progressive decline in the chronotropic response to exercise. During strenuous dynamic exercise, the response is mediated by sympathetic stimulation, while, during mild exercise, it depends on withdrawal of the cardiac vagal activity (Robinson *et al.*, 1966). Sympathetic activity at rest and its increase during exericse have been reported to be greater in elderly (Esler *at al.*, 1995; Saito *at el.*, 1993). Studies using heart rate variability to assess vagal control of heart rate have reported that the resting level of the cardiac vagal activity, referred to as "cardiac vagal reserve," shows a marked decease with age (Lipsitz *et al.*, 1990; Taylor *et al.*, 1995). Furthermore, in a recent study, I found that such decrease in cardiac vagal reserve correlated with the decrease in chronotropic response to dynamic exercise (Okada, 1996). It is therefore plausible that elderly people, compared with young, may be withdrawn in their cardiac vagal reserve at a lower intensity of dynamic exercise and need to depend more on the sympathetic augmentation to obtain further chronotropic response. However, no studies have been reported on the age-associated differences in such functional transitions of autonomic control mechanisms during dynamic exercise of increasing intensity.

I investigated age-associated differences in the autonomic functional transition during dynamic exercise in healthy subjects using the frequency analysis of heart rate and blood pressure variability. To determine the exercise intensity at which the autonomic functional transitions occur, I devised a unique exercise protocol, in which the exercise intensity was increased in a linear manner using a computer-controlled ergometer. I also employed complex demodulation (CDM) (Hayano *et al.*, 1993 and 1994; Taylor *et al.*, 1995), a nonlinear time-domain method of time series analysis, to analyze the continuous changes in the frequency components of heart rate and blood pressure variability as a function of time.

Methods

Subjects

Subjects included 12 healthy young men (mean age ± SD, 21 ± 1 yr) and 10 healthy elderly men (mean age ± SD, 72 ± 5 yr). None of the subjects was an athlete. Subjects were excluded if they had a medical history of chronic disease or were currently receiving any medication. Subjects underwent routine laboratory tests, chest radiography, and resting and exercise electrocardiography (ECG) to exclude cardiovascular or pulmonary disease, uremia, diabetes mellitus, and other diseases that can affect autonomic nervous system function. None of the subjects was a current smoker. The study protocol was in accordance with the Ethical Guidelines of Nagoya City University Medical School. Written informed consent was obtained from all subjects.

Study protocol

Subjects were instructed to refrain from consuming any beverages containing caffeine or alcohol after 2100 h the night before the experiment. All examinations were performed between 1100 h and 1500 h at least 3 hours after a light meal. Subjects were placed on the padded table of a supine bicycle ergometer (232C model 50M, Combi, Tokyo, Japan) and their long-axial movements were restricted by shoulder pads fixed to the table. Arterial blood pressure was monitored continuously by tonometry (Jentow 7000, Colin Electronics, Komaki, Japan). The left arm and wrist were fixed in the abduction position with a moldable pad (Vac-Pac, Nikko Fines Industries, Tokyo, Japan) supported on a sideboard attached to the table. Subjects remained in this position for at least 30 minutes before beginning the exercise.

After 5-minutes supine rest for baseline data collection, symptom-limited leg cycling was performed in the supine position. The workload was continuously increased so that I could identify the exercise intensity at which the cardiac functional autonomic transition occurred. For this purpose, I used workload control software (232C Software Library, Load control program ver. 2.0, Combi, Tokyo, Japan) that generated a linear increase in workload pattern from 0 W at a rate of 0.33 W /s. During the exercise, subjects pedalled at a frequency of 50 rounds /min until exhaustion, which was defined as the point at which subjects could no longer maintain a pedal frequency > 45 rounds /min.

Measurements

During rest and exercise, a surface ECG (CM5 lead), an uncalibrated spirogram obtained by thoracic impedance (AI-601G, Nihon Koden, Tokyo, Japan), and the tonometric noninvasive blood pressure of the left radial artery were recorded continuously on frequency-modulation (FM) tape with a data recorder (MR-30, Teac, Tokyo, Japan). The reliability of tonometry for noninvasive measurement of continuous blood pressure has been confirmed in previous studies (Chen *et al.*, 1997; Hayano *et al.*, 1993), but I was concerned that movement artifacts due to intense dynamic exercise could compromise accurate tonometric measurement of the blood pressure waveform and its variability. Therefore, I assessed the validity and accuracy of tonometry for the measurement of blood pressure during dynamic exercise in 4 healthy men who underwent simultaneous recordings of direct and tonometric measurement of arterial blood pressure during dynamic exercise performed according to the study protocol. I found that the tonometric blood pressure obtained from the left radial artery showed a high beat-to-beat correlation with the right radial arterial blood pressure measured with an 18-gage cannula (Nihon Koden, Tokyo, Japan) (r=0.98 to 0.99, 0.74 to 0.99, and 0.97 to 0.99 for systolic, diastolic, and mean blood pressures, respectively; P<0.001 for all variables).

Data analysis

Signals of ECG, blood pressure and spirogram were digitized on a personal computer (P5-150, Gateway 2000, Sioux City, SD, USA) using an analog-to-digital converter (DI200AC ver. 1.58, DATAQ Instruments Inc., Akron, Ohio, USA) at a sampling frequency of 1 kHz. R-wave peaks were detected automatically with a fast peak detection algorithm. Beat-to-beat systolic and diastolic blood pressures were determined as the maximum and minimum values, respectively, during each R-R interval. The mean blood pressure was

calculated from the area under the blood pressure waveform within each R-R interval. When an R-R interval was ectopic or in some other way abnormal, the R-R interval and the corresponding blood pressure data were excluded from the data analysis. No subjects showed 2 or more ectopic beats during the measurement. The time series of the R-R interval and the mean blood pressure were interpolated with a cubic spline function and the interpolated time series with the spirogram signals were then resampled at 2 Hz to obtain equidistant time series. This 3-variate time series was used for further analysis.

We performed CDM of heart rate and blood pressure fluctuation to assess the time- dependent changes in the amplitude of the low-frequency (0.04 to 0.15 Hz) and the high-frequency (0.15 to 0.85 Hz) components in their fluctuations. I assessed cardiac vagal activity by the amplitude of the high-frequency component of R-R interval (RRHF) (Pomeranz *et al.*, 1985), cardiac sympathetic nerve activity by the ratio of amplitude of low-frequency to high-frequency components (RRLF/RRHF) (Mukai & Hayano, 1995) and vasomotor sympathetic activity by the amplitude of the low-frequency component of mean blood pressure variability (BPLF) (Furlan *et al.*, 1987; Rimoldi *et al.*, 1990).

CDM was performed using a previously published algorithm and parameters (Hayano *et al.*, 1993 and 1994) on the personal computer. Briefly, CDM consists of three steps: a frequency shift of the spectral region of interest to zero frequency, low-pass filtering and polar form conversion of the real and imaginary parts of the low-pass filtered signal. For analysis of the RRHF, RRLF and BPLF, frequencies were shifted by -0.500, -0.095, and -0.095 Hz, respectively, and low-pass filtering was performed with corner frequencies of 0.350, 0.055, and 0.055 Hz, respectively, using a 61-term, zero-phase-shift, least-square filter with convergence factors (transitional band width, 0.033 Hz). Thus, the frequency bands for RRHF, RRLF, and BPLF ranged from 0.150 to 0.850 Hz, from 0.040 to 0.150 Hz, and from 0.040 to 0.150 Hz, respectively. The respiratory frequency was also obtained by CDM of the spirogram signal with a frequency shift of -0.450 Hz and a corner frequency of 0.400 Hz, resulting in a frequency range of 0.050 to 0.850 Hz.

Statistical analysis

Except for the raw data in the figure showing representative results, the heart rate, blood pressure, and autonomic parameters during exercise are presented in figures as the change from the baseline value. The baseline values were defined as the average of a parameter over the 5 minutes immediately preceding exercise. Because the time to exhaustion varied among subjects, the values during exercise are presented as a function of the time to exhaustion in each subject (Taylor *et al.*, 1995), expressed as the average value over every 10 % of the exercise duration.

Data were analyzed using the Statistical Analysis System program (SAS Institute Inc., Cary, NC, USA). The differences in mean values between groups were evaluated by the Student's t-test. The time-dependent changes in hemodynamic and autonomic parameters were evaluated by repeated-measures analysis of variance (ANOVA) using a Helmert transformation for baseline values and the SAS General Linear Model Procedure. Data are presented as the mean ± SEM in figures and as the mean ± SD in the text. A P value <0.05 was considered significant.

Results

Fig. 1 shows the instantaneous heart rate, systolic and diastolic blood pressure, respiration and autonomic parameters in young and elderly subjects during rest and dynamic exercise. Fig. 2 and 3 show the ensemble averages of the mean values over every 10 % of the exercise duration in young and elderly men.

Baseline values

The resting heart rate was lower in elderly men than in young men (59 ± 5 vs 68 ± 12 bpm, $P<0.001$) and systolic, diastolic, and mean blood pressures were higher in elderly men than in young men (137 ± 9 vs 114 ± 7, 77 ± 9 vs 66 ± 7 and 97 ± 10 vs 81 ± 5 mm Hg, $P<0.001$ for all). There were no significant differences in the respiratory frequency between groups (0.27 ± 0.05 vs 0.26 ± 0.03 Hz). The amplitude of the RRHF was lower in elderly men than in young men (14 ± 6 vs 42 ± 11 ms, $P<0.001$), although there was no significant difference in the RRLF/RRHF or the amplitude of the BPLF between groups (1.2 ± 0.6 vs 0.9

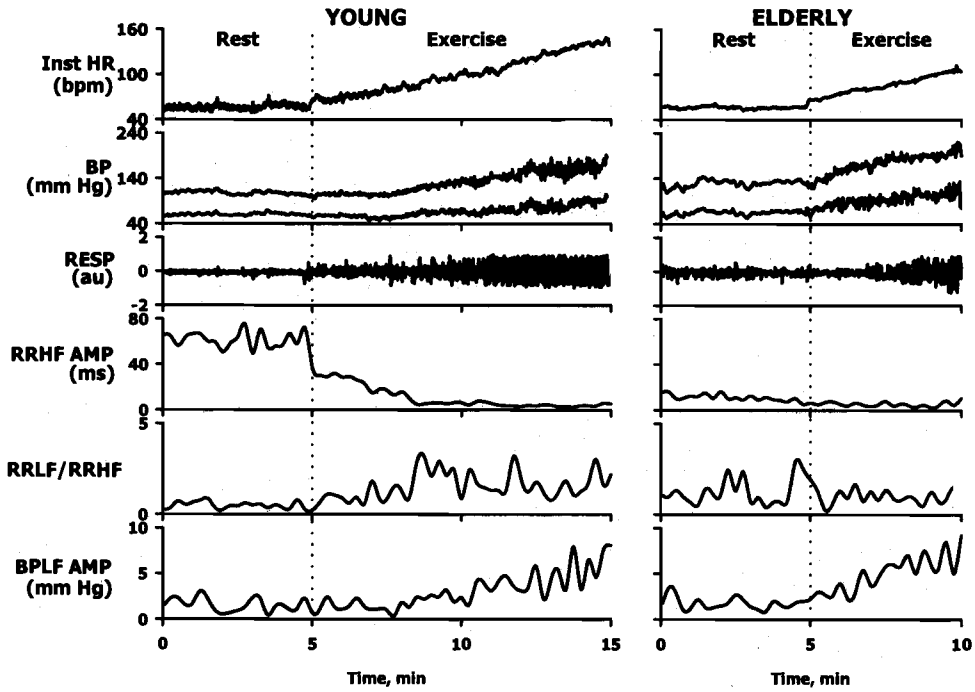

Fig. 1. Instantaneous heart rate (HR), systolic and diastolic blood pressures (BP), respiration (RESP), the amplitude of the high-frequency component of R-R interval variability (RRHF), the ratio of the low-frequency component to the high-frequency component of R-R interval variability (RRLF/RRHF), and the amplitude of the low-frequency component of mean blood pressure (BPLF) in representative young and elderly subjects during 5-minute rest and dynamic exercise with a linear-ramp workload (0.33 W/s). Time-dependent changes in RRLF/RRHF and the amplitude of RRHF and BPLF were assessed continuously by complex demodulation.

± 0.5 for RRLF/RRHF; 1.6 ± 0.4 vs 1.7 ± 0.6 mm Hg for the amplitude of BPLF in elderly and young, respectively).

Response to dynamic exercise

The peak exercise intensity was lower in elderly men than in young men (119 ± 15 vs 199 ± 14 W). Although the heart rate increased progressively with increasing exercise intensity in both groups ($P<0.001$, repeated measures ANOVA for the effect of intensity), the increase was smaller in elderly men (Fig. 2). Systolic, diastolic, and mean blood pressures increased with exercise and the increases were similar in both groups. The respiratory frequency also increased progressively with increasing exercise intensity until peak exercise in both groups ($P<0.001$ for both; Fig. 2). The increases in the respiratory frequency were similar in both groups throughout exercise (Fig. 2).

Although the amplitude of the RRHF decreased progressively with increasing exercise intensity in both groups ($P<0.001$, repeated measures ANOVA for the effect of intensity), the decrease was smaller in elderly men than in young men (Fig. 3). The amplitude of the RRHF reached its nadir at 50% of the maximum exercise intensity in elderly men and at 80% of the maximum exercise intensity in young men ($P<0.05$; repeated measures ANOVA with Helmert transformation). At the nadir of the RRHF amplitude, similar low values of the RRHF amplitude were found in the both groups (3 ± 1 ms). At the intensity, After reaching its nadir, the RRHF amplitude showed no further changes throughout exercise in either group. The RRLF/RRHF did not change significantly during dynamic exercise in either group (Fig. 3).

In young men, the amplitude of the BPLF did not change significantly until 60 % of maximum exercise intensity at which it started to increase, while in elderly men the amplitude of the BPLF started to increase at 40 % of the maximum exercise intensity ($P<0.05$ for both; repeated measures ANOVA with Helmert transformation; Fig. 3). The increases in the amplitude of the BPLF from rest to exhaustion did not differ significantly between groups.

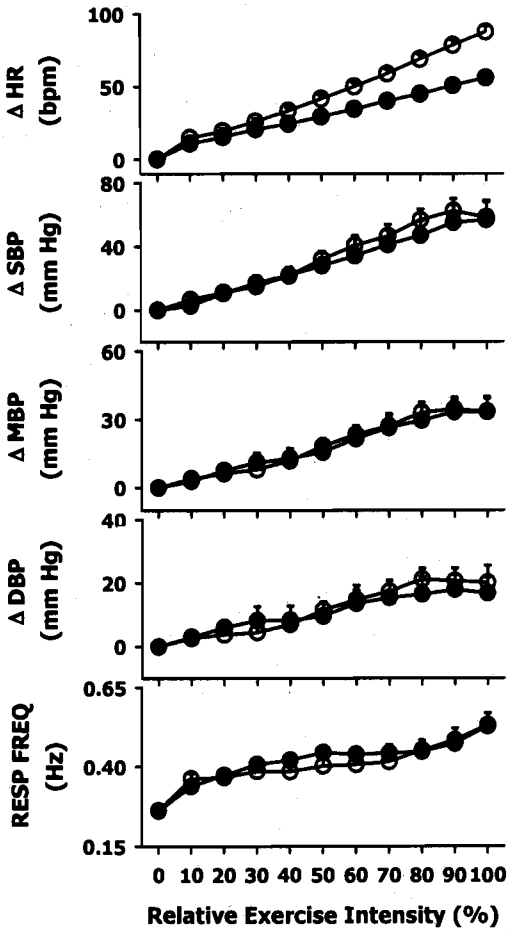

Fig. 2. Changes in heart rate (ΔHR), systolic blood pressure (ΔSBP), mean blood pressure (ΔMBP), and diastolic blood pressure (ΔDBP) and the absolute respiratory frequency (RESP FREQ) in young and elderly men during exercise. Data represent the mean of the average for every 10% period of the individual exercise duration. Open circles indicate young men and closed circles indicate elderly men.

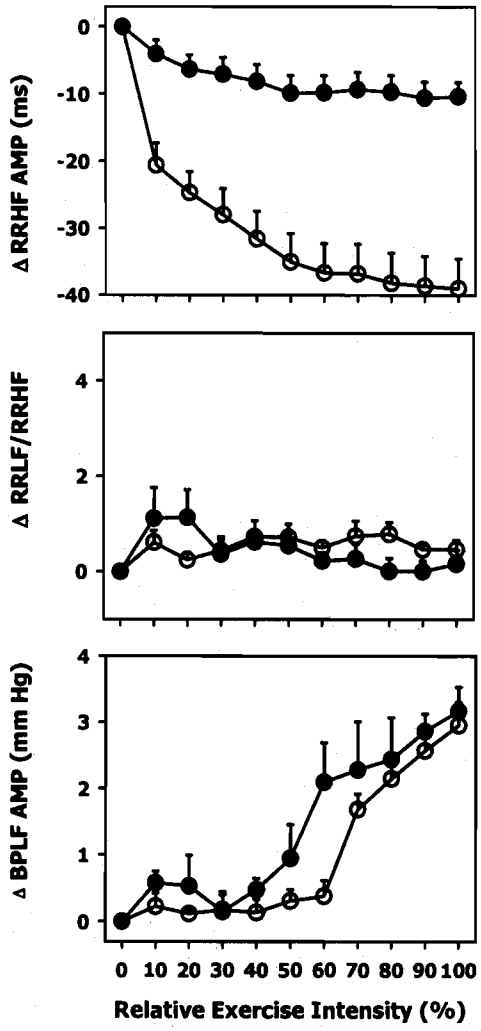

Fig. 3. Absolute changes in the amplitude of the high-frequency component of R-R interval variability (ΔRRHF), the ratio of the amplitude of the low-frequency to the high-frequency components of R-R interval variability (ΔRRLF/RRHF), and the amplitude of the low-frequency component of mean blood pressure variability (ΔBPLF) in young and elderly men during exercise. Data represent the mean of the average over every 10% period of individual exercise duration. Open circles indicate young men and closed circles indicate elderly men.

Discussion

The amplitude of the RRHF in elderly men was characterized by a lower baseline value, a smaller absolute decrease in response to dynamic exercise and a complete withdrawal at a lower intensity of dynamic exercise compared with young men. Although there was no difference in the amplitude of the BPLF at peak exercise between the young and elderly men, it began to increase at a lower exercise intensity in elderly men. Although previous studies have shown a decrease in the RRHF amplitude (Gregoire *et al.*, 1996; Yamamoto *et al.*, 1991) and an increase in the BPLF amplitude (Rimoldi *et al.*, 1990) in response to dynamic exercise, these studies did not examine age-associated differences in the transition of these autonomic indices when the intensity of exercise was progressively increasing.

1. Assessment of autonomic responses by heart rate and blood pressure variavility

The RRHF is mediated solely by the vagus nerve (Pomeranz *et al.*, 1985) and its amplitude reflects vagal modulation of sinus node activity (Hayano *et al.*, 1991). The amplitude of the BPLF has been proposed as an index of vasomotor sympathetic activity, because it increases during head-up tilt and dynamic and isometric exercise (Furlan *et al.*, 1987; Rimoldi *et al.*, 1990; Mukai & Hayano, 1995).

The RRLF/RRHF has been proposed as an index of cardiac sympathetic activity, because it increases in the standing position (Hirsch & Biship, 1981; Lipsitz *et al.*, 1990; Mukai & Hayano, 1995). However, this index tends to change reciprocally to the change in the amplitude of the RRHF by its definition (Casadei *et al.*, 1995). Furthermore, the RRLF may reflect the fluctuation of the R-R interval caused by the BPLF via the baroreceptor reflex mechanism (Madwed *et al.*, 1991), and the sensitivity of the baroreceptor reflex decreases with age (Gribbin *et al.*, 1971) and with exercise (Stratton *et al.*, 1994). Thus, RRLF/RRHF during exercise may not accurately reflect cardiac sympathetic activity. In the present study, indeed, the RRLF/RRHF did not change significantly during dynamic exercise in either group.

I used the mean blood pressure to assess blood pressure variability, because the systolic blood pressure can be influenced not only by the original driving pressure but also by secondary modulations of the pressure waveform caused by the Windkessel effect, summation of the reflected pulse wave (Kelly *et al.*, 1989), and increasing pulse pressure with transmission to the periphery (Kelly *et al.*, 1989). Although the pressure transmission may be influenced by the intensity of dynamic exercise as well as by age (Kelly *et al.*, 1989), the mean blood pressure seems less affected by these secondary effects (Smith *et al.*, 1990). The mean blood pressure appears to be superior to the systolic blood pressure for blood pressure variability analysis when the goal is to quantify the fluctuations in peripheral resistance, which reflect vasomotor sympathetic activity.

Previous studies have examined heart rate variability (Gregoire *et al.*, 1996; Yamamoto *et al.*, 1991) and blood pressure variability (Rimoldi *et al.*, 1990) in response to dynamic exercise using spectral analysis. These studies used a steady-state exercise protocol because spectral analysis assumes that data are stationary during the period of analysis. In the present study, I used a linear ramp (0.33 W/s) exercise protocol to determine the exact time at which the autonomic functional transition of cardiac vagal withdrawal and sympathetic augmentation occurred (Okada, 1996). The technique of CDM allowed us to analyze non-stationary heart rate and blood pressure variability data and continuously provided time-dependent changes in the amplitudes of the RRHF and BPLF with a temporal resolution of <15 s (Hayano et al., 1993 and 1994; Taylor *et al.*, 1995).

2. Effects of age on cardiac vagal response to dynamic exersise

Although the RRHF amplitude at rest in the supine position was lower in elderly men than in young men, which is consistent with the results of previous studies (Lipsitz *et al.*, 1990), the RRHF amplitude at the nadir during dynamic exercise was similar in both groups. Consequently, the absolute decrease in the RRHF amplitude in response to dynamic exercise was smaller in elderly men, which is consistent with the result by Gregoire *et al.*, (1996) who studied in middle aged subjects. More importantly, however, the amplitude reached its nadir earlier in the elderly than in the young men (50% vs 80 % of relative exercise intensity). The amplitude remained stable after reaching the nadir in both groups. Ribeiro *et al.*, (1991) reported that atropine did not increase the heart rate at peak exercise, suggesting that there is no residual cardiac vagal activity at peak exercise. Thus, the present findings that the RRHF amplitude reached its nadir before peak exercise and remained stable thereafter suggest that cardiac vagal activity had withdrawn before the peak exercise intensity was achieved and that the withdrawal occurred earlier in the elderly than the young men.

The heart rate increased linearly, even after the RRHF reached its nadir in both groups, although the maximal increase in heart rate at peak exercise was smaller in elderly men. Thus, the increase in heart rate after the nadir appeared to have been caused by factors other than cardiac vagal withdrawal, such as sympathetic augmentation and/or an increase in the intrinsic heart rate (Jose *et al.*, 1970; Saito *et al.*,1993; Shen *et al.*, 1992). These findings seem to be consistent with the widely accepted theory that tachycardia at a lower intensity of exercise is caused primarily by cardiac vagal withdrawal, while tachycardia at a higher intensity of exercise is caused by cardiac sympathetic augmentation (Robinson *et al.*, 1966). The present results further suggest that this functional transition in autonomic control occurred earlier in elderly men and

that the subsequent tachycardic response to dynamic exercise was less dependent on cardiac vagal withdrawal in elderly men.

The relationship between the tachycardic response and the autonomic functional transition is illustrated in Fig. 4, which shows the age-associated difference in the increase in heart rate due to cardiac vagal withdrawal. Although the overall tachycardic response was smaller in the elderly men than the young men, the tachycardic response after cardiac vagal withdrawal was greater in elderly men, suggesting that the decreased tachycardic response in elderly men was mainly attributable to a decreased reserve of cardiac vagal activity, which was reflected by decreased cardiac vagal activity at rest. This theory is consistent with the results of our previous study on age-associated differences in the cardiac vagal response to isometric exercise

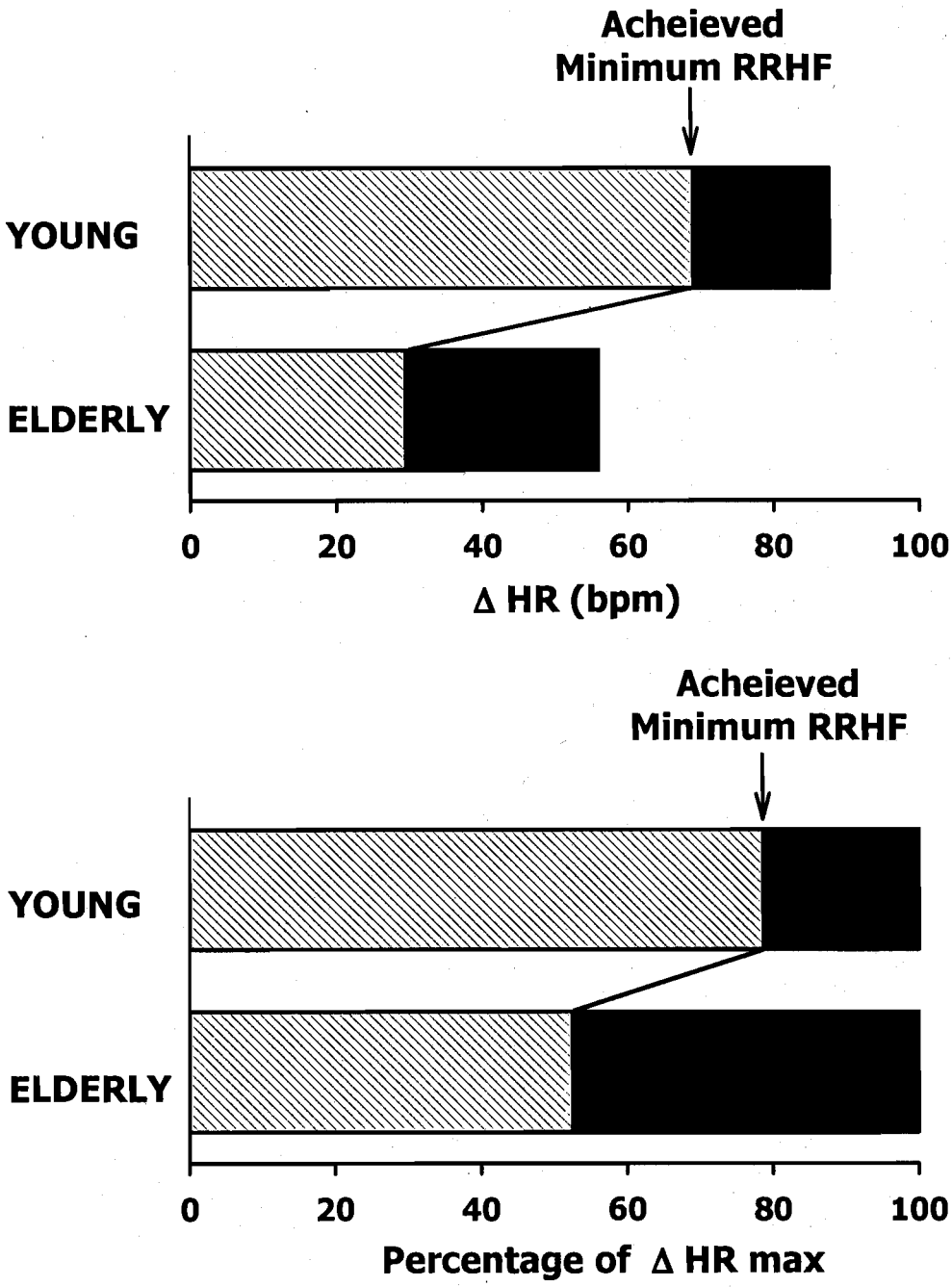

Fig. 4. The relationships between tachycardiac response and the autonomic functional transition. The fraction of the increase in heart rate (ΔHR) in both absolute (upper panel) and relative (lower panel) attributable to achieved minium RRHF (hatched bars) was greater in young men. Despite a smaller overall tachycardic response in elderly men, the fraction of the absolute heart rate increase after achieving minimum RRHF (solid bars) was greater in the elderly men (upper panel).

(Taylor *et al.*, 1995), in which the decreased tachycardic response to isometric exercise in the elderly men was associated with an inability to reduce the cardiac vagal tone below the already decreased baseline level.

3. Effects of age on vasomoter sympathetic responses to dynamic exercise

The amplitude of the BPLF did not differ significantly between the elderly and the young men either at rest or in the maximal increase at peak exercise. These findings seem consistent with those of Veerman *et al.* (1994), who observed a decrease in the power of the BPLF (the mid-frequency component in their study) with age at rest in the supine and standing positions. Previous studies have shown increases in the muscle sympathetic nerve activity and in the serum concentration of norepinephrine at rest (Esler *et al.*, 1995) and increased responses of sympathetic markers to dynamic exercise in the elderly (Esler *et al.*, 1995; Saito *et al.*, 1993). These observations, the findings of Veerman *et al.* (1994), and the present study suggest that absolute value of BPLF amplitude may not reflect accurately age-dependent changes in vasomotor sympathetic activity either at rest or during exercise.

Nevertheless, the BPLF amplitude has been reported to increase in response to dynamic and isometric exercise (Furlan *et al.*, 1987; Rimoldi *et al.*, 1990). I also observed a clear increase in the BPLF amplitude during dynamic exercise in both the young and the elderly men. This apparent discrepancy suggests that the BPLF amplitude reflects only *intra*-individual changes, not *inter*-individual differences in vasomotor sympathetic activity.

The controversy over the relationship between the BPLF amplitude and sympathetic nerve activity may be attributable to the fact that they may reflect different aspects of sympathetic function. The muscle sympathetic nerve activity and the serum level of norepinephrine may reflect presynaptic sympathetic activity, while the BPLF amplitude may reflect the overall sympathetic vasomotor function, which includes end-organ responsiveness as well as neural activity. Thus, it is plausible that even if sympathetic nerve activity is increased in elderly men, their vasomotor responses may be attenuated by decreased vascular responsiveness, resulting in a BPLF amplitude comparable to that in young men. This hypothesis is consistent with a recent study by Sugiyama *et al.* (1996), who reported that the pressor response to muscle sympathetic nerve activity was diminished in elderly men. I, therefore, suggest that the changes in the BPLF amplitude observed in the present study present a relative index of intra-individual changes in vasomotor sympathetic activity.

Despite this limitation, the response of the BPLF amplitude to dynamic exercise provides valuable information. In the present study, the BPLF amplitude did not increase until the exercise intensity reached a certain level and then increased with increasing exercise intensity until peak exercise was achieved. Also, the relative exercise intensity at which the amplitude began to increase was lower in elderly men than in young men (40 vs 60%). These findings are also consistent with an earlier autonomic functional transition in elderly men during dynamic exercise, suggesting that vasomotor sympathetic activity increases at a lower exercise intensity in elderly men.

4. Study limitations

The amplitude of the RRHF decreases with increasing respiratory frequency and a decrease in the tidal volume (Eckberg, 1983; Hirsch & Bishop, 1981); these changes are not mediated by the change in cardiac vagal activity (Grossman *et al.*, 1991; Hayano *et al.*, 1994). Respiratory parameters may be influenced by both dynamic exercise and age, which could explain the age-associated difference in the amplitude of the RRHF during exercise. During dynamic exercise, however, both the respiratory frequency and the tidal volume increase; these factors have opposite effects on the amplitude of the RRHF. In addition, there were no differences in the respiratory frequency at rest or during dynamic exercise between the elderly and young men. Although I was unable to evaluate the difference in the tidal volume between groups because we did not calibrate the spirogram, the tidal volume has a smaller influence on the amplitude of the RRHF than the respiratory frequency, particularly at higher tidal volumes (Eckberg, 1983).

I defined the relative exercise intensity as the percentage of the peak exercise intensity, which was determined by symptom limitations (Taylor *et al.*, 1995). It is possible that the relative intensity of peak exercise is influenced by age, which could affect the results of this study. There was no difference, however,

in the percentage of the peak heart rate relative to the predicted maximal heart rate for age ([220 − age] bpm) between the young and the elderly men (75 ± 13% vs 75 ± 5%, respectively). The findings in this study may not be due to aging per se, but age-associated condition such as deconditioning and/or blood pressure elevation with a decrease in baroreflex gain.

Conclusions

The present results suggest that elderly men have a decreased reserve of cardiac vagal activity at rest, leading to an earlier autonomic transition from vagal withdrawal to sympathetic augmentation in response to increasing exercise intensity, even though the peak exercise intensity is lower. The decreased cardiac vagal reserve in elderly men resulted in a decreased dependency of the chronotropic response on cardiac vagal withdrawal and may have been responsible, at least in part, for the decreased chronotropic response itself.

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