Influence of physical training on heart rate variability and baroreflex circulatory control

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SEALS, DOUGLAS R., AND PETER B. CHASE. *Influence of physical training on heart rate variability and baroreflex circulatory control.* J. Appl. Physiol 66(4): 1886–1895, 1989.—Nineteen males (aged 45–68 yr) were studied before and after either a period of regular endurance exercise [walk/jog 3–4 days/wk for 30 ± 1 (SE) wk, $n = 11$] or unchanged physical activity (38 ± 2 wk, $n = 8$) (controls) to determine the influence of physical training on cardiac parasympathetic (vagal) tone and baroreflex control of heart rate (HR) and limb vascular resistance (VR) at rest in middle-aged and older men. Training resulted in a marked increase in maximal O_2 uptake (31.6 ± 1.2 vs. 41.0 ± 1.8 ml·kg^{-1}·min^{-1}, 2.56 ± 0.16 vs. 3.20 ± 0.18 l/min, $P < 0.05$) and small ($P < 0.05$) reductions in body weight (81.2 ± 3.5 vs. 78.7 ± 4.0 kg) and body fat (23.8 ± 1.3 vs. 20.9 ± 1.3%). HR at rest was slightly, but consistently, lower after training (63 ± 2 vs. 58 ± 1 beats/min, $P < 0.05$). In general, HR variability (index of cardiac vagal tone) was greater after training. Chronotropic responsiveness to either brief carotid baroreflex stimulation (neck suction) or inhibition (neck pressure), or to nonspecific arterial baroreflex inhibition induced by a hypotensive level of lower body suction, was unchanged after training. In contrast, the magnitude of the reflex increase in forearm VR in response to three levels of lower body suction was markedly attenuated after training (38–59%; $P < 0.05$ at −10 and −30 mmHg; $P = 0.07$ at −20 mmHg). None of these variables or responses was altered over time in the controls. These findings indicate that in healthy, previously sedentary, middle-aged and older men, strenuous and prolonged endurance training *1*) elicits large increases in maximal exercise capacity and small reductions in HR at rest, *2*) may increase cardiac vagal tone at rest, *3*) does not alter arterial baroreflex control of HR, and *4*) results in a diminished forearm vasoconstrictor response to reductions in baroreflex sympathoinhibition.

exercise; exercise training; cardiac vagal tone; respiratory sinus arrhythmia; autonomic nervous system

IT IS WIDELY PRESUMED that regularly performed endurance (aerobic) exercise induces adaptations in the autonomic nervous system (ANS) that result in both changes in several cardiovascular variables at rest and in alterations in the reflex control of the circulation (21, 25, 33).

One example of the former is the lower heart rate (HR) at rest that is often observed in the physically trained state (14, 15, 23, 25). This bradycardia is thought to be mediated in part by a training-induced increase in cardiac parasympathetic (vagal) tone (1, 25). However, comparisons of endurance-trained and untrained humans and laboratory animals have failed to demonstrate consistent differences in vagal tone with the use of a variety of experimental approaches (14, 16, 17, 23).

Training-associated changes in baroreflex control of HR and vascular resistance (VR) have also been proposed (2, 12, 18, 33). However, the experimental findings to date have been remarkably inconsistent. Chronotropic responsiveness to arterial baroreflex perturbations has been reported to be greater (1), not different (10, 36), and blunted (30, 32, 33) in endurance-trained vs. untrained humans. Similarly, the reflex-mediated increases in limb VR evoked during experimentally induced reductions in cardiopulmonary/arterial baroreceptor sympathoinhibition have been reported to be markedly augmented (12), unchanged (10, 31), and attenuated (18, 36) in the endurance-trained state.

Because most of the information obtained to date has been derived from cross-sectional comparisons of trained and untrained groups of subjects, we reasoned that intersubject variability within a group and/or nontraining-related genotypic differences between groups may have contributed to the apparent discrepancies in the results of previous studies. Furthermore, because it has been suggested that increases in cardiac parasympathetic tone may exert a protective effect against life-threatening arrhythmias (4), we thought it important to determine whether such autonomic adaptations could be elicited by physical training in sedentary men of an age typically associated with the clinical manifestation of coronary heart disease. Thus, the purpose of the present investigation was to determine whether prolonged, moderately strenuous, endurance exercise training would evoke changes in cardiac vagal tone and in baroreflex control of HR and limb VR in previously untrained, healthy, middle-aged and older men.

METHODS

Subjects

Nineteen men (aged 45–68 yr) participated in this study after providing their written, informed consent. All subjects were normotensive (blood pressure ≤ 140/90 mmHg) and were free of overt cardiopulmonary disease as assessed by medical history and by electrocardiography both at rest and during an incremental exercise test to subjective exhaustion. At the time of enroll-

ment, none of the subjects was performing endurance (aerobic) exercise on a regular basis. Eleven men [aged 53 ± 2 (SE) yr] volunteered to participate in an exercise training program (exercise group). The other eight men (aged 54 ± 3 yr) volunteered to participate in the study as nonexercising controls (control group). All protocols and procedures in which these subjects were involved were approved by the institutional Committee on Human Subjects Use.

Experimental Procedures

Maximal O_2 uptake ($\dot{V}O_{2\,max}$). In the present study, $\dot{V}O_{2\,max}$ was used as a measure of maximal exercise capacity. Increases in $\dot{V}O_{2\,max}$ in response to the exercise program were used to document that a sufficient stimulus had been applied to evoke adaptations in the cardiovascular and skeletal muscle systems.

$\dot{V}O_{2\,max}$ was determined during graded treadmill walking or jogging as described previously (26). Inspired volumes were measured using a dry gas meter (Parkinson Cowan), and expired fractions of O_2 and CO_2 were determined using Applied Electrochemistry gas analyzers (S-3AI and CD-3A, respectively). Data were collected and analyzed in 30-s intervals with a microcomputer (Apple IIe). The criteria used to document that $\dot{V}O_{2\,max}$ had been attained during each test were *1*) a lack of increase in O_2 uptake with an increase in work rate (leveling off criterion), *2*) attainment of the maximal HR predicted for each subject's age, and *3*) a respiratory exchange ratio (O_2 uptake/CO_2 production) >1.1.

Body fatness. Body fatness was estimated from skinfold thicknesses determined at six sites using a Harpenden caliper. Body density and percent body fat were estimated using the equations of Jackson and Pollock (11) and Siri (29), respectively.

HR variability. Cardiac vagal tone was estimated by noninvasive measurement of HR variability (i.e., the respiratory sinus arrhythmia) with the use of conventional electrocardiography as described previously (23). The magnitude of the variability in HR is highly and directly correlated with the level of parasympathetic cardiac vagal tone as assessed using a variety of pharmacological and physiological experimental approaches (7, 8, 13).

After 15 min of quiet rest in the supine position, the subject's electrocardiogram (ECG, V-5 lead, Cardiotach Amplifier, Gould ES1000 recorder) was sampled by a microcomputer (IBM-PC model 176) that measured and stored each R-R interval over a 5-min period. To control for the effects of breathing on HR variability, subjects breathed at the rate of 10 breaths/min (3 s inspiration, 3 s expiration); the depth of breathing was increased slightly to maintain normal minute ventilation.

HR variability was assessed using *1*) the standard deviation of the R-R intervals (1, 7, 23) and *2*) the difference between the minimum and maximum R-R intervals (7, 17, 23). The difference between the average of the five shortest (minimum) and the average of the five longest (maximum) R-R intervals was used to calculate the latter. Nonsinus beats were infrequent (n = 21 for both testing sessions in the 2 subject groups combined) but were excluded from the analysis. The HR at rest was determined from the average R-R interval over the entire 5-min measurement period.

Arterial baroreflex control of HR. CAROTID BAROREFLEX. Carotid baroreflex control of HR was assessed noninvasively using a neck collar that allowed application of both negative (suction) and positive pressure to the anterior region of the neck as described previously (23). Neck suction increases carotid baroreceptor (inhibitory) afferent discharge by increasing carotid sinus transmural pressure and elicits a reflex prolongation of the R-R interval mediated primarily by an increase in efferent cardiac vagal tone (19). Neck pressure decreases carotid transmural pressure, which reduces carotid afferent discharge and evokes a shortening of the R-R interval initially because of a decrease in efferent cardiac vagal nerve activity (19). In the present study, carotid baroreflex control of HR was assessed by determining the magnitudes of the changes in R-R interval in response to neck suction and pressure.

With the subject in the supine position, suction (−10, −25, and −40 mmHg) or pressure (10 and 30 mmHg) was applied for 2 s during a held end expiration; the onset of the stimulus was timed to begin 0.8 s before the appearance of the next anticipated P wave as described by Eckberg (6). Each level of pressure and suction was applied at least seven times in random order with at least 45 s separating consecutive applications.

The R-R intervals before and after the onset of suction/pressure were measured and stored using the ECG signal that was fed into the microcomputer. The two R-R intervals immediately before the onset of the stimulus were averaged, and this value was used as the base-line (control) R-R interval. The length of these two R-R intervals differed by <50 ms in each trial. The R-R interval representing the largest change from control over the first five cardiac cycles after the onset of the stimulus was used to calculate the change in R-R interval (ΔR-R) for that trial. The maximum change in R-R interval normally occurred in the first or second cardiac cycle after the onset of the stimulus. For each stimulus level, the ΔR-R was calculated as the average response over all seven trials.

NONSPECIFIC ARTERIAL BAROREFLEX. Lower body negative pressure (LBNP) applied at levels >20 mmHg decreases venous return, central venous pressure, systolic blood pressure (SBP), and pulse pressure (PP) and results in reflex tachycardia (20). This tachycardia is likely mediated by reductions in both aortic and carotid (arterial) baroreflex central inhibition (20). We assessed nonspecific arterial baroreflex control of HR by examining the magnitude of the tachycardia (beats/min) per mmHg change in either SBP (ΔHR/Δ SBP) or PP (ΔHR/ΔPP) during a 2-min period of LBNP at −30 mmHg (see below).

Baroreflex control of forearm vascular resistance (FVR). Baroreflex control of VR was studied using LBNP to produce graded reductions in baroreflex-mediated sympathoinhibition and, thus, stepwise increases in sympathetic outflow and VR in the forearm (20). Recent evidence indicates that the reflex cardiovascular adjust-

ments to even low (nonhypotensive) levels of LBNP may result from reductions in both cardiopulmonary and arterial baroreceptor sympathoinhibition (5). In the present study, the magnitude of the increase in FVR from base line during a specific level of LBNP was used as a measure of nonspecific baroreflex vasoconstrictor responsiveness.

Subjects were positioned supine and sealed at the waist in a metal tank designed for administration of LBNP. After 15 min of rest, blood flow was measured in the right forearm every 15 s using venous occlusion plethysmography (28), arterial blood pressure was determined once per minute in the left arm by sphygmomanometry, and HR was measured continuously via ECG for an additional 2 min of rest (control) followed by 2 min of LBNP at −10 mmHg. After a subsequent 10-min period of rest, the same sequence of control and LBNP was repeated at −20 mmHg and then at the −30-mmHg levels of LBNP.

LBNP was produced by a commercial vacuum cleaner with the desired level of suction attained by altering input voltage to the vacuum cleaner with a rheostat. The actual level of negative pressure in the tank was measured continuously using a pressure transducer (Statham P50) and was both monitored on an oscilloscope and recorded on a Gould ES1000 recorder.

Experimental Design

All of the experimental procedures described above were performed in three sessions on separate days as follows: *session 1*, $\dot{V}O_{2\,max}$ and body fatness; *session 2*, HR variability and arterial baroreflex control of HR; and *session 3*, baroreflex control of FVR. All experimental procedures were performed on all 19 subjects before and immediately after the respective intervention periods (see below). Exercise group subjects maintained their final training levels throughout their second round of testing. However, no training session was performed before an experimental procedure on any day.

Interventions

Endurance exercise training. Subjects in the exercise group underwent a prolonged program of physical training [30 ± 1 (SE) wk, range 25–36]. During the initial 14 ± 1 wk of training, subjects walked/jogged 3.0 ± 0.1 days/wk for 33 ± 1 min/day at 68 ± 2% of their heart rate reserve [HRR; calculated as HRR = HR at rest + 0.68 (HR during maximal exercise − HR at rest)]. The level of exercise was progressively increased so that during the final 16 ± 1 wk of training subjects jogged an average of 3.5 ± 0.1 days/wk for 43 ± 1 min/day at 81 ± 2% of their HRR. The intensity of the exercise was assessed by monitoring HR during each training session. The amount and intensity of exercise performed in each session was recorded and used to document compliance with the individually prescribed programs.

Unchanged physical activity. After completing the initial round of testing, the subjects in the control group maintained their normal levels of physical activity over a period of time similar to the intervention period for the exercise group subjects (38 ± 2 wk, range 34–49 wk).

Data Analysis

Baroreflex control of FVR. Mean arterial blood pressure (MAP) was calculated from the sphygmomanometry-determined SBP and diastolic blood pressure (DBP) by the formula: MAP = DBP + 1/3 (SBP − DBP). PP was calculated by subtracting the DBP from the SBP. FVR was calculated from the corresponding values for MAP and forearm blood flow (FBF) by the formula: FVR = MAP ÷ FBF. FBF and FVR were expressed in ml·min^{-1}·100 ml^{-1} and in arbitrary units, respectively.

FBF during the control and LBNP periods was determined for each minute by averaging the four flow values. FVR values during each 2-min level of LBNP were calculated for each of the minutes separately and as an average for both minutes. The 2 min of control were averaged for all variables, and this value was used to calculate individual changes from base line during LBNP.

Data for the −30 mmHg LBNP trial were not obtained on one exercise group subject during the initial round of testing because of equipment failure. Thus, group data for this level of LBNP are based on 10 subjects.

Statistics. Changes in a dependent variable from control during LBNP and differences in a dependent variable or in the magnitude of the response of that variable before vs. after the intervention periods were assessed using an analysis of variance for repeated measures designs. Post hoc comparisons were made using a Fisher's least significant difference analysis. Differences of $P < 0.05$ were considered to be significant for all statistical analyses. All data are presented as means ± SE.

RESULTS

$\dot{V}O_{2\,max}$ and body composition. $\dot{V}O_{2\,max}$ increased 25% (ml·kg^{-1}·min^{-1}) and 30% (l/min) in the exercise group in response to physical training (both $P < 0.05$, Fig. 1). HR measured during maximal exercise was slightly, but significantly, reduced after training (174 ± 3 vs. 169 ± 3 beats/min, $P < 0.05$). Body weight (81.2 ± 3.5 vs. 78.7 ± 4.0 kg) and body fat (23.8 ± 1.3 vs. 20.9 ± 1.3%) were also decreased after training (both $P < 0.05$). In contrast, $\dot{V}O_{2\,max}$ (2.60 ± 0.19 vs. 2.62 ± 0.20 l/min and 31.0 ± 1.7 vs. 30.8 ± 1.8 ml·kg^{-1}·min^{-1}, initial vs. 6 mo), maximal exercise HR (175 ± 3 vs. 177 ± 4 beats/min), body weight (84.0 ± 4.4 vs. 85.2 ± 4.7 kg), and body fat (23.1 ± 1.6 vs. 22.8 ± 1.5%) were unchanged over the same period of time in the control group.

HR and its variability at rest. HR at rest was slightly, but consistently, lower (mean change = 5 beats/min; $P < 0.05$) after training in the exercise group (Fig. 2). HR variability, when expressed as the difference between the minimum and maximum R-R intervals, was unchanged after training in the group as a whole ($P = 0.38$). This index of cardiac vagal tone was higher in seven subjects and lower in four subjects after vs. before training. However, when expressed as the standard deviation of the R-R intervals, HR variability was increased slightly (15%), but significantly ($P = 0.01$), after training. This variable was higher in eight subjects, the same in one subject, and lower in two subjects after compared with before training.

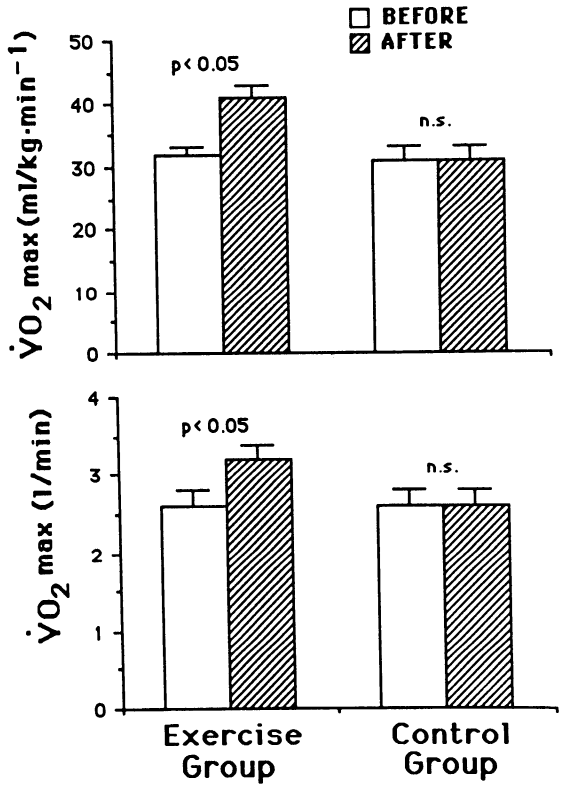

FIG. 1. $\dot{V}O_{2\,max}$ before and after respective intervention periods for exercise and control groups. Values are expressed both corrected for body weight (ml·kg^{-1}·min^{-1}, *top*) and uncorrected (l/min, *bottom*). $\dot{V}O_{2\,max}$ was increased significantly after training in exercise group, whereas no change occurred over a similar period of time in controls.

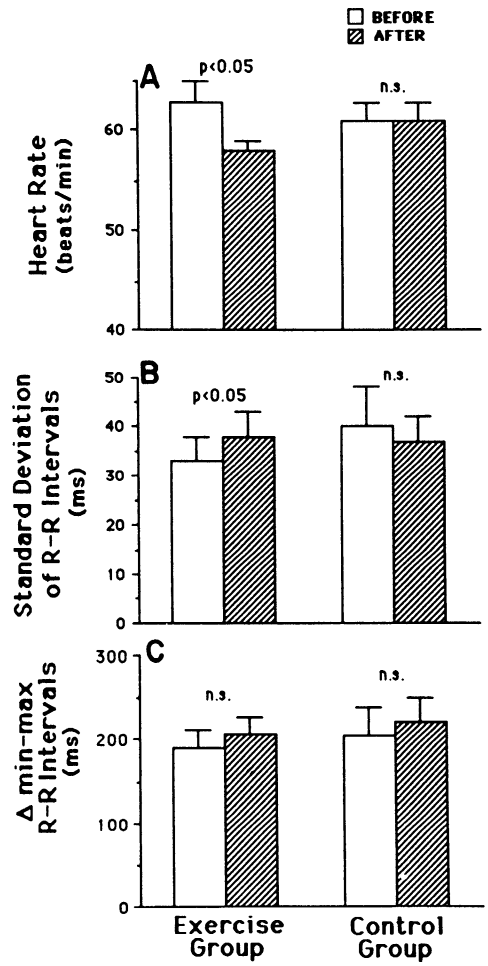

FIG. 2. Heart rate (*A*) and its variability at rest before and after respective intervention periods for exercise and control groups. Heart rate variability was assessed both as standard deviation of R-R intervals (*B*) and as difference between minimum and maximum R-R intervals (*C*). Heart rate was slightly but significantly lower after training in exercise group; however, heart rate variability was increased only when expressed in former manner. There was no change in heart rate or in either index of its variability over time in control group. ms, milliseconds; min, minimum; max, maximum.

HR at rest and its variability did not change over a similar period of time in the control subjects.

Baroreflex control of HR. R-R INTERVAL RESPONSES TO NECK SUCTION AND PRESSURE. Neck suction and pressure produced stimulus intensity-dependent increases and decreases in the R-R interval, respectively, in both groups of subjects (Fig. 3). The magnitudes of the changes induced by neck pressure were similar in the two groups, although the responses to neck suction were somewhat smaller in the controls. There were no significant changes in the R-R interval responses to any level of suction or pressure after training in the exercise group. The R-R interval responses to neck suction and pressure also were unchanged over time in the controls.

HR responses to LBNP. There were no significant differences in the ΔHR/ΔSBP or ΔHR/ΔPP during either minute of LBNP before vs. after training in the exercise group (Table 1). For example, after training ΔHR/ΔSBP for each minute of the 2-min LBNP period was higher in five subjects and lower in the other five subjects compared with before training. The ratios also were not different over time in the control subjects.

Baroreflex control of FVR. In both groups of subjects, FVR increased and FBF decreased from control levels in response to LBNP; the magnitudes of the changes were directly related to the stimulus intensity (Figs. 4 and 5, Table 2). MAP did not change from control during any level of LBNP in either group (Table 3). In general, PP and HR did not change from control levels during LBNP at −10 and −20 mmHg; however, PP decreased (7–11 mmHg; $P < 0.05$) and HR increased (6–10 beats/min; $P < 0.05$) in both groups of subjects during LBNP at −30 mmHg.

The magnitudes of the increases in FVR in response to LBNP at −10, −20, and −30 mmHg were reduced by 59 ($P < 0.05$), 38 ($P = 0.07$), and 39% ($P < 0.05$), respectively, in the exercise group after physical training when the FVR responses were assessed using the average values over the entire 2-min period of LBNP at each stimulus level (Fig. 4). Very similar training-related decreases in the FVR responses to LBNP were observed when percent changes in FVR from control were used rather than changes in absolute units (data not shown). These training-induced reductions in the forearm vasoconstrictor responses to LBNP were not associated with either changes in control FVR (26.9 ± 2.3 vs. 25.3 ± 2.4, 26.3 ± 2.3 vs. 25.1 ± 2.7, and 26.4 ± 2.2 vs. 24.3 ± 2.8 U, before vs. after training at −10, −20, and −30 mmHg LBNP, respectively, all NS) or changes in the blood pressure responses to LBNP (Table 3). When the FVR responses were assessed for each minute of LBNP (Fig.

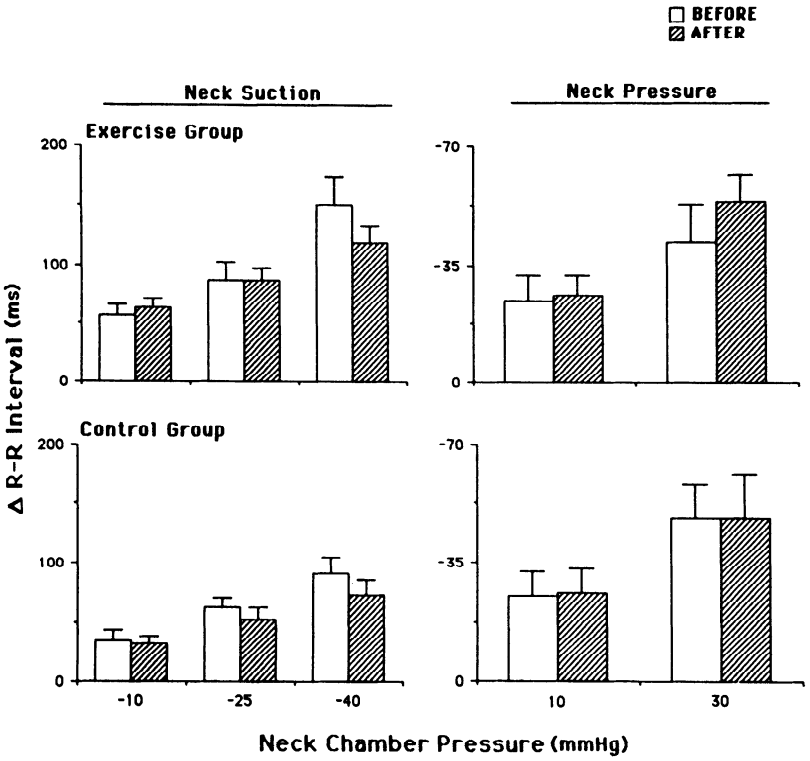

FIG. 3. Changes in R-R interval in response to neck suction (*left*) and pressure (*right*) before and after respective intervention periods for exercise (*top*) and control (*bottom*) groups. There were no significant changes in responses during any condition in either group after intervention periods.

TABLE 1. *Ratios of average change in HR to average change in SBP or PP during LBNP for 2 min at −30 mmHg*

Group	ΔHR/ΔSBP				ΔHR/ΔPP			
	Before		After		Before		After	
	Min 1	*Min 2*	*Min 1*	*Min 2*	*Min 1*	*Min 2*	*Min 1*	*Min 2*
Exercise	0.64±0.37	−0.76±1.34	0.24±0.22	0.37±0.17	0.63±0.20	1.06±0.27	0.53±0.48	1.22±0.77
Control	0.35±0.17	0.89±0.26	0.27±0.39	1.51±0.22	0.24±0.14	0.76±0.24	0.20±0.38	1.18±0.15

Values are means ± SE. HR in beats/min; SBP in mmHg; PP in mmHg. There were no significant differences in either ratio during either minute of LBNP before vs. after an intervention in either group of subjects. Note that pressure values used to calculate ratios were expressed as positive numbers; thus, in general the ratios are positive.

5), it was evident that the attenuation in the 2-min average responses were primarily the result of a markedly blunted vasoconstriction during the 1st min of LBNP at each stimulus level (63, 53, and 53% attenuation at the −10, −20, and −30 mmHg levels, respectively, all $P < 0.05$). The magnitudes of the increases in FVR during the 2nd min of LBNP were not significantly different at any stimulus level in the exercise group before vs. after training.

No changes in the FVR, FBF, MAP, PP, or HR responses to any level of LBNP were observed in the control subjects after their intervention period.

DISCUSSION

In the present study, we reinvestigated the question of whether regularly performed endurance exercise induces adaptations in the ANS that alter cardiovascular variables at rest and/or baroreflex circulatory control. We attempted to answer this question more definitively than in most previous investigations by studying the same subjects before and after a vigorous program of exercise. Our findings provide experimental support for at least four major conclusions regarding this issue. Our results indicate that in healthy, previously sedentary, middle-aged and older men strenuous endurance training *1*) elicits a marked increase in maximal exercise capacity and a slight, but consistent, reduction in HR at rest, *2*) may increase cardiac vagal tone at rest as assessed noninvasively by HR variability, *3*) does not alter arterial baroreflex control of HR at rest, and *4*) results in a transient attenuation of the forearm vasoconstrictor response to reductions in baroreflex sympathoinhibition.

Maximal exercise capacity. In the present study, we used the magnitude of the training-induced increase in $\dot{V}O_{2\,max}$ as an indication of the adequacy of the exercise stimulus for producing systemic cardiovascular adaptations. We observed substantial increases in $\dot{V}O_{2\,max}$ in our exercise group subjects after training; the magnitude of change was as great or greater than the largest increases reported previously in healthy subjects of similar age (9, 12, 26). In contrast, $\dot{V}O_{2\,max}$ was unchanged over a similar period of time in the nonexercising controls.

These findings are important in at least three ways. First, they confirm our recent observation (26) that vigorous, prolonged endurance training elicits marked increases in aerobic capacity in this population and pro-

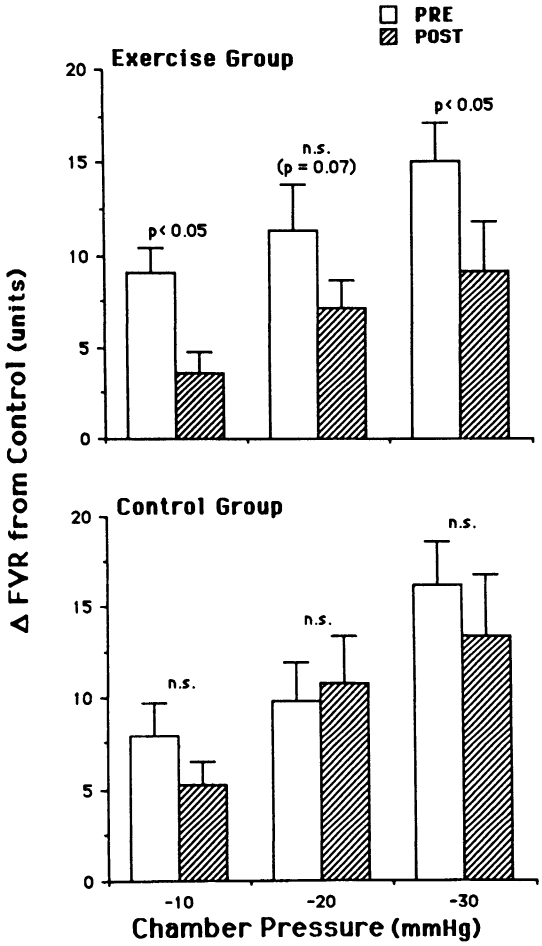

FIG. 4. Changes in FVR from control during 3 levels of LBNP before and after respective intervention periods for exercise (*top*) and control (*bottom*) groups. Responses were calculated using average FVR over entire 2-min period of LBNP at each level. Notations above each set of bars represent statistical comparisons for responses before vs. after intervention periods. Note that magnitude of reflex increase in FVR during all levels of LBNP was attenuated after training in exercise group ($P < 0.05$ for −10- and −30-mmHg levels and $P < 0.08$ for −20-mmHg level), whereas no significant changes occurred over the same period of time in control group.

vides support for the postulate that the ability to adapt physiologically to this chronically applied stimulus is not compromised in healthy, middle-aged and older men. Second, our findings demonstrate that a striking, systemic cardiovascular adaptation occurred in response to the exercise program. Thus, if changes in the ANS do result from endurance training, our stimulus should have been adequate to evoke these adaptations. Finally, the lack of change in $\dot{V}O_{2\,max}$ over time in the control subjects indicates that the increases in maximal exercise capacity observed in the exercise group were the result of physical training per se, rather than some nonexercise factor associated with study participation.

HR at rest and its variability. In the present study, we found a small but consistent reduction in HR at rest after training in the exercise group subjects. The magnitude of the change was similar to that reported in several previous investigations in which the same subjects were studied before and after training (9, 25, 36). No change in HR was observed over time in the control subjects.

To determine whether the training-induced bradycardia was mediated, at least in part, by an increase in cardiac vagal tone, we assessed the variability in HR (respiratory sinus arrhythmia) before and after the exercise program. An increase in HR variability after training would be suggestive of a higher level of vagal tone and vice versa. We found that one of our expressions of HR variability, i.e., the standard deviation of the R-R intervals, was higher after training in 8 of the 11 exercise group subjects ($P < 0.05$ for the group as a whole). However, our other commonly used expression of HR variability, i.e., the minimum to maximum R-R interval difference, was not different after vs. before training in the exercise group as a whole, although levels were increased in 7 of the 11 individual subjects. Taken together, these results suggest that cardiac vagal tone may have been higher after training in the majority of our subjects.

Although it is commonly assumed and frequently stated (cf. 25) that cardiac vagal tone is increased at rest in the endurance-trained state, there is very little experimental support for this concept in young humans and in laboratory animals. In a preliminary report, Barney et al. (1) found that HR variability at rest was greater in endurance-trained vs. untrained subjects. However, recent findings from our laboratory (23) and those of Maciel et al. (17) indicate that HR variability at rest is similar in highly trained endurance athletes and their untrained peers, in spite of a markedly lower HR in the athletes. Maciel et al. (17) also found that in previously sedentary males 10 wk of aerobic exercise training elicited an increase in peak O_2 uptake and an 11 beat/min decrease in HR at rest but did not alter HR variability. The results of these latter studies using HR variability to assess cardiac vagal tone are supported by findings of similar or even smaller increases in HR in endurance-trained vs. untrained humans and rats on removal of cardiac vagal influences with atropine (14, 16, 22, 35). Thus, the vast majority of the experimental evidence to date fails to support the view that endurance exercise training augments cardiac vagal tone at rest in young subjects. Instead, these results and the findings of others (15, 21, 34) indicate that the bradycardia often observed in the endurance-trained state is mediated by decreases in cardiac sympathetic tone and/or by nonautonomic mechanisms that lower the "intrinsic" HR.

The apparent discrepancy between the present findings and those discussed above may be the result of differences in the ages of the subjects studied. It is well documented that cardiac vagal tone at rest decreases with advancing age (27) and, therefore, may have been considerably lower before training in the middle-aged and older men studied here compared with the young subjects studied previously. This postulate is supported by the fact that HR variability was substantially lower in the subjects of the present study compared with the levels we recently reported in young men using the same methodology (23). Based on this assumption, interventions such as regular exercise would be more likely to evoke increases in cardiac vagal tone in older persons or in other populations with low base-line levels than in young healthy subjects with high initial levels.

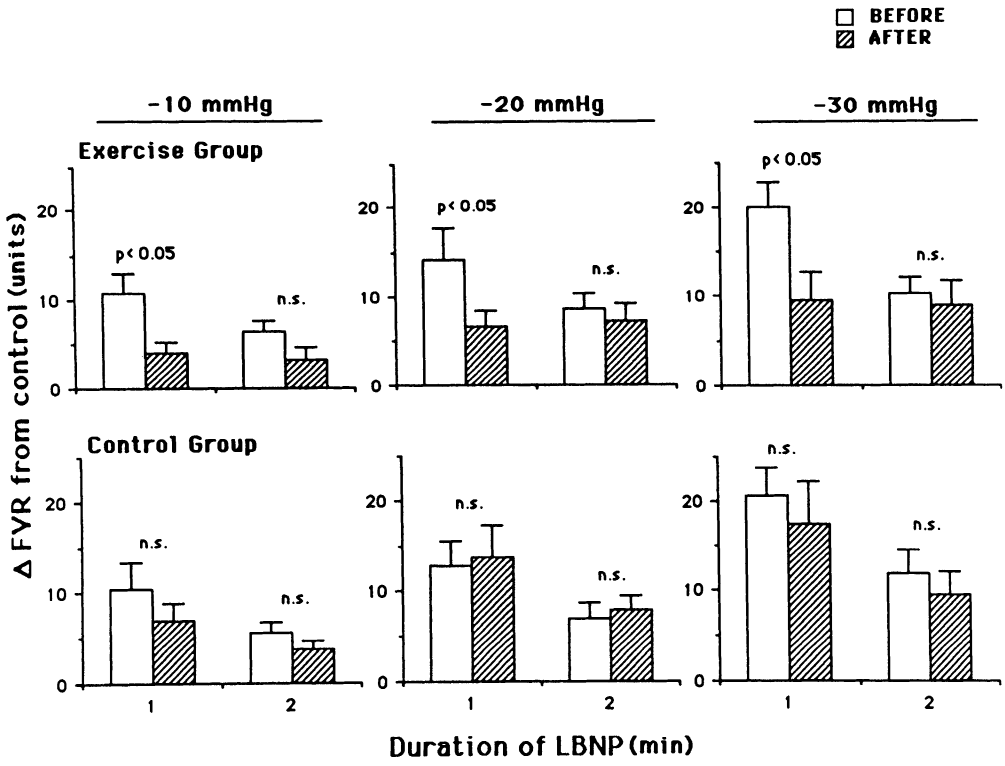

FIG. 5. Changes in FVR from control during each minute of LBNP before and after respective intervention periods for exercise (*top*) and control (*bottom*) groups. Note that training-induced attenuation in FVR response to LBNP depicted in Fig. 4 is primarily the result of a markedly blunted vasoconstrictor response during 1st min of stimulus.

TABLE 2. *FBF and HR before and during LBNP*

	−10 mmHg LBNP			−20 mmHg LBNP			−30 mmHg LBNP		
	Control	*Min 1*	*Min 2*	Control	*Min 1*	*Min 2*	Control	*Min 1*	*Min 2*
				FBF, ml·min^{-1}·100 ml^{-1}					
Exercise									
Before	3.9±0.4	2.7±0.2*	3.0±0.2*	3.9±0.3	2.5±0.2*	2.8±0.2*	3.9±0.4	2.2±0.2*	2.7±0.2*
After	4.1±0.4	3.4±0.3*†	3.6±0.3	4.2±0.4	3.2±0.3*†	3.2±0.3	4.4±0.5	3.0±0.3*†	3.1±0.3*
Control									
Before	3.1±0.4	2.5±0.4*	2.6±0.3*	3.3±0.4	2.3±0.3*	2.5±0.3*	3.4±0.3	2.0±0.3*	2.4±0.3*
After	3.8±0.4†	3.0±0.4*†	3.3±0.4*†	3.8±0.4	2.6±0.4*	3.0±0.3*	4.2±0.5†	2.7±0.5*†	3.1±0.5*
				HR, beats/min					
Exercise									
Before	66±2	65±2	65±2	64±1	66±2	67±2	64±2	68±2	70±2*
After	60±1†	59±2†	59±1†	59±1†	60±2†	62±2	61±2	65±3	69±3*
Control									
Before	61±1	60±1	62±1	60±1	61±2	65±2*	61±1	64±2	68±2*
After	61±2	61±2	63±2	60±2	63±2	66±3*	61±2	67±2*	71±3*

Values are means ± SE. * $P < 0.05$ vs. control value for same trial. † $P < 0.05$ vs. before value for same condition.

If, as our present findings suggest, cardiac vagal tone is increased by regular exercise in middle-aged and older men, this could have important clinical ramifications. The prevalence of coronary heart disease increases with advancing age and is especially high in the age range of the men studied here (3). Since high cardiac vagal tone may protect against certain life-threatening consequences of the atherosclerotic process (4), our findings may provide further rationale for use of regular aerobic exercise in the treatment of patients with coronary heart disease.

Arterial baroreflex control of HR. In the present study, we measured both the magnitudes of the changes in R-R interval in response to neck suction and pressure and the degree of tachycardia during LBNP-induced reductions in SBP and PP to assess arterial baroreflex control of HR. We failed to observe any change in chronotropic responsiveness after training in our exercise group subjects with either of these experimental approaches. To our knowledge, these are the only data available on carotid baroreflex responsiveness before and after physical training in the same subjects. However, the lack of change in the tachycardic response to LBNP observed in the present study is consistent with the recent findings of Vroman et al. (36), who reported that ΔHR/ΔSBP during LBNP at −40 mmHg was very similar before vs. after endurance training in a group of young, healthy subjects. As in our study, large increases in $\dot{V}O_{2\,max}$ occurred in response to training, indicating that arterial baroreflex responsiveness was unchanged in spite of the fact that a marked systemic cardiovascular adaptation had been demonstrated.

TABLE 3. *MAP and PP before and during LBNP at −10, −20, and −30 mmHg for 2 min each*

	−10 mmHg LBNP			−20 mmHg LBNP			−30 mmHg LBNP		
	Control	*Min 1*	*Min 2*	Control	*Min 1*	*Min 2*	Control	*Min 1*	*Min 2*
				MAP, mmHg					
Exercise									
Before	96±1	94±1	96±1	95±1	94±1	93±1	96±1	94±2	95±1
After	94±3	94±3	94±3	95±3	94±3	94±3	95±3	92±3	92±3
Control									
Before	94±2	94±2	93±2	95±1	94±2	93±2	96±2	93±2	94±2
After	93±2	92±2	91±1	93±2	91±2	93±2	93±2	91±2	92±2
				PP, mmHg					
Exercise									
Before	44±3	41±3	42±4	43±3	39±3	40±4	44±3	37±3*	37±4*
After	50±4†	46±3†	48±4†	49±4†	44±4	45±5	49±4	40±5*	40±5*
Control									
Before	43±2	41±2	39±2	43±2	40±2	38±2	43±2	35±2*	32±2*
After	44±3	45±4	45±4	45±3	42±4	37±3*	44±3	37±4*	36±3*

Values are means ± SE. * $P < 0.05$ vs. control value for same trial. † $P < 0.05$ vs. before value for same condition. There were no significant changes in MAP either from control to LBNP or before vs. after the 6-mo interventions for either group.

The results of studies that have compared various measures of arterial baroreflex control of HR in groups of endurance-trained vs. untrained subjects have been surprisingly inconsistent. The magnitude of the increase in HR (decrease in R-R interval) in response to either hypotensive levels of LBNP (10, 30, 32, 36) or neck pressure (1, 23, 33) has been reported to be greater (1), not different (10, 23, 36), and attenuated (30, 32, 33) in endurance-trained vs. untrained subjects. Similarly, the magnitude of the decrease in HR (increase in R-R interval) in response to either neck suction (1, 23, 33) or to increases in blood pressure produced by phenylephrine infusion (30) also have been reported to be greater (1), not different (33), and smaller (30) in endurance-trained subjects. In support of the latter finding, we recently observed a twofold attenuation in the slope of the R-R interval changes during graded neck suction in young, highly trained cross-country runners when compared with their untrained peers (23).

Thus, the results of the present study and the recent findings of Vroman et al. (36) indicate that in previously untrained, healthy subjects endurance exercise training does not evoke changes in chronotropic responsiveness to either carotid baroreflex stimulation or inhibition, or to nonspecific unloading of the arterial baroreceptors. Comparisons of groups of endurance-trained and untrained subjects have produced somewhat equivocal results, with the majority of studies performed to date reporting either unchanged or attenuated chronotropic responsiveness to both arterial baroreceptor stimulation and inhibition in the endurance-trained state.

Baroreflex control of limb VR. We assessed nonspecific baroreflex control of limb VR by measuring the magnitude of the increase in FVR in response to LBNP-induced reductions in tonic baroreceptor sympathoinhibition. We found that the vasoconstrictor response to each level of LBNP was markedly attenuated in the exercise group after training. In contrast, the FVR responses to LBNP were unchanged over time in our nonexercising control subjects.

The diminished vasoconstrictor response to LBNP after training could be explained by changes in the baroreflex per se or by nonbaroreflex adaptations. Concerning the latter, changes in base-line FVR or central venous pressure (a stimulus for cardiopulmonary baroreceptor discharge), differences in the magnitudes of the changes in central venous pressure or arterial pressure (arterial baroreceptor stimulus) from base line during LBNP, or changes in nonspecific reflex responsiveness or in vascular responsiveness to neurotransmitter release all could have contributed to the altered FVR responses to LBNP after training. Neither base-line FVR nor the changes in arterial pressure during LBNP were altered after training; thus, it is unlikely that these potential mechanisms were involved. Although we did not assess the other possible mechanisms in the current study, the results of previous investigations may provide some insight. For example, Jingu et al. (12) have recently reported that neither base-line central venous pressure nor the change from base line during LBNP at −10 and −40 mmHg was different before vs. after endurance training in a group of men and women aged 36–67 yr. In the same study, there was no change in nonspecific reflex responsiveness as judged by similar increases in FVR during a cold pressor test before vs. after training. Furthermore, vasoconstrictor responsiveness to α-adrenergic receptor stimulation has been reported to be increased (21), not different (2), and even decreased (37) in the endurance-trained state. Thus, although the findings of previous investigations in general fail to support these alternative hypotheses, we cannot state with certainty that the attenuated vasoconstrictor responses to LBNP observed in the present study were the result of autonomic adaptations that resulted in changes in baroreflex control of VR.

We also found that most of the diminished FVR response to LBNP after training was the result of changes in the initial minute (Fig. 5). Although complete time course data on FBF from the onset of LBNP are rare, the present findings and those of Rowell and colleagues

(24) indicate that the magnitude of the reflex decrease in blood flow (increase in FVR) is greatest during the 1st min. Although the mechanism responsible for this particular time course is unknown, it appears that in the current study the primary training-induced changes in the FVR response to LBNP occurred during this initial phase of the reflex adjustment.

Previous investigators have employed both cross-sectional comparisons of endurance-trained vs. untrained subjects and longitudinal comparisons of the same subjects before and after endurance training to address this question. Cross-sectional comparisons have reported both similar (10, 31) and attenuated (18) FVR responses to LBNP in endurance-trained vs. untrained subjects. During the preparation of this manuscript, the results of two studies employing longitudinal comparisons were reported. Vroman et al. (36) found a marked attenuation in the FVR response to a hypotensive level of LBNP (−40 mmHg), but an unchanged response to a nonhypotensive level (−10 mmHg), in a group of young subjects after endurance training. In striking contrast, Jingu and co-workers (12) observed a 2.5-fold augmentation in the FVR response to LBNP at −10 mmHg after endurance training in a group of primarily middle-aged men and women. In their study, the increase in FVR in response to LBNP at −40 mmHg was approximately twofold greater after vs. before training, although the difference did not attain statistical significance.

Thus, as with the available data concerning the influence of endurance exercise training on arterial baroreflex control of HR, the information on possible training-associated changes in baroreflex control of limb VR is somewhat inconsistent. The present results and those of Vroman et al. (36) provide support for the hypothesis that endurance training results in a diminished FVR to acute reductions in baroreflex sympathoinhibition in previously sedentary, healthy subjects; however, the findings of Jingu et al. (12) suggest that the opposite is true. The results of cross-sectional studies are equivocal with reports of both similar (10, 31) and attenuated (18) FVR responses in endurance-trained vs. untrained subjects. Taken together, the majority of the available evidence would suggest that limb reflex vasoconstrictor responsiveness may be blunted in the endurance-trained state. However, additional studies of baroreflex circulatory control in the same subjects before and after training are needed if this question is to be more definitely answered.

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