Endurance exercise training has a minimal effect on resting heart rate: the HERITAGE study

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ABSTRACT

WILMORE, J. H., P. R. STANFORTH, J. GAGNON, A. S. LEON, D. C. RAO, J. S. SKINNER, and C. BOUCHARD. Endurance exercise training has a minimal effect on resting heart rate: the HERITAGE study. *Med. Sci. Sports Exerc.*, Vol. 28, No. 7, pp. 829–835, 1996. This study determined the effects of a 20-wk endurance training program (The HERITAGE Family Study) on resting heart rate (HR_{rest}). HR_{rest} was obtained on a sample of 26 men and 21 women during sleep; during resting metabolic rate and resting blood pressure measurement periods in the early morning following a 12-h fast and 24-h post-exercise; and at rest prior to a maximal bout of exercise. Following training, the subjects exhibited a 16.0 ± 9.4% (mean ± SD) increase in $\dot{V}O_{2max}$ ($P < 0.05$), but the HR_{rest} for each of the resting conditions was decreased by only 1.9 to 3.4 bpm ($P < 0.05$), or an average across the three conditions of 2.7 bpm. In a larger sample of 253 HERITAGE subjects, HR_{rest} obtained only at the time of the resting blood pressure measurement decreased by only 2.6 bpm, while $\dot{V}O_{2max}$ increased 17.7 ± 10.0%. It is concluded that there is a significant, but small, decrease in resting heart rate as a result of 20 wk of moderate- to high-intensity endurance training; which suggests a minimal alteration in either, or both, intrinsic heart rate and autonomic control of HR_{rest}.

RESTING HEART RATE, AUTONOMIC CONTROL OF HEART RATE, INTRINSIC HEART RATE, ENDURANCE TRAINING

On the basis of scholarly reviews of the existing research literature, it has become widely accepted that resting heart rate (HR_{rest}), and heart rates during steady-state submaximal exercise at the same absolute rate of work, are substantially reduced consequent to aerobic exercise training (2,5,10,24,30,32,40). While the research literature is unequivocal regarding the attenuation of submaximal exercise heart rates with endurance training, the response of HR_{rest} is less clear. Part of the confusion concerning the HR_{rest} response might be the result of using cross-sectional comparisons, where highly trained endurance athletes, when compared with untrained individuals, generally have HR_{rest} values that are 15 to 30 bpm lower (32,45). In longitudinal studies, where untrained individuals have been endurance-trained for varying periods of time, the findings are mixed. Studies have clearly demonstrated reductions in HR_{rest} with endurance training in humans (6,8,14,33,44), horses (12,35), dogs (27), and rats (3,25,29,42). However, other studies have not found reductions in HR_{rest} following endurance training in humans (11,15,22,23,26,34), horses (4,12,41), and rats (42). An explanation for these differences in results is not immediately obvious, but could be related to the exercise training stimulus or the conditions under which the resting measurements were obtained.

Thus, the purpose of the present study was to determine the response of the HR_{rest} to a highly controlled 20-wk endurance training program, where resting measurements were obtained under three separate, but tightly controlled, conditions. This study is part of a larger multicenter clinical trial investigating possible genetic bases for the variability in the response of physiological measures, and cardiovascular disease and diabetes risk factors, to endurance exercise training—the HERITAGE Family Study. This study has been described in detail in a previous publication (7).

METHODS

The HERITAGE subject population is composed of families, including the natural father and mother, and at least two (African-American families) or three (Caucasian families) offspring 17 yr of age or older. Subjects for this study were recruited from the University of Texas at Austin HERITAGE Family Study Clinical Center. This site was selected from among the four HERITAGE clinical centers, since it was the primary site for a substudy on resting metabolic rate, in which highly controlled

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TABLE 1. Physical characteristics of the subject population.

Characteristics RMR Sub-Study	Men (N = 26)	Women (N = 21)	Total (N = 47)
Age (yr)	35.9 ± 15.9*	37.5 ± 14.7	36.6 ± 15.3
Height (cm)	178.1 ± 6.7	162.7 ± 5.9	171.2 ± 10.1
Weight (kg)	85.2 ± 15.3	71.4 ± 14.7	79.0 ± 16.6
Characteristics HERITAGE Study	**Men (N = 122)**	**Women (N = 131)**	**Total (N = 253)**
Age (yr)	37.3 ± 15.2	36.2 ± 14.3	36.7 ± 14.7
Height (cm)	177.5 ± 6.1	163.1 ± 6.4	170.1 ± 9.5
Weight (kg)	85.3 ± 16.2	68.7 ± 13.9	76.7 ± 17.2

* Mean ± SD.

measures of HR_{rest} were obtained. A total of 47 subjects (26 men, 21 women), between the ages of 17 and 63 yr, completed this substudy. Subject characteristics are presented in Table 1. Prior to acceptance for participation in the HERITAGE study, all subjects had to meet a set of inclusionary criteria (7) and pass a physician-administered physical examination, including a 12-lead electrocardiogram (ECG), during a maximal exercise test (7). The study protocol had been previously approved by The University of Texas at Austin Institutional Review Board, and informed consent was obtained from each subject. For those under 18 yr of age, parental consent was also obtained.

HR_{rest} measurements were made under three highly controlled conditions, and under conditions used in previous studies. For the resting metabolic rate (RMR) protocol, heart rate was measured continuously from the time the subject went to bed the evening prior to the RMR study to the end of the RMR measurement period the next morning. This provided a well-controlled measure of the lowest heart rate attained for a period of at least 1 h during sleep (HR_{rest}-sleep) as well as a measure of the heart rate during the RMR test (HR_{rest}-RMR). Diet and prior exercise were controlled in that the subjects had fasted, except for water, for 12 h prior to the RMR measurement, and had not exercised for the preceding 24 h.

The RMR test period included a 30-min rest period and a 30-min heart rate and RMR measurement period, with the subject seated in a semirecumbent position in a comfortable recliner chair. The test room was dimly lit and isolated from noise. Heart rate during both the sleep and RMR test periods was measured using a Polar Vantage XL heart rate monitor (Polar USA, Inc., Montvale, NJ) with a memory transfer system. Sleep and RMR heart rates were downloaded from the monitor to an IBM PC at the end of the RMR test period. This technique for monitoring heart rate during both sleep and RMR tests has been described in detail in a previous publication (43). All RMR testing was completed prior to 10:00 a.m., and the RMR test was administered on two different days, both pre- and post-training. The data presented here represent the mean of the two test days both pre- and post-training.

HR_{rest} was also obtained during the measurement of resting blood pressure according to the HERITAGE protocol (7). This measurement period employed the same recliner chair and test room conditions specified above for the RMR measurement. Blood pressure was measured using a Colin STBP-780 automated blood pressure device (San Antonio, TX). Heart rate was monitored by ECG continuously during the test, and a minimum of four blood pressure readings were taken following a 5-min rest period, with 2-min intervals between readings. HR_{rest} was recorded with each blood pressure measurement, and the values averaged. This procedure was conducted on two separate days both pre- and post-training, and the data presented here represent the mean of the two test days, both pre- and post-training, i.e., HR_{rest}-BP.

Finally, in an effort to replicate the conditions under which resting measurements have been taken in many previous studies, heart rate was also obtained prior to a maximal exercise test (HR_{rest}-Pre-Ex) on a cycle ergometer (SensorMedics Ergo-Metric 800S ergometer). The subject was seated in a chair for approximately 5 min before resting blood pressure and heart rate were obtained just prior to the exercise test. Prior to and during the exercise test, heart rate was obtained by ECG (SensorMedics, Yorba Linda, CA).

The subjects completed two additional submaximal tests on the cycle ergometer, both pre- and post-training, during which steady-state heart rates were obtained at a power output of 50 W. The data presented here represent the mean of the two test days both pre- and post-training.

During the maximal exercise test, metabolic rate was measured using a SensorMedics 2900 metabolic measurement cart (SensorMedics, Yorba Linda, CA). $\dot{V}O_{2max}$ was defined as the mean of the highest $\dot{V}O_2$ values determined on each of the maximal tests, or the highest of the two values if they differed by more than 5% of the highest value.

Following the pre-training testing period, the subjects participated in a 20-wk exercise training program using standardized cycle ergometers. Details of the training program have been reported elsewhere (7). Briefly, the subjects trained an average of three times per week, starting at a heart rate representing an intensity of 55% of their initial $\dot{V}O_{2max}$ for 30 min during the first 2 wk of training, and building up to 75% of their initial $\dot{V}O_{2max}$ for 50 min during the last 6 wk. Heart rate was controlled during all training sessions by a computerized cycle ergometer system, which varied ergometer resistance to maintain the target heart rate. Exercise sessions were supervised by trained exercise leaders.

All data were analyzed using the SAS statistical package. Except where noted, data are expressed as mean ± SD. Intraclass correlations were computed to estimate reproducibility of each of the HR_{rest} measurements (i.e., HR_{rest}-sleep, HR_{rest}-RMR, and HR_{rest}-BP), which were taken on two different days, both pre- and post-training,

TABLE 2. Changes in measures of sleeping, resting, pre-exercise, and exercise heart rate and maximal oxygen uptake (N = 47).

Variable	Pre-Training	Post-Training	Δ[a]	Δ, %[b]
	Mean ± SD	Mean ± SD	Mean	Mean
Heart rate (bpm during sleep)	59.8 ± 7.8	57.9 ± 8.1	−1.9†	−3.2
Heart rate (bpm during RMR)	60.1 ± 8.0	57.4 ± 7.1	−2.7†	−4.5
Heart rate (bpm during resting BP)	61.0 ± 9.1	57.6 ± 8.9	−3.4†	−5.3
Heart rate (bpm pre-exercise)	75.1 ± 12.0	74.3 ± 10.4	−0.8	−1.1
Heart rate (bpm 50 watts exercise)	126.6 ± 20.4	110.4 ± 14.7	−16.2†	−12.8
$\dot{V}O_{2max}$ (ml · min^{-1})	2440 ± 789	2831 ± 830	391†	+16.0

a Difference between pre- and post-training measures (post - pre).
b Percent change [(post - pre)/pre].
† Significantly different from the pre-training value, $P \leq 0.05$.

as well as across these three measurement conditions, using the (1,1) model of Shrout and Fleiss (36). The general linear models (PROC GLM) procedure for repeated measures analysis of variance was used to test the significance of changes from pre- to post-training for each of the HR_{rest} conditions; HR_{rest}-sleep, HR_{rest}-RMR, and HR_{rest}-BP, using Tukey's Studentized Range (HSD) Test to detect individual paired data (i.e., pre- to post-training) significant differences. The significance of pre- to post-training differences in the HR_{rest}-Pre-Ex, the steady-state heart rate at 50 W (HR_{50W}), and $\dot{V}O_{2max}$ were determined by a simple t-test. A statistical power analysis to determine sample size was conducted according to the method of Kraemer and Thiemann (19), using an expected decrease in HR_{rest} of 5 bpm (N = 26 for a power > 0.80). Statistical significance was set at the $P \leq 0.05$ level.

RESULTS

The reproducibility of HR_{rest} measurements was determined across two separate days, both pre-training and post-training. The intraclass correlation coefficients were as follows: HR_{rest}-sleep, R = 0.94 and 0.80; for HR_{rest}-RMR, R = 0.88 and 0.83; and for HR_{rest}-BP, R = 0.88 and 0.88, pre- and post-training, respectively.

The pre- and post-training HR_{rest} under each of the conditions described in the methods section, the HR_{50W}, and $\dot{V}O_{2max}$ values are presented in Table 2. First, $\dot{V}O_{2max}$ was increased by an average of 16.0 ± 9.4% (mean ± SD), confirming the attainment of a significant endurance training effect. This was further confirmed by a 12.8 ± 8.7% decline in the steady-state heart rate response to a constant power output of 50 W. HR_{rest} declined significantly for each of the measures taken under highly controlled conditions, i.e., HR_{rest}-sleep, HR_{rest}-RMR, and HR_{rest}-BP. However, these changes were small, with mean differences ranging between 1.9 ± 5.4 and 3.4 ± 4.9 bpm, and an average difference across the three highly controlled conditions of 2.7 bpm. These mean HR_{rest} pre- to post-training changes remained statistically significant after controlling for gender, age, and gender-by-age interaction effects. The HR_{rest}-Pre-Ex was unchanged following the training period.

There was very good agreement among the HR_{rest} values obtained during sleep, during the RMR test period, and during the resting blood pressure test period. Mean HR_{rest} across these three conditions agreed within 1.5 bpm pre-training, and within 0.5 bpm post-training. Furthermore, the intraclass correlations among these three conditions were R = 0.84 pre-training and R = 0.84 post-training.

Since there was such good agreement for HR_{rest} across conditions, both pre- and post-training, a decision was made to include the pre-training and post-training HR_{rest} values obtained during the resting blood pressure protocol for the total HERITAGE sample completed at that time from all four Clinical Centers (Arizona State University, Laval University, the University of Minnesota, and The University of Texas at Austin). Thus, this sample included not only those at The University of Texas at Austin who had participated in the RMR study, but subjects from all four Clinical Centers who had completed the resting blood pressure protocol, but not the RMR protocol. This sample of 253 subjects had met the inclusion criteria established in the Methods section and had completed the 20-wk training program. See Table 1 for their physical characteristics. From this sample, the HR_{rest} obtained during the resting blood pressure test (mean of two test sessions pre-training and two test sessions post-training) was 65.0 ± 8.7 bpm pre-training and 62.4 ± 9.0 bpm post-training. The mean difference of 2.6 bpm ($P < 0.05$) was nearly identical to that of the 47 subjects in the RMR substudy. The mean increase in $\dot{V}O_{2max}$ for this secondary sample of 253 subjects was 17.7 ± 10.0%, similar to the 16.0 ± 9.4% increase noted previously for the 47 subjects in the RMR substudy.

Since the reduction in heart rate at rest (~2.7 bpm) was substantially less than the reduction in heart rate at a steady-state, constant power output of 50 W (16.2 bpm), a decision was made to analyze the heart rate response to increasing rates of work to determine if the magnitude of reduction in submaximal heart rate increased with increasing rates of work. With the initial maximal test protocol, the work rate was increased by 25 W every 2 min until exhaustion. The final heart rate at the end of each of these 25-W increments was recorded, and was presumed to approximate the steady-state heart rate for that rate of work. From the initial sample of 47 subjects, 19 of the 26 men completed work rates ≥200 W both pre- and post-training. Their mean heart rate values at rest and at 50, 100, 150, and 200 W, both pre- and post-training, are plotted in Figure 1. From this figure, it can be clearly seen that the reduction in heart rate consequent to endurance training increases in a linear fashion with increasing rates of work.

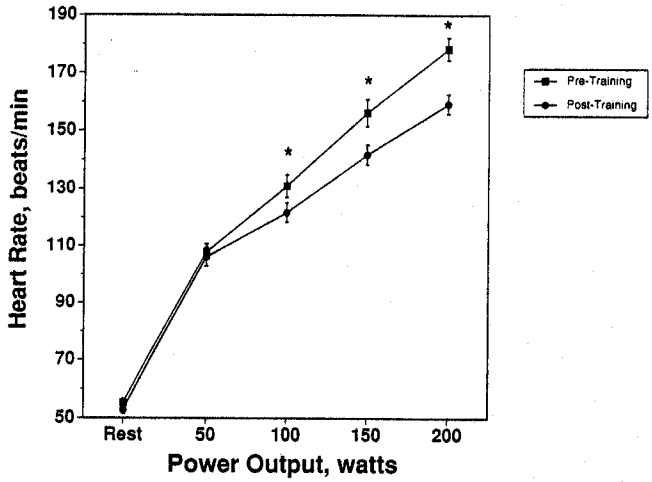

Figure 1—Resting and submaximal heart rates pre- and post-training in 19 men who completed the HERITAGE 20-wk endurance training protocol. Values are means ± SE. *$P = < 0.05$.

DISCUSSION

The major purpose of this study was to determine the effects of a 20-wk cardiorespiratory endurance training program on HR_{rest} as determined under highly controlled conditions. Following training, HR_{rest} obtained under the three most highly controlled conditions (i.e., HR_{rest}-sleep, HR_{rest}-RMR, and HR_{rest}-BP), decreased from 1.9 to 3.4 bpm (mean of 2.7 bpm) for the primary sample of 47 subjects. In the larger sample of 253 subjects undergoing the identical training program, with approximately the same increase in $\dot{V}O_{2max}$, HR_{rest} obtained during the resting blood pressure measurement period decreased by a similar amount (2.6 bpm). Thus, HR_{rest} declined significantly in both subject groups with endurance training, but the magnitude of change was small.

In most studies where decreases in HR_{rest} have been reported consequent to endurance training, the magnitude of change has been considerably larger. Blumenthal et al. (6) reported a decline of 11 bpm in 20 middle-aged men following a 12-wk training program. Maciel et al. (21) reported a similar decrease of 11 bpm in seven young men following 10 wk of endurance training. Byrd et al. (8) endurance-trained 13 middle-aged men over a period of 12 wk and reported a decrease in HR_{rest} of 10 bpm. In a study that included baseline measurements, a period of 20 d of bed rest, and a subsequent 2-month period of endurance training, Saltin et al. (31) reported mean supine HR_{rest} values of 63.0 bpm at baseline, 68.0 bpm following bed rest, and 50.8 bpm after training in five young men. Sitting heart rates under the exact same conditions were 76.4, 82.6, and 57.0 bpm, respectively. Shi et al. (35) reported a mean decrease in HR_{rest} of 9 bpm in eight young men who had endurance trained for a period of 8 months. This decrease was associated with an increase in vagal tone without change in the intrinsic heart rate.

Others have found more modest decreases in HR_{rest}. In his review of the literature up to 1973, Pollock (30) reported a mean HR_{rest} reduction of 6.6 bpm for 18 published endurance training studies. Spina et al. (38) studied six young men and six young women before and after 12 wk of endurance training. HR_{rest} (which was not reported but calculated by the present authors from the reported cardiac output and stroke volume values), declined by 6 bpm. This might not have been a significant decrease, as neither resting cardiac output nor stroke volume changed with training. Seals and Chase (33) reported only a 5 bpm decrease in HR_{rest} in 11 middle-aged and older men who had undergone endurance training for a period of 30 wk. This smaller decline with endurance training most likely is not an age-related phenomenon, as Spina et al. (39) found a decline in HR_{rest} of 10 bpm in older women who had endurance trained for 9–12 months.

Still others have found no change in HR_{rest} with endurance training. Sedgwick et al. (34) reported no change in HR_{rest} taken either at night during sleep or during the day at the time of the resting ECG following 12 wk of endurance training. They did report a small decrease of 4.5 bpm in mean heart rate averaged over the working day. Edwards (11) reported no change in HR_{rest} following a 4-wk endurance training program in 12 young women. Martin et al. (22) found no change in HR_{rest} in 12 middle-aged men and women who had completed a 12-wk intense swim training program. O'Connor et al. (26) found no change in the lowest heart rate during sleep in nine young men who had endurance-trained for 12 wk. Meredith et al. (23) found no change in HR_{rest} in either young or older subjects following a 12-wk endurance training program. Finally, Gossard et al. (15) reported no change in HR_{rest} in two groups of middle-aged men who endurance-trained at either low or high intensities.

In all of the studies cited above, the training programs met the established criteria for attaining a cardiorespiratory endurance training effect in previously sedentary subjects (1). Furthermore, the alterations in $\dot{V}O_{2max}$ consequent to training were similar to those reported in the present investigation, ranging from 8% to 27%, with the greater increases being associated with either or both longer training periods and higher intensities. The mean increase across these studies was 16%, identical to that of the present study.

There is no obvious explanation for the differences in results seen across these studies, with one possible exception. In most studies that have demonstrated either no change or a small decrease in HR_{rest} (11,22,26,33,34) the measurement of HR_{rest} was obtained under what could be considered ideal resting conditions, where the subject was asleep or maintained at quiet rest for at least 10–15 min with no distractions. In other studies, HR_{rest} has been obtained at rest just prior to an exercise test, just prior to or immediately after the insertion of a venous catheter, or

under other conditions that have not been well-controlled for obtaining true resting values. It is quite possible that at least part of the reduction in "resting" heart rate is the result of adjusting to the testing environment where "resting" conditions have not been as tightly controlled, although the data for the HR_{rest}-Pre-Ex from the present study (Table 2) would tend not to support this contention.

There has been considerable interest in those mechanisms potentially involved in the bradycardic response to endurance training. The mechanism may differ for changes at rest compared with submaximal exercise bradycardic responses. A reduction in the intrinsic heart rate has been proposed to explain the bradycardia at rest. Katona et al. (17) from their study of highly trained, world-class oarsmen compared with untrained controls, concluded that training-induced bradycardia at rest is the result of a reduction in the intrinsic cardiac rate, not to an increase in parasympathetic tone. These results were similar to those of Lewis et al. (20), who compared highly trained male cyclists and untrained men. Their results support what they refer to as a nonautonomic component (i.e., intrinsic cardiac rate) as the primary factor explaining training resting bradycardia. However, Ordway et al. (27) reported that cardiac-denervated dogs did not decrease their HR_{rest} with endurance training, while similarly trained, sham-operated dogs had a decrease in HR_{rest} from 64 to 51 bpm. They concluded that the bradycardia at rest resulting from endurance training must be controlled by the autonomic nervous system.

Kenney (18) assessed the degree of parasympathetic control over HR_{rest} using the variation in heart period during cardiopulmonary synchronization of respiration. In 21 healthy young subjects, he found that individuals with a higher $\dot{V}O_{2max}$ maintained a lower HR_{rest}, mainly via an increase in parasympathetic tone, as opposed to a decrease in sympathetic tone. Using heart rate variability as a marker of cardiac vagal tone, Seals and Chase (33) found increased heart rate variability at rest following a 30-wk endurance training program and concluded that the small reduction of 5 bpm that they observed was likely due to this increased cardiac vagal tone. And, as mentioned previously, Shi et al. (35) concluded that the reduction in HR_{rest} that they found consequent to endurance training was the result of an increased vagal tone, and not to a reduction in the intrinsic heart rate. However, Maciel et al. (21) were unable to find an increase in resting parasympathetic activity with 10 wk of endurance training.

Smith et al. (37) compared the resting parasympathetic and sympathetic influence, intrinsic heart rate, and resting autonomic balance in 10 endurance-trained and 10 untrained young men. Using a modeling approach, they concluded that the difference in HR_{rest} between these two groups could be explained by a decrease in intrinsic cardiac heart rate, an increase in parasympathetic autonomic control, and a slight decrease in sympathetic autonomic control.

From these studies, there is no clear and consistent conclusion that can be drawn regarding the potential mechanism for the resting bradycardia associated with endurance training. Part of the confusion in the literature can be attributed to at least two factors. First, assessment of the contribution of factors such as intrinsic heart rate and autonomic control are limited by the indirect techniques available to measure these variables in humans. Furthermore, the literature has been confounded by potential differences between the results of cross-sectional and longitudinal studies. With cross-sectional studies, which comprise a major portion of the existing literature, the magnitude of differences noted between highly trained and untrained populations are substantial (i.e., 15–30 bpm or greater). It is impossible to determine if these differences between disparate groups are due to the training stimulus or to inherent differences in the two populations being compared. Further, it is also possible that it might take years of endurance training to alter factors such as increased parasympathetic autonomic control and decreased intrinsic heart rate, while most studies have been less than 20 wk in duration.

One additional adaptation to exercise training known to affect cardiovascular responses to exercise must be discussed. It has been well established that endurance training results in an increase in total blood volume, largely as a result of an expanded plasma volume (13). Oscai et al. (28) reported a 18.3% increase in $\dot{V}O_{2max}$ and a 6% increase in total blood volume following a 16-wk endurance training program (47 total sessions, intensity not specified) in 14 previously sedentary men, 26–64 yr of age. This increase was due almost exclusively to a 6.4% increase in plasma volume. Convertino et al. (9) reported a 9% increase in total blood volume in 14 men (mean age of 36 yr) following 10 wk of cycle ergometer training, 30 min/d^{-1}, 4 d/wk^{-1}. This was associated with a 20% increase in $\dot{V}O_{2max}$ and a decrease in HR_{rest} from 63 to 57 bpm. Finally, Green et al. (16) reported a 13.8% increase in plasma volume associated with a 17.2% increase in $\dot{V}O_{2max}$ following 8 wk of cycle ergometer training, 2 h/d^{-1}, averaging 4 to 6 training sessions per week.

In these studies, the subject population, training mode, and training protocol were similar to those used in the present study. While not measured, the training protocol in the present study should have resulted in an increase in total blood volume, through plasma volume expansion. It is likely that an expanded plasma volume was a major contributor to the decrease in submaximal heart rate at the same absolute work rates post-training through its affect on stroke volume. Yet, the plasma volume expansion affect on stroke volume, if it occurred, was not a major factor at rest for reasons that are not immediately obvious. This apparent paradox deserves further study.

The results from the present study indicate that the changes in HR_{rest} with 20 wk of endurance training are much smaller than what is normally assumed (2,5,10,30,32,40), but are statistically significant. These results would suggest that there is little or no change in the intrinsic heart rate or in autonomic control of HR_{rest}. However, the results strongly suggest that with progression from rest to increasing intensities of exercise, there is a progressively greater decrease in the heart rate response following training. At least three controlling factors could be operating here; namely, an increase in plasma volume, a reduction in sympathetic autonomic control, and a reduction in the circulating catecholamines, since each work rate represents a reduction in the relative work rate (i.e., percentage of $\dot{V}O_{2max}$), and catecholamine levels are highly correlated to the relative intensity of exercise (45).

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