ORIGINAL PAPER

Medicina Sportiva Vol. 5 (EE1): E49-E58, 2001

HEART RATE VARIABILITY FOLLOWING 5 WEEKS OF DETRAINING IN COMPETITIVE SWIMMERS

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ABSTRACT

The purpose of this investigation was to evaluate HRV following a period of detraining. Therefore, six elite collegiate swimmers were examined for heart period (RR) and HRV (i.e., standard deviation of normal R-R intervals (SDNN), and normalized high- and low-frequency spectral power), and maximal oxygen consumption ($\dot{V}O_2$max) during incremental arm-ergometry. ECG data were collected for 5 minutes in the supine position under conditions of spontaneous and paced (0.20 Hz) breathing. The athletes were examined 2 weeks prior to the end of the season, at end season, and 1, 2, 3, and 5 weeks post-season. During the first 2 weeks of the post-season period the swimmers reduced their training duration from 20 to 10 hours per week, and during the 3rd through 5th weeks post-season the swimmers did not swim-train. $\dot{V}O_2$max was significantly reduced by the 2nd week of the post season (34.0 + 8.3 vs. 28.2 + 6.9 ml•kg^{-1}•min^{-1}, respectively) and did not change thereafter. RR intervals were 60-100 msec shorter at the 5th week of detraining than at the other observation periods (regardless of breathing condition), but HRV did not change across the observations. Paced breathing was consistently associated with shorter RR intervals and greater SDNN, but no differences in normalized units of low-and high-frequency power. The results of this investigation are consistent with differential effects of detraining on aerobic power and autonomic modulation of the heart, and provide further indirect evidence for the use of HRV as an index of autonomic modulation of the heart.

Key words: detraining, heart rate variability, exercise, autonomic, cardiovascular

Heart rate variability (HRV) has emerged as an indicator of risk for cardiac and all-cause mortality among patients with disease and in the general population (7, 8, 14, 20, 23-25, 41, 43). Various computations have been employed to express HRV, including time domain indices such as the standard deviation of normal R-R intervals collected over 5-minute periods (SDNN), and frequency domain variables such as power spectral components at low- and high-frequency power bands (0.04-0.15Hz, and 0.15-0.4 Hz, respectively). While the clinical relevance of HRV has been well documented, the physiologic correlates of HRV are less clear.

Early investigations of autonomic control of HRV in animals (4, 13, 38, 39) indicated that low-frequency (LF) modulations of heart rate (i.e., 0.04-0.15 Hz) are sympathetic-mediated, and that HF modulations of heart rate (i.e., 0.15- 0.40 Hz) occur in response to vagal activity. In humans, however, the picture is less clear. The exami-

nation of HRV in humans is limited to indirect methods, such as: during maneuvers that change autonomic activity, e.g. exercise (2, 17, 26), tilt (17, 36), head-down neck flexion (28), and psychological challenge (9); during pharmacologic interventions (see Berntson et al. (4) for a review); and as a consequence of hyperadrenergic conditions including cardiovascular diseases (7, 8, 23-25, 41, 43) and diabetes mellitus (29, 33). The cumulative results of these studies confirm that the time domain indices of HRV and HF power reflect vagal modulation of the heart; however, LF power appears to reflect mixed sympathetic and vagal and control.

Further indirect evidence comes from studies linking aerobic power and endurance training to HRV (5,6, 15-18, 21, 30, 32, 35). Jensen-Urstad et al. (16) reported lower resting heart rates (HRs) and greater HRV in elite runners when compared to sedentary controls. DeMeersman (12) observed, across a wide age-range of subjects (15-83 years), that trained runners had 23% greater HRV than age-matched, less active counterparts. Exercise interventions have yielded impressive results in enhancing HRV in healthy young and older adults by a magnitude of 17% and 58%, respectively (25). While the effects of exercise training on HRV have been explored, the effects of detraining (or reduced volume training) on HRV have not been addressed in an experimental fashion. A few studies have approached the issue of detraining by examining athletes presumed to be in a detrained state and subsequently retraining them (5, 17, 34). Results from Furlan et al. (17) and Bonaduce et al. (5) suggest that "retraining the "detrained" athlete results in an increase in resting heart period (i.e. decrease in resting heart rate), but no change in HRV parameters. In the absence of evidence of shifts in sympathovagal balance, these authors attributed exercise training induced changes in resting heart rate to changes in the intrinsic rhythm of the heart. While true detraining data on HRV parameters are currently lacking, the results of the above are not inconsistent with our present understanding of the influence of detraining on autonomic cardiovascular regulation. Detraining is characterized by differential rates of change in aerobic power and autonomic activity. Coyle et al. (12) demonstrated that $\dot{V}O_2$max is significantly reduced by as early as 2 weeks into a detraining period, but that changes in autonomic nervous system activity, indicated by reduced plasma catecholamine concentration, do not occur until 12 weeks of detraining.

The purpose of the present investigation was to examine changes in aerobic power, RR intervals, and HRV during a 5-week period of reduced volume training in elite swimmers. Based on Coyle's (12) findings, and in concert with what is known about "retraining" (5,17), we hypothesized that a 5-week reduction in training volume would evoke a significant decrease in aerobic power, and resting RR intervals, but that HRV parameters would not change. Lastly, as the study involved a repeated-measures design, and inasmuch as breathing frequency influences HRV spectral density (10, 19), we elected to include a paced breathing condition in an attempt to standardize the test environment.

Methods

Participants

Ten healthy elite collegiate swimmers (8 male, 2 female) volunteered to participate in the study. At the outset of the study, all participants had been training a minimum of 20 hours per week for a minimum of 4 months. Exclusion criteria included acute medical conditions such as active infection or orthopedic injury, or taking prescription medication, ephedrine, acetominophen, ibuprofen, or aspirin. The participant characteristics can be found in Table 1.

Table 1. *Patient Characteristics*

VARIABLE	MEAN +/- SD
AGE	19.8 +/- 1.2 yrs.
WEIGHT	80.0 +/- 7.3 Kg
HEIGHT	180.7 +/- 6.1 cm.
% BODY FAT	10.3 +/- 5.1 %

Experimental design

This study involved the implementation of a quasi-experimental time series design consisting of six observational periods (O_1-O_6) and a 5-week volume reduction treatment (initiated at point "T"), as follows: O_1 O_2 T O_3 O_4 O_5 O_6 (Figure 1). Observations 1 and 2 were conducted two weeks prior to the end of the competitive swim season and the end of the season, respectively. Observations 3 and 4 were conducted following the first and second week of a 2-week tapering (reduced volume) period. Observation 5 and 6 were performed following the first and third week of a 3-week period completely void of swimming activities. ECG data were collected at all observation points. $\dot{V}O_2$max was measured at observation points 1, 2, 4, 5, and 6.

Instruments

A Biopac MP100 data acquisition system and its companion AcqKnowledge ACK100 software program were used to capture, process, and analyze electrocardiograph (ECG) data. A standard three-site skinfold measurement was employed as an index of body composition. Breath-by-breath gas and spirometry measurments were made using the Sensormedics Vmax 29c Metabolic Cart.

Procedures

All procedures described herein were approved by the host site's institutional review board for research using human subjects.

Upon arriving at the laboratory for the initial visit (O_1), the participant signed a standard informed consent form and completed a medical questionnaire. Subsequently, the height, weight, and body composition (3-site skinfold test) were measured using standard laboratory techniques. During subsequent visits (O_2-O_6), a brief medical review was conducted to ensure that the participant's medical history had not changed, and that they had not begun taking any medications.

ECG data were collected during all visits. ECG electrodes were placed on the patient in a standard three-lead configuration, and the leads from the Biopac MP100 ECG module were attached such that the electrical activity of the heart was obtained from the "Lead-III" perspective. Following the placement of the electrodes, the patient was asked to lie quietly in the supine position for a 10-minute acclimatization period. Five minutes of ECG data were then collected while the participant remained resting quietly. During this time, the participant received no instruction, nor interaction from the examiner, and the breathing pattern was "spontaneous". This rest period was followed by a 5-minute paced respiratory control task. This procedure was invoked in an effort to standardize breathing pattern across observations, so that the results could be attributed to the treatment rather than potential within-subjects differences in breathing pattern. During this phase of the investigation, the participant was instructed to pace his or her breathing at a rate of 0.2

Hz (5 seconds per cycle). The participant practiced pacing his or her respiration for a period of 1 minute, and then performed this activity for 5 minutes, during which continuous ECG data were acquired.

At baseline (O_1 & O_2), and weeks 2,3, and 5 (O_4-O_6), into the treatment, the participant was evaluated for peak oxygen consumption using incremental arm-ergometry. A Polar monitor was placed around the participant's chest, and was used to collect heart rate data during the test. The participant was instructed to lie in a prone position on a tilt table set at 5-degree upright. The participant was given test instructions and then the Vmax 29c mouthpiece was placed in the participant's mouth. The participant was instructed to warm-up on a Monark arm-ergometer at zero resistance for a period of 2 minutes. Immediately after the warm-up period, the participant began the incremental arm-ergometer protocol consisting of 2-minute stages beginning at 50 watts. The workload was increased by 25W per stage, until the participant was unable to maintain a crank frequency of 100 (+/- 10) RPMs, or until other termination criteria for testing were apparent (e.g. signs or symptoms of adverse CV responses, participant requests to stop). Heart rate and rating of perceived exertion (Borg 20-point scale) were recorded at least once per stage. The criteria used to verify that $\dot{V}O_2$max was achieved were a respiratory quotient > 1.15, plateau in heart rate and relativeVO_2, and an RPE > 19. Participants were excluded from the study if they did not achieve at least three of the 4 criteria on each test.

Table 2. *$\dot{V}O_2$max Test Results*

	Baseline		Reduced Training Volume		
			Week 2	Week 3	Week 5
	O_1	O_2	O_4	O_5	O_6
$\dot{V}O_2$max ($ml \cdot kg^{-1} \cdot min^{-1}$)	33.2 ± 7.4	34.0 ± 8.3	28.2 ± 6.9*	28.0 ± 6.9*	28.5 ± 5.6*
HRmax (bpm)	178.8 ± 7.5	177.8 ± 7.2	175 ± 12.4	179.3 ± 5.5	181 ± 7.9
RQ	1.06 ± .17	1.09 ± .05	1.09 ± .14	1.12 ± .20	1.10 ± .11
VE ($l \cdot min^{-1}$)	75.8 ± 11.4	83.2 ± 18.9	71.3 ± 20.8	76.7 ± 30.6	77.4 ± 20.1

* = different from end-season (O_2), p< .05
HR = heart rate
RQ = respiratory quotient (VCO_2/VO_2).
VE = ventilation

Data reduction

Measures of HRV were derived according to the recommendations of the European Task Force on Pacing and Electrophysiology (42). ECG data were collected in real-time at a sampling rate of 250 Hz and plotted as a tachogram of heart period. The ECGs (and tachograms) were visually inspected for non-sinus activity. The tachograms were evaluated for the standard deviation of all R-R intervals (SDNN) and then re-sampled at a rate of 4.0 Hz, and interpolated using a Hamming window. The spectral power density of the tachogram waveform was then derived via the squared modulation of a linear fast Fourier transformation (FFT). This transformation requires a number of data points equal to a power of 2. Since 5 minutes of data sampled at 4.0 Hz results in the acquisition of 1200

Table 3. *Heart Rate Variability*

	Baseline		Reduced Training Volume			
			Week 1	Week 2	Week 3	Week 5
	O_1	O_2	O_3	O_4	O_5	O_6
R-R PB	894 + 154	897 + 127	956 + 91	912 + 131	957 + 161	833 + 124 *
R-R SB	1043 + 102 ‡	971 + 93 ‡	1060 + 134 ‡	1000 + 137‡	1040 + 156 ‡	931 + 78 †‡
HR PB	67.1 + 5.8	66.9 + 7.1	62.8 + 10.5	65.8 + 7.0	62.7 + 5.9	72.0 + 6.7 *
HR SB	57.5 + 10.2 ‡	61.8 + 10.4 ‡	56.6 + 7.9 ‡	60.0 + 7.3 ‡	57.7 + 6.7 ‡	64.0 + 11.9 †‡
SDNN PB	101.1 + 46.3	114.3 + 49.8	123.8 + 47.5	122.5 + 47.3	122.3 + 43.2	89.4 + 15.6
SDNN SB	65.1 + 33.3 ‡	77 + 32.2 ‡	89.1 + 30.3 ‡	103.1 + 42 ‡	86.1 + 34.4 ‡	66.5 + 25.8 ‡
[1]LF PB	6.34 + 4.51	6.95 + 4.53	6.71+ 5.50	3.73 + 2.53	8.33 + 6.33	4.00 + 2.77
[1]LF SB	1.83 + 1.41	1.91 + 1.40	2.51 + 1.10	3.73 + 3.54	1.87 + 1.32	1.84 + 1.49
HF PB	4.83 + 4.11	5.14 + 3.36	4.11 + 3.81	3.26 + 3.17	2.60 + 2.70	10.0 + 10.4
HF SB	1.97 + 1.43	1.61 + 1.51	2.16 + 2.18	3.11 + 2.70	2.70 + 2.70	1.56 + 1.54
[2]LFnu PB	68 + 33	62 + 26	61 + 19	52 + 27	79 + 11	67 + 19
[2]LFnu SB	50 + 25	63 + 20	59 + 14	54 + 15	49 + 21	57 + 18
[3]lnLFnu PB	4.2 + 2.5	4.1 + 2.2	4.1 + 1.9	3.9 + 2.3	4.4 + 1.4	4.2 + 1.9
[3]lnLFnu SB	3.9 + 2.2	4.1 + 2.0	4.1 + 1.6	4.0 + 1.7	3.9 + 2.0	4.0 + 1.9

All values represent mean + s.d.

RR = heart period in msec, HR = heart rate in bpm, SDNN = standard deviation of normal RR intervals in msec, LF and HF are low- and high-frequency power (units x 103 $msec^2$), nu = normalized units, ln = natural logarithm.

PB and SB are the test conditions: paced breathing and spontaneous breathing, respectively

* = different from O_2-O_5 , $p<0.01$

† = different from O_3-O_5 , $p<0.01$

‡ = different from paced breathing , $p<0.05$

1 = Absolute units of LF and HF are reported, but not subjected to statistical analyses due to poor reliability.

2 = by definition, mean HFnu is 100-mean LFnu, and the s.d. of HFnu = s.d. LFnu

3 = natural logarithm transformation used to correct for violations of assumptions of linear models. Inferences will be the same for lnHFnu.

data points, 1024 points acquired during the 5-minute spontaneous and paced breathing periods were evaluated. The resultant power density spectra were then analyzed according to recent guidelines established for analysis of HRV (35). Values were derived for total power output (<0.40 Hz), LF power output (0.04-0.15 Hz), thought to reflect sympathetic as well as baroreceptor mediated vagal control, and HF power output (0.15-0.40 Hz), thought to reflect vagal modulation. Due to the poor reliability of LF power under short term data collections (1), and in accordance with the guidelines cited above (42), the LF and HF power outputs were normalized (LFnu and HFnu, re-

spectively) by dividing each by the total power minus very low-frequency power (<0.04Hz), and expressed as a percent (multiplying by 100). As HRV data violate assumptions of linear models, natural logarithm transformations of the normalized HRV data were made. Both the normalized units (LFnu, HFnu) and the transformed values (lnLFnu, lnHFnu) were submitted for statistical analysis.

Data analysis

The SAS System 6.12 statistical analysis software (Cary, North Carolina, USA) was employed for all analyses. Repeated measures ANOVA was used to compare $\dot{V}O_2$max and HRV indices (RR, SDNN, LFnu, HFnu, lnLFnu, and lnHFnu) at O_1 and O_2. A separate set of repeated-measures ANOVAs was used to compare the dependent measures at O_2, O_3, O_4, O_5, and O_6, and to compare $\dot{V}O_2$max data at O_2, O_4, O_5, and O_6. In addition, dependent t-tests were run on HRV parameters between spontaneous and paced breathing conditions for each observation period. An alpha of $p< 0.05$ was used to determine significant differences, and the Bonferroni correction equation was used when multiple comparisons were made.

Results

Six participants (five males and one female) completed O_2 -O_6. Only five participants (four males and one female) completed O_1. Therefore, O_1 was compared to O_2 only. Two of the ten participants dropped out of the study, and the other two participants did not achieve at least 3 of the 4 criteria for maximal effort on at least one arm-ergometer test.

There were no significant differences in $\dot{V}O_2$max, RR, mean RR interval (i.e., heart period/rate), and HRV data at baseline (O_1 vs. O_2) The data for these variables can be found in tables 2 and 3, respectively. The metabolic test results are listed in Table 2. The repeated measures ANOVAs revealed that HRpeak, RQ and VE did not change across time. $\dot{V}O_2$max was significantly lower ($p < 0.0125$) by the 2nd week into the detraining protocol (O_4), but did not change thereafter. The data indicate that $\dot{V}O_2$max dropped approximately 20% from baseline (O_2). The $\dot{V}O_2$max values for each participant are plotted in figure 2. The repeated measures ANOVA on the RR intervals reveal a shorter mean RR interval (i.e., heart period/rate) at O_6 compared to the other observation periods ($p<0.01$), and that this appeared regardless of breathing condition

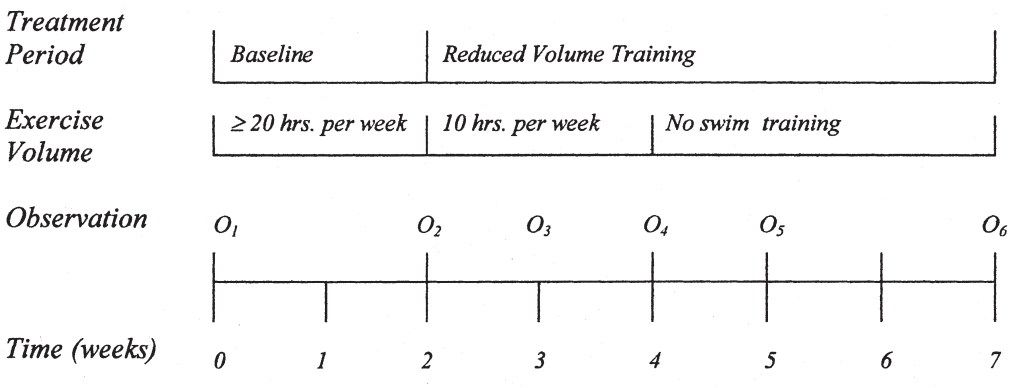

Figure 1. *Experimental Design: The quasi-experimental time series design involved two baseline observations (O_1 and O_2), two weeks of reduced training volume (O_3 and O_4), and three weeks of no swim-training (O_5 and O_6)*

(Table 3). Analysis of the HRV data (see Table 3) revealed no significant differences in any parameter across time under either the paced or spontaneous breathing conditions. Dependent t-tests revealed that paced breathing was associated with significantly shorter RR intervals and greater SDNN as compared to spontaneous breathing ($p<0.05$).

Discussion

The present study was designed to examine the effect of reduced training volume on maximum oxygen consumption and autonomic modulation of the heart in collegiate swimmers. The contribution of this study is the evidence that the rapid decline in $\dot{V}O_2$max following detraining is not associated with changes in time domain or spectral components of HRV at rest.

The group means for $\dot{V}O_2$max and HRV indices in the present study are as expected for this population. The $\dot{V}O_2$max values are similar to those that have been previously reported during incremental arm crank exercise, including recent data from Kang et al. (22) and Swaine & Winter (40). The observed values for HRV are comparable to those observed in other highly trained endurance athletes (5, 17, 32, 35). Moreover, the values for the frequency domain parameters for the present study appear similar to those of earlier reports in other athletes.

Considering the inherent difficulties in finding elite athletes willing to participate in reduced training studies, it is not surprising that there are few such studies available (5, 17, 34). In addition, the majority of those studies have used quasi-experimental design (lack of a control group), and small sample sizes. Despite these limitations the absolute decline in $\dot{V}O_2$max (35.4 + 10.1 vs. 28.15 + 6.9 $ml \cdot kg^{-1} \cdot min^{-1}$) in the present study are similar to those reported by Coyle et al. (12), and Raven et al. (33). Similar to Coyle et al. (12) the decline in $\dot{V}O_2$max was most rapid in the first two weeks following detraining (i.e. from O_2 to O_4, see Figure 2), after which $\dot{V}O_2$max stabilized over the remaining observation pe-

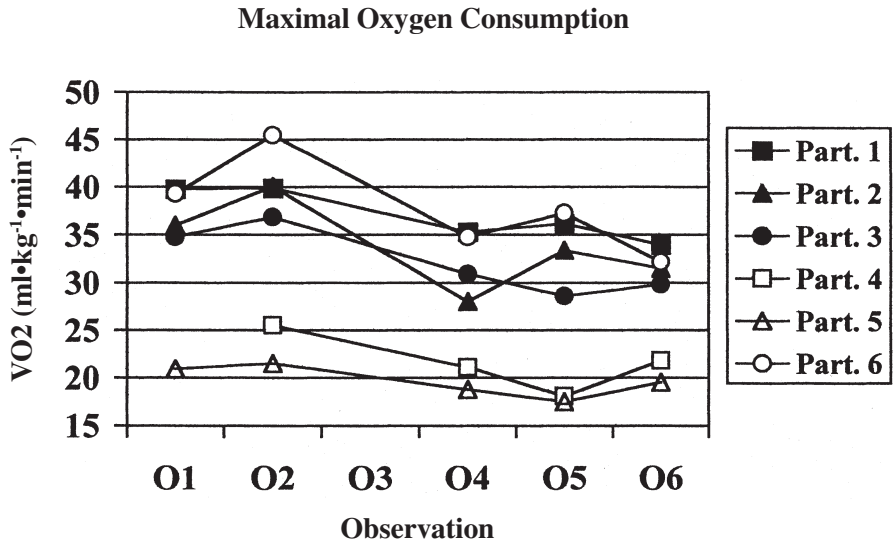

Figure 2. *Individual responses of $\dot{V}O_2$max to reduced training. Each line is a plot of the values observed for each of the participants in the study*

riods. Coyle et al. (12) proposed a rapid decline in blood volume as a major contributor to this early decline in $\dot{V}O_2$max.

The results of this investigation indicate that HRV indices did not change across the 5 weeks of reduced volume training. This is in agreement with previous studies (5, 17, 34). However, comparisons to previous studies are complicated because of design differences. In fact, the previous investigations are not per se detraining studies and none provide measures of HRV prior to the "detraining" period. Furthermore, the "detraining" periods that preceded the investigations by Rebelo et al. (34) and Furlan et al. (17), are not well described, and with respect to Bonaduce et al. (5), the detraining period consisted of 1 hour of cardiovascular work conducted 5 days per week, which is not a particularly strong "detraining" stimulus. Despite these limitations Furlan et al. (17) and Bonaduce et al. (5), reported no differences between trained and detrained athletes in time and frequency domain measures of HRV, nor did they observe changes in autonomic modulation of the heart with "retraining". It should also be mentioned that studies involving complete bed rest or simulated space flight studies (31, 37) reveal a shift towards sympathetic dominance (LFnu) The disparity between these studies and the results of the present investigation is likely reflecting differences in the magnitude of the stimuli, that is differences between reduced volume training and complete bed rest or "zero gravity."

The results also indicate that there was a significant shortening of the RR interval (higher HR) by the end of the detraining period. Prior to and during the first several weeks of detraining the athletes resting HRs averaged roughly 57 b•min^{-1} under spontaneous breathing conditions, whereas the mean HRs were 64 b•min^{-1} at O_6. This finding is also consistent with reports from Furlan et al. (17) and Bonaduce et al. (5) In addition, the results of the present study suggest that the changes in mean RR are not evident until the 5th week of detraining. In the absence of changes in autonomic modulation of the heart, the changes in mean RR interval may be the result of a heightened intrinsic heart rate and/or blood volume contraction. Such a suggestion is consistent with the position of Coyle et al. (12), who suggested that early changes in $\dot{V}O_2$max during detraining are not the result of altered autonomic function, but rather are more likely due to reduced blood volume (11, 28). Close inspection of the present data, however, suggests a trend towards reduced parasympathetic control of the heart by the end of the detraining period (O_6). This trend, while non-significant, makes it difficult to rule out changes in autonomic modulation as contributing to the sharp increase in HR (decrease in RR interval) between O_5 and O_6. However, in the absence of a statistically significant change in indicators of autonomic function, blood volume contraction and changes in intrinsic HR should be considered as potential mediators of the changes in RR interval.

This investigation included the collection of ECG data during conditions of paced and spontaneous breathing (PB and SB, respectively). This was done in an attempt to control for potential within subjects variability in breathing frequency across the treatment period. The inferences regarding the influence of the detraining period do not differ according to condition (PB vs. SB). While it is a limitation of this study that tidal volume and spontaneous breathing frequency are not reported, the lack of changes in HRV with detraining even under paced breathing conditions suggests that a detraining effect has not been obscured by within-subjects changes in ventilatory pattern.

In conclusion, the findings of this study suggest that HRV does not change after 5 weeks of reduced volume training. This is consistent with what is known about autonomic activity following detraining, and thereby provides further indirect evidence regarding the physiologic correlates of HRV. In addition, the results of this study support claims that the early change in $\dot{V}O_2$max during detraining is not the result of changes in autonomic control. Future studies should continue to address the relationships between physical activity, physical fitness and HRV, and the degree to which changes in activity and/or fitness relate to risk of cardiac and all-cause death.

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Received: October 09, 2000
Accepted: April 22, 2001

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