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## Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load

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**Buchheit, Martin, and Cyrille Gindre.** Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. *Am J Physiol Heart Circ Physiol* 291: H451–H458, 2006. First published March 3, 2006; doi:10.1152/ajpheart.00008.2006.—The objective of this study was to establish the separate associations between parasympathetic modulations of the heart [evaluated through heart rate (HR) variability (HRV) indexes and postexercise HR recovery (HRR) indexes] with cardiorespiratory fitness and training load. We have measured cardiorespiratory fitness through peak oxygen consumption ( $\dot{V}O_{2\max}$ ) and estimated weekly training load with the Baecke sport score in 55 middle-aged individuals ( $30.8 \pm 1.8$  yr, body mass index  $24.5 \pm 0.4$  kg/m<sup>2</sup>). HRV indexes were analyzed at rest under controlled breathing, and HRR was estimated from HR curve fitting after maximal exercise or from measurements of the number of beats recovered at 60 s after exercise. Multiple linear regressions were used to investigate the separate relationships between vagal-related HRV indexes and  $\dot{V}O_{2\max}$  and Baecke scores. On the basis of their  $\dot{V}O_{2\max}$  and Baecke scores, subjects were classified as fit or unfit and as low trained (LT) or moderately trained (MT), which yielded four groups: Unfit<sub>LT</sub>, Unfit<sub>MT</sub>, Fit<sub>LT</sub>, and Fit<sub>MT</sub>. Vagal-related HRV indexes were positively correlated with  $\dot{V}O_{2\max}$  ( $P < 0.05$ ) but not with Baecke scores. In contrast, HRR indexes were related to Baecke scores ( $P < 0.05$ ) but not with  $\dot{V}O_{2\max}$ . Fit<sub>LT</sub> and Fit<sub>MT</sub> had significantly higher ( $P < 0.05$ ) normalized vagal-related HRV indexes than Unfit<sub>LT</sub> and Unfit<sub>MT</sub>, but HRR did not change. Moderate training was associated with significantly lower HRR indexes both in Unfit<sub>MT</sub> and Fit<sub>MT</sub> compared with Unfit<sub>LT</sub> and Fit<sub>LT</sub>, but there was no difference in vagal-related HRV indexes. These results indicate that vagal-related HRV indexes are related more to cardiorespiratory fitness, whereas HRR appears to be better associated with training load.

vagal-related indexes; postexercise heart rate recovery; aerobic training

TIME AND FREQUENCY DOMAIN ANALYSES of heart rate (HR) variability (HRV) constitute noninvasive methods commonly used to evaluate parasympathetic tone and sympathovagal balance (38, 48). More precisely, high-frequency power (HF) and especially the normalized HF ratio, HF/(LF + HF), where LF stands for low-frequency power, have been recognized to give insight into vagal activity. HR postexercise recovery (HRR) has also been advanced as another index of vagal activity (1, 42). The main interest of measuring cardiac vagal activity lies in its prognostic value in cardiovascular risk because it has been shown to exert a cardioprotective effect through enhanced cardiac electrical stability (4). Thus low HRV levels (48, 49) and slower HR recovery (16), both giving evidence of impaired

parasympathetic activity, have been related to increased cardiovascular risk. In complement to this clinical interest, vagal activity has recently been shown to be an important determinant of aerobic training response (19, 24, 25). The use of HRV (20, 21, 29) and HRR (27) as training monitoring tools has progressively increased and could hold interest for monitoring endurance training in a wide range of populations, in a health as well as in a competition perspective.

Previous research has suggested that vagal modulation of HR is influenced by certain physiological factors such as aging (54), body fatness (15), aerobic training, and physical fitness (2, 6, 12, 13). Although the negative effects of age and body fatness are clear, there still remains some uncertainty about the beneficial effects of physical fitness and training load on vagal-related HRV indexes and HRR. Excluding overtraining conditions (26, 53), most authors have observed higher HRV vagal-related indexes (2, 12, 13, 51) and faster HR decay after exercise in well-trained athletes and fit individuals (17, 22) and in patients after participation in cardiac rehabilitation programs (32). HR recovery has also been shown to be related to exercise capacity (16). Nevertheless, other investigations have failed to show any association between high aerobic fitness and enhanced indexes of parasympathetic activity (11, 34) or have reported no significant HRV improvement after training (5, 14, 35, 43).

Because athletes who display the highest cardiorespiratory capacities are generally those engaged with the highest training loads, and the less fit are those who train less, the respective effects of physical fitness and training load on autonomic function are often confounded, which may partially explain the inconsistent results reported (22, 30, 37, 50–52). In most cross-sectional studies, the relationship between maximum oxygen uptake ( $\dot{V}O_{2\max}$ ) and HRV (51) or HRR (37) has been investigated without regard for individual training loads, or by comparing fit and trained subjects to unfit and sedentary ones (17, 18). In longitudinal investigations, the effect of aerobic training on HRV (30, 50, 52) or HRR (22) might easily have been masked by concomitant cardiorespiratory fitness improvement resulting from the training program.

Such a confounding influence does not occur in sedentary individuals who present spontaneously high cardiorespiratory fitness without any training load. Indeed, there exist individuals with advantageous predispositions and genetic parameters that determine  $\dot{V}O_{2\max}$ . In this particular type of individual, the link between physical fitness and autonomic modulation can be

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observed without the masking effect of training load. Atypical cases of trained subjects with paradoxically poor physical fitness help investigate the effect of training load without the complicating effect of fitness condition.

The purpose of this cross-sectional study was to examine the respective associations between vagal modulation of HR and either aerobic fitness or training load in a population of middle-aged men. We simultaneously measured vagal-related HRV indexes and HRR time constant ( $HRR_t$ ) to investigate their separate links with cardiorespiratory fitness as evaluated by  $\dot{V}O_{2\max}$  and weekly training loads as estimated by the Baecke sport score. The relationships between autonomic activity and physical fitness and training load will be first investigated in the overall population of subjects. Next, we compare HRV and HRR in four subgroups that allow us to observe the incidence of low and high fitness in individuals with similar training loads and the effect of different training loads in subjects having equivalent cardiorespiratory fitness levels. To ensure the absence of overload or stressful stimuli, which can affect autonomic regulation (2, 41), subjects were selected with regard to the profile of mood states (POMS) (39) questionnaire for assessing fatigue and psychoemotional states. We also exclude highly trained athletes to avoid paradoxical HRV levels previously observed (8, 9)

## METHODS

**Subjects.** The sample size was calculated by using Minitab 13.2 Software (Minitab, Paris, France). To attain statistical significance ( $P = 0.05$  at a power of 80% with a confidence interval of 5%), we required a sample size of 12 subjects for each of our four groups to show a 0.2 difference in  $HF/(LF + HF)$ . The magnitude of expected differences was derived from previous investigations (7, 9). This total sample was then rounded up to 60. Sixty-one middle-aged nonsmoking, nonobese men [aged  $33.8 \pm 3.8$  yr (mean  $\pm$  SD), body mass index (BMI)  $24.5 \pm 0.4$  kg/m<sup>2</sup>], with no history or clinical signs of cardiovascular or pulmonary disease, were recruited for this study. Candidates were not taking any kind of medication and did not show abnormal blood pressure or electrocardiographic patterns. This study conforms with the recommendations of the Declaration of Helsinki, and the participants gave voluntarily their written consent to participate in this experiment, which had been approved by the local Ethics Committee.

**Fatigue and mood assessment.** Before beginning HR recordings, the subjects were asked to fill out the POMS questionnaire (39) so as to assist researchers in detecting any mood disturbances commonly associated with overload. The subject presenting a measure of mood (total POMS score) higher than 180 was not retained ( $n = 1$ ).

**Cardiorespiratory fitness testing.** Respiratory gas exchanges were measured by using an automated breath-by-breath system (CPX, Medical Graphics; St Paul, MN). Before each test, the O<sub>2</sub> and CO<sub>2</sub> analysis systems were calibrated. Subjects performed a ramp test on a horizontal motor-driven treadmill (Powerjog, J601, Sport Engineering; Birmingham, UK) or on a cycle ergometer (Orion STE; Toulouse, France), depending on their sport specialty. After a 10-min warm-up at 10 km/h (or 100 W), the test began at an initial speed of 10 km/h and was increased by 0.5 km/h (or 25 W) per 1-min stage. The test was stopped when a subject could no longer maintain the running speed or pedaling rate.  $\dot{V}O_{2\max}$  was defined as the highest  $\dot{V}O_2$  attained in a 15-s period. Subjects were considered to have reached  $\dot{V}O_{2\max}$  if at least three of the following four criteria were met: 1) a plateau in  $\dot{V}O_2$  despite an increasing work load, 2) a final respiratory exchange ratio  $>1.1$ , 3) a HR within 10 beats/min of the age-predicted maximum,

and 4) visible exhaustion (28). Subjects who did not meet three of these criteria were not retained for the analyses ( $n = 3$ ).

**Training load assessment by questionnaire.** The sport score of the Baecke questionnaire (3), which gives insight into the intensity of physical activity, was used to evaluate weekly training load. The Baecke sport score is obtained from activity duration (h/wk), frequency (mo/yr), and intensity at which the activity is performed. Intensity codes are unitless and were originally based on energy costs. To avoid the special case of low HRV in highly trained individuals (8, 9), we did not include subjects having a sport score higher than 14 ( $n = 2$ ).

**Autonomic control assessment protocol.** The experiments were performed between 10:00 and 12:00 AM and 4:00 and 6:00 PM in an air-conditioned room with ambient temperature maintained at 21°C. The subjects were requested to avoid strenuous exercise for 2 days before the experiments. They had their usual breakfast at least 3 h before the beginning of the experimentation, avoiding coffee. After 30 min of rest, the subjects were asked to remain quietly supine for 10 min without speaking or making any movements. Subjects breathed at 12 cycles/min by synchronizing their breathing pattern with an electronic metronome rhythm, so that the respiratory rate would influence the HRV indexes of each subject in the same way.

**HR measurements.** After application of conductive gel, an electrode transmitter belt (T61, Polar Electro; Kempele, Finland) was fitted to the chest of each subject as described by the manufacturer. HR was continuously monitored during the 40 min of rest by using an S810 HR monitor (Polar Electro; Kempele, Finland).

**HRV analysis.** The R-R intervals, i.e., the time between the R peaks of consecutive QRS complexes recorded by the S810, were calculated and visually checked for artifacts. Occasional ectopic beats were identified and replaced with interpolated R-R intervals. HRV analyses were performed on the last 5 min of the 10-min controlled breathing in the lying-resting position that assures stationarity of the data. The mean of R-R intervals (mR-R), the standard deviation of normal R-R intervals (SDNN), the percentage of successive R-R differences greater than 50 ms ( $pNN_{50}$ ), and the root-mean-square difference of successive normal R-R intervals (RMSSD) were calculated for the 5-min period. Power frequency analysis of the 5-min recordings was performed sequentially with a fast Fourier transform based on a nonparametric algorithm with a Welch window after the ectopic-free data were detrended and resampled. A fixed linear resampling frequency of 1,024 equally spaced points per 5 min period was used. The power densities in the LF band (0.04–0.15 Hz) and the HF band ( $>0.15$ –0.50 Hz) were calculated from each 5-min spectrum by integrating the spectral power density in the respective frequency bands. The different HRV indexes, SDNN, RMSSD,  $pNN_{50}$ , LF, HF, and  $HF/(LF + HF)$  were calculated.

**Postexercise HR recovery assessment.** HR was analyzed during the recovery period, immediately after the exercise. HRR obtained by fitting postexercise HR recovery to a first-order exponential decay curve was used as the HR recovery index (42). At least 6 min of HR were recorded after the end of exercise. Because different body posture results in different values for HRR, we paid attention that all subjects remained inactive in a sitting position. The subjects who had undertaken cycling testing stayed on the ergometer and were allowed to place their feet on the footrest near the flywheel. Those tested by a running exercise were asked to sit immediately on a chair placed near the treadmill when they stopped their effort. HRR was then calculated in two ways. The first HRR indexes were obtained by modeling the resultant first 5 min of HR vs. time data with an iterative technique by using SigmaplotE (SPSS Science; Chicago, IL) by the following equation:  $HR = HR_0 + HR_{\text{amp}}e^{-T/HRR_t}$ , where  $HR_0$  is resting (final) HR;  $HR_{\text{amp}} = \text{maximal HR (HR}_{\text{max}}) - HR_0$ ;  $T$  is time (s), and  $HRR_t$  is the decay constant.  $HRR_t$ ,  $HR_{\text{max}}$ , and  $HR_{\text{amp}}$  were retained for statistical analysis. A complementary and more commonly used HRR

index is also defined as the difference between the HR at the end of exercise and the HR recorded 60 s later ( $HRR_{60s}$ ) (16).

**Respective relationships between physical fitness, training load, and cardiovascular autonomic indexes.** Because, as mentioned, we eliminated a total of six individuals on the basis of our selection criteria, data of 55 subjects were used for the regression analyses.

**Physical fitness- and training load-matched groups.** Subjects presenting a  $\dot{V}O_{2\max} < 50 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$  were considered as unfit (Unfit) and those with  $\dot{V}O_{2\max}$  values  $> 55 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$  as fit (Fit). Subjects having intermediate  $\dot{V}O_{2\max}$  (between 50 and  $55 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ ) were not retained ( $n = 6$ ). As previously described (9), we considered that subjects displaying a sport score below 5 and having  $< 2 \text{ h/wk}$  of physical activity to have a low weekly training load (LT), and subjects with a sport score between 6 and 12 and practicing 4–6 h/wk of various aerobic activities, such as running or aerobic sports, to have a moderate weekly training load (MT). None of the subjects was practicing resistance sports. Matching the remaining 49 subjects according to fitness level and training load yielded four groups, which are presented in Table 1.

**Statistical analysis.** Statistical analyses were carried out by using Minitab 13.2 software. The distribution of each variable was examined with Kolmogorov-Smirnov and Shapiro-Wilk normality tests. Because absolute spectral indexes are skewed, these data were transformed by taking the natural logarithms of LF and HF power indexes to allow parametric statistical comparisons that assume normal distributions. As the relationships between the different parameters were similar for exercise protocols, results from treadmill and bicycle testing were pooled. Multiple linear regressions were used to establish the respective relationships between HRV and HR recovery indexes and  $\dot{V}O_{2\max}$  (using Baecke sport score as covariable) and between HRV and HR recovery indexes and weekly training load (using  $\dot{V}O_{2\max}$  as covariable). Other polynomial regressions were rejected on the basis of much higher residuals. These linear relationships were also adjusted on potential confounders (exercise protocol, BMI, and age), which were introduced in the model as additional covariables. Influential observations were identified through calculation of Cook's distance for each observed parameter. Colinearity among parameters was checked for and absent. The sample size/factors number ratio was high enough to exclude any overfitting effect (40). Concerning the four fitness- and training load-matched groups, there were no significant differences either between mean age and BMI or between exercise protocols. A three-way ANOVA with Tukey's post hoc test was used to compare HRV and HR recovery index distributions as a function of 1) cardiorespiratory fitness (Fit vs. Unfit), 2) weekly training load (LT vs. MT), and 3) exercise protocol (treadmill vs. bicycle testing), with results adjusted on BMI and age. Interaction factors were considered to evaluate any possible heterogeneity of the associations across levels of the other factors. All data are presented as means ( $\pm$ SD) and least-squares means ( $\pm$ SE). The level of significance was set at  $P < 0.05$ .

## RESULTS

**Relationships between cardiorespiratory fitness and HRV vagal-related indexes.** Zero-order relationships between  $\dot{V}O_{2\max}$  and HRV vagal-related indexes [RMSSD, PNN<sub>50</sub>, HF and HF/(LF + HF)] were all significant ( $n = 55$ ,  $P < 0.001$ ). The positive association between  $\dot{V}O_{2\max}$  and HF/(LF + HF) ( $P < 0.001$ ) is illustrated in Fig. 1, *top left*. The relationships between cardiorespiratory fitness and each HRV vagal-related index adjusted on the Baecke sport score, BMI, and age are presented in Table 2. Partialling out the effect of training reduced the level of significance, but mR-R and all vagal-related HRV indexes remained positively correlated with  $\dot{V}O_{2\max}$  ( $P < 0.05$ ).

**Relationships between cardiorespiratory fitness and HR recovery indexes.** Figure 1, *bottom left*, illustrates the nonsignificant zero-order relationship between  $\dot{V}O_{2\max}$  and  $HRR_t$ . Table 2 presents the correlations adjusted on Baecke score, BMI, and age between  $\dot{V}O_{2\max}$  and HR recovery indexes.  $HRR_t$  and  $HRR_{60s}$  were not linked to  $\dot{V}O_{2\max}$ , whatever the regression model used (with or without adjustment on  $\dot{V}O_{2\max}$ , BMI, and age,  $P > 0.05$ ).

**Relationships between training loads and HRV vagal-related indexes.** Zero-order regressions showed that SDNN is linked to Baecke sport score ( $n = 55$ ,  $P < 0.05$ ) but none of the vagally mediated HRV indexes ( $P > 0.05$ ). Figure 1, *top right*, shows the nonsignificant zero-order relationship between Baecke sport score and HF/(LF + HF). Table 2 presents the relationship between Baecke sport score and HRV vagal-related indexes adjusted on  $\dot{V}O_{2\max}$ , BMI, and age. After adjustment on  $\dot{V}O_{2\max}$ , BMI, and age, SDNN was no longer significantly linked with the Baecke sport score ( $P = 0.09$ ), as were also none of the other HRV indexes ( $P > 0.05$ ).

**Relationships between training loads and HR recovery index.** The negative zero-order relationship between  $HRR_t$  and Baecke sport score is illustrated in Fig. 1, *bottom right* ( $P < 0.001$ ,  $n = 55$ ). Relationships between Baecke score and HR recovery indexes adjusted on  $\dot{V}O_{2\max}$ , BMI, and age are given in Table 2.  $HR_{\max}$ ,  $HR_{\text{amp}}$ , and  $HRR_t$  were all inversely related to training load, with ( $P < 0.001$ ) or without adjustment on  $\dot{V}O_{2\max}$ , BMI, and age ( $P < 0.001$ ). In contrast,  $HRR_{60s}$  only tended to be associated with training load without ( $P = 0.06$ ) or with adjustment ( $P = 0.09$ ).

**Indexes of cardiovascular parasympathetic activity in fitness- and training load-matched groups.** Table 3 gives adjusted means of HRV and postexercise HR recovery indexes

Table 1. Characteristics of the subjects at different cardiorespiratory- and training load-matched levels

	Unfit <sub>LT</sub>	Unfit <sub>MT</sub>	Fit <sub>LT</sub>	Fit <sub>MT</sub>
<i>n</i> (bicycle/treadmill testing)	12 (10/2)	13 (7/6)	11 (7/4)	13 (5/8)
Age, yr	31.8 $\pm$ 3.2	30.6 $\pm$ 5.1	27.7 $\pm$ 2.9	27.3 $\pm$ 2.5
BMI, kg/m <sup>2</sup>	25.0 $\pm$ 0.7	24.4 $\pm$ 0.8	23.1 $\pm$ 0.5	23.0 $\pm$ 0.5
$\dot{V}O_{2\max}$ , ml $\cdot$ min <sup>-1</sup> $\cdot$ kg <sup>-1</sup>	44.1 $\pm$ 1.9	45.4 $\pm$ 1.8	61.4 $\pm$ 1.3*†	64.2 $\pm$ 2.1*†
Baecke sport score	2.4 $\pm$ 0.4	8.9 $\pm$ 0.7*	3.6 $\pm$ 0.3†	8.1 $\pm$ 0.6*‡
Total of mood score	167.7 $\pm$ 7.9	155.9 $\pm$ 12.1	158.4 $\pm$ 8.5	153.6 $\pm$ 9.7

Values are means ( $\pm$ SD) for age, body mass index (BMI), cardiorespiratory fitness [maximal O<sub>2</sub> uptake ( $\dot{V}O_{2\max}$ )] training load (Baecke sport score), and total score on the Profile of Mood State questionnaire (see Ref. 39). Fit, subjects presenting a  $\dot{V}O_{2\max} > 55 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ ; Unfit, subjects presenting a  $\dot{V}O_{2\max} < 50 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ . LT and MT refer to low or moderately trained subjects, respectively. \*Significant difference vs. Unfit<sub>LT</sub> ( $P < 0.05$ ). †Significant difference vs. Unfit<sub>MT</sub> ( $P < 0.05$ ). ‡Significant difference vs. Fit<sub>LT</sub> ( $P < 0.05$ ).

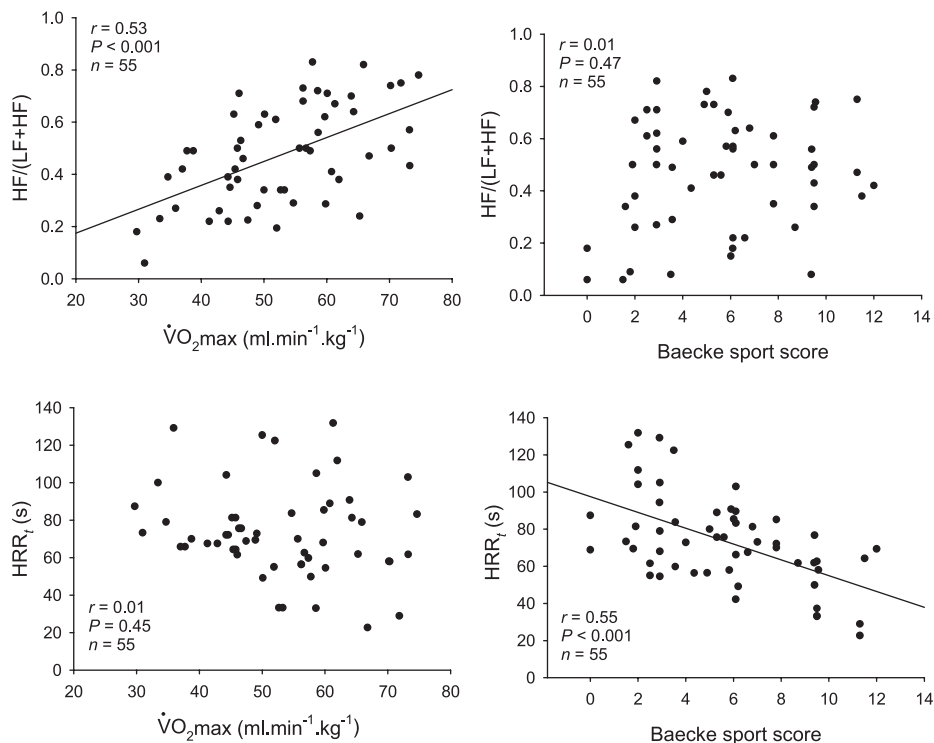


Fig. 1. Zero-order correlation between cardiorespiratory fitness [maximum O<sub>2</sub> uptake ( $\dot{V}O_{2max}$ ); *left*], training load (Baecke sport score; *right*) and normalized heart rate (HR) variability (HRV) vagal-related index [HF/(LF + HF), where HF and LF are high- and low-frequency power, respectively; *top*] and postexercise HR recovery time constant (HRR<sub>t</sub>; *bottom*) in 55 middle-aged subjects.

for the four fitness- and training load-groups. Figure 2 shows the values of HF/(LF + HF) and HRR<sub>t</sub> as a function of  $\dot{V}O_{2max}$  and Baecke score for the four groups. Intragroup comparisons revealed that both Fit<sub>LT</sub> and Fit<sub>MT</sub> had significantly higher HRV vagal-related indexes [indicated by higher RMSSD, pNN<sub>50</sub>, RMSSD, HF, and HF/(LF + HF); P < 0.05] than Unfit<sub>LT</sub> and Unfit<sub>MT</sub> (P < 0.05). Unfit<sub>MT</sub> and Fit<sub>MT</sub> had faster HRR<sub>t</sub> (P < 0.05) than Unfit<sub>LT</sub> and Fit<sub>LT</sub> but not higher HRR<sub>60s</sub> (P > 0.05).

**DISCUSSION**

By analyzing HRV indexes and postexercise HR recovery in middle-aged subjects with simultaneous consideration of

$\dot{V}O_{2max}$  and Baecke sport score, the present study is the first to establish the separate associations of cardiorespiratory fitness and training load with vagal modulation of the heart. Current findings indicate that HRV indexes and postexercise HR recovery time constant, although both vagally mediated, are not similarly associated with cardiorespiratory fitness and training load. Vagal-related HRV indexes are related more to cardiorespiratory fitness and do not reflect habitual training, whereas HRR<sub>t</sub> is negatively correlated with training load but not associated with  $\dot{V}O_{2max}$ .

*Cardiorespiratory fitness and HRV vagal-related indexes.* Our results confirm that vagal-related HRV indexes are significantly associated with  $\dot{V}O_{2max}$ . Whatever the training load, the

Table 2. Relationships between HRV indexes and physical fitness and training load

	$\dot{V}O_{2max}$		Baecke Sport Score	
	$\beta$	P	$\beta$	P
<b>HRV indexes</b>				
mR-R, ms	7.63 ± 2.42	<0.01	15.97 ± 7.69	0.04
SDNN, ms	0.62 ± 0.39	0.11	2.19 ± 1.16	0.08
RMSSD, ms	1.07 ± 0.43	0.01	1.64 ± 1.37	0.24
pNN <sub>50</sub> , %	0.29 ± 0.13	0.04	0.46 ± 0.42	0.28
LF, ln; ms <sup>2</sup> /Hz	0.01 ± 0.01	0.36	0.07 ± 0.04	0.13
HF, ln; ms <sup>2</sup> /Hz	0.03 ± 0.01	0.05	0.05 ± 0.05	0.40
HF/(LF + HF)	0.01 ± 0.01	0.05	-0.01 ± 0.01	0.45
<b>Postexercise HR recovery indexes</b>				
HR <sub>max</sub> , beats/min	0.15 ± 0.13	0.22	-1.74 ± 0.37	<0.001
HR <sub>amp</sub> , beats/min	0.40 ± 0.17	<0.01	-0.99 ± 0.46	<0.001
HRR <sub>t</sub> , s	-0.01 ± 0.32	0.98	-3.62 ± 0.88	<0.001
HRR <sub>60s</sub> , beats/min	0.47 ± 0.69	0.21	0.93 ± 0.48	0.09

Values are  $\beta$ -coefficient ( $\pm$ SE) from multiple linear regressions between heart rate (HR) variability (HRV) indexes and cardiorespiratory fitness ( $\dot{V}O_{2max}$ ) and training load (Baecke sport score), adjusted on age, BMI, exercise protocol, and Baecke sport score for  $\dot{V}O_{2max}$ , and adjusted on age, BMI, exercise protocol and  $\dot{V}O_{2max}$  for Baecke sport score, in 55 middle-aged subjects. mR-R, mean R-R interval; SDNN, SD of normal R-R intervals; RMSSD, root-mean-square of successive normal R-R interval differences; pNN<sub>50</sub>, percentage of successive R-R differences greater than 50 ms; LF, low-frequency power; HF, high-frequency power; HF/(LF + HF), normalized HF power; HR<sub>max</sub>, maximal HR; HR<sub>amp</sub>, HR recovery amplitude; HRR<sub>t</sub>, HR recovery time constant; HRR<sub>60s</sub>, absolute difference between the final HR at end of exercise and the HR recorded 60 s after.

Table 3. HRV and postexercise HR indexes at different cardiorespiratory- and training load-matched levels

	Unfit <sub>LT</sub>	Unfit <sub>MT</sub>	Fit <sub>LT</sub>	Fit <sub>MT</sub>
HRV indexes				
mR-R, ms	897.81 ± 47.12	1018.57 ± 50.87	1023.11 ± 43.33	1089.21 ± 47.12*
SDNN, ms	40.52 ± 6.90	67.32 ± 7.06*	57.82 ± 6.01*	80.00 ± 6.53*†
RMSSD, ms	32.04 ± 7.70	42.03 ± 8.41*	49.99 ± 7.16*	73.45 ± 7.78*†
pNN <sub>50</sub> , %	6.27 ± 2.57	9.98 ± 2.62	12.58 ± 2.23	18.31 ± 2.42*†
LF, ln; ms <sup>2</sup> /Hz	6.22 ± 0.25	6.61 ± 0.21	6.92 ± 0.25	7.18 ± 0.24*
HF, ln; ms <sup>2</sup> /Hz	5.63 ± 0.32	6.31 ± 0.33*	6.88 ± 0.28*	7.13 ± 0.30*†
HF/(LF + HF)	0.39 ± 0.07	0.38 ± 0.06	0.55 ± 0.06*†	0.53 ± 0.06*†
Postexercise HR recovery indexes				
HR <sub>max</sub> , beats/min	188.0 ± 2.2	178.7 ± 2.7‡	187.3 ± 2.1	182.1 ± 2.4*
HR <sub>amp</sub> , beats/min	76.76 ± 3.07	75.68 ± 3.27	82.66 ± 2.85†	75.86 ± 3.41
HRR <sub>6</sub> , s	83.66 ± 6.17	63.81 ± 6.28*	77.93 ± 5.35†	58.79 ± 5.82*‡
HRR <sub>60s</sub> , beats/min	37.8 ± 3.1	40.2 ± 2.7	39.2 ± 3.2	44.1 ± 3.0

Values are adjusted means (±SE). \*Significant differences vs. Unfit<sub>LT</sub> ( $P < 0.05$ ). †Significant differences vs. Unfit<sub>MT</sub> ( $P < 0.05$ ). ‡Significant differences vs. Fit<sub>LT</sub> ( $P < 0.05$ ).

fit subjects (Fit<sub>LT</sub> and Fit<sub>MT</sub>) had the highest HRV indexes. This positive relationship between HRV and fitness is in agreement with a large number of previous studies that have linked variation in heart “period” (31), total HRV spectral power (41), or HRV triangular index (33) to  $\dot{V}O_{2\max}$  and with other cross-sectional investigations suggesting that higher cardiorespiratory fitness is associated with enhanced vagal activity (36, 46). However, in most of these previous studies, observation of HRV indexes was made without consideration of training load of the participant, so that it was not possible to determine whether the enhanced vagal-related HRV indexes were influenced by cardiorespiratory level alone. The confounding effect of training load on the fitness effect on HRV indexes is illustrated by the fact that in our study, when eliminating the effect of training, the level of significance of the fitness vs. HRV relationship decreased [remaining significant ( $P < 0.001$  vs.  $P < 0.05$ )]. Nonetheless, it is only the comparison of autonomic indexes between fit and unfit subjects with similar training loads that allows us to identify the specific link between cardiorespiratory fitness and HRV. All the sub-

jects who showed a high  $\dot{V}O_{2\max}$ , even those having a low physical activity (Fit<sub>LT</sub>), displayed high vagal-related HRV indexes. We can hypothesize that in the absence of physical activity to improve cardiovascular function, the high fitness level of these subjects, as well as their high parasympathetic background, is partly attributable to an advantageous genetic predisposition (23, 47). We can also speculate that these subjects, if engaging in physical activity, would derive greater benefit from endurance training than their sedentary Unfit<sub>LT</sub> counterparts (24).

**Cardiorespiratory fitness and HR recovery indexes.** We found no relationship between  $\dot{V}O_2$  and either HRR indexes, which contrasts with the study of Darr et al. (17). Nevertheless, because the fit athletes in their cross-sectional study were more trained than the less fit individuals to whom they were compared, training load levels may have confounded the interpretation. A recent study has also failed to show any significant relationship between postexercise HR response and  $\dot{V}O_{2\max}$  (37), and another one (12) has reported only a poor correlation ( $r = 0.15$ ,  $n = 3,446$ ) between 2-min postexercise HR recov-

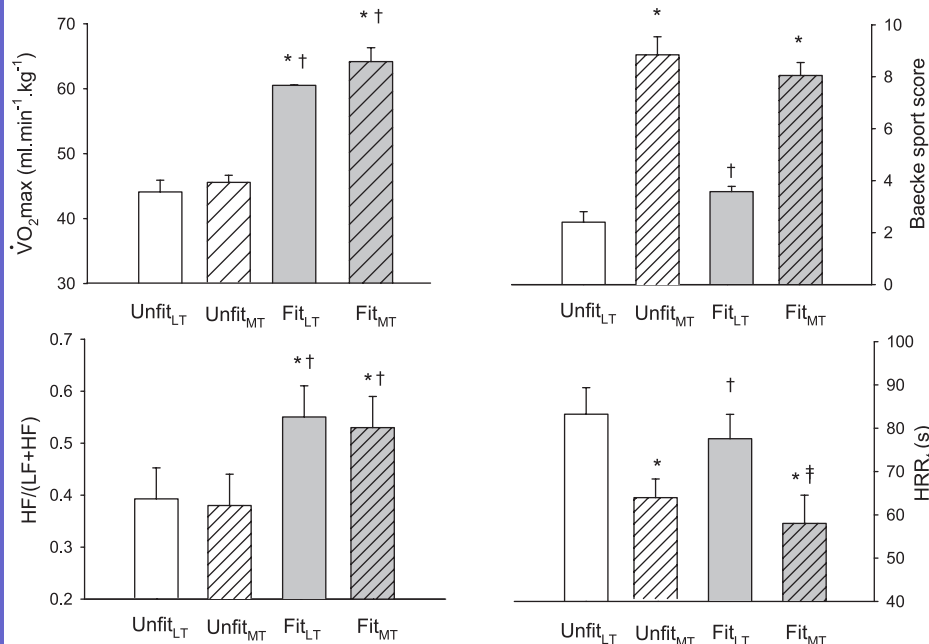


Fig. 2. Cardiorespiratory fitness ( $\dot{V}O_{2\max}$ ), training load (Baecke sport score), normalized HF power [HF/(LF + HF)], and HRR<sub>6</sub> in unfit subjects with low training load (Unfit<sub>LT</sub>;  $n = 12$ ), unfit subjects with moderate training loads (Unfit<sub>MT</sub>;  $n = 13$ ), fit subjects with low training loads (Fit<sub>LT</sub>;  $n = 11$ ), and fit subjects with moderate training loads (Fit<sub>MT</sub>;  $n = 13$ ). Values are least-squares means (±SE). \*Significant differences vs. Unfit<sub>LT</sub> ( $P < 0.05$ ). †Significant differences vs. Unfit<sub>MT</sub> ( $P < 0.05$ ). ‡Significant differences vs. Fit<sub>LT</sub> ( $P < 0.05$ ).

ery and performance on a treadmill test. We can assume that any effect of fitness on HR recovery (17) should be attributed rather to training-induced adaptations (44) that generally accompany cardiorespiratory fitness improvement than to  $\dot{V}O_{2\max}$  itself or genetically determined factors (e.g., percentage of oxidative fibers).

**Training load and HRV vagal-related indexes.** After accounting for the effect of cardiorespiratory fitness, no HRV index was significantly associated with training load. This is in opposition with many studies that have reported increased HRV levels in active individuals or trained athletes (2, 6, 12, 13). We hypothesize that the usual positive effect of aerobic training on HRV (30, 50, 52) should be attributed more to the progress of cardiorespiratory fitness resulting from the exercise program than to the increased amount of physical activity itself. This would confirm that genetic parameters known to determine  $\dot{V}O_{2\max}$  (23) influence cardiac autonomic level more than behavioral factors (47). Having both poor physical fitness and low HRV indexes despite moderate training loads ( $Unfit_{MT}$ ) may appear surprising, but it might be explained by the facts that 1) the training program of these subjects could have been insufficient to improve fitness (and consequently HRV) and 2) some of the subjects could have had genetically low HRV values, which would have, according to Hautala et al. (24), limited their ability to improve  $\dot{V}O_{2\max}$  (and HRV indexes). Nonetheless, in a clinical or health perspective, such subjects with genetically low HRV should be advised to train more frequently or to endorse more vigorous activities. We have previously shown that moderate-to-intense activities are more effective for HRV improvement than low ones, even when leading to an equivalent total energy expenditure (7).

**Training load and HR recovery indexes.** We found that Baecke sport score was significantly related to  $HRR_t$  and tended to be associated with  $HRR_{60s}$ , no matter what the fitness level. Only the moderately trained subjects ( $Unfit_{MT}$  and  $Fit_{MT}$ ) displayed fast HR recovery kinetics. The shorter recovery time constant observed in subjects with moderate training loads is consistent with literature reports. For example, it has been reported that HR recovery is significantly related to total physical activity (12), that trained athletes displayed short HR recovery times (17), and that aerobic training is associated with faster HR adjustment after exercise compared with pretraining levels (22). However, we cannot exclude that the interpretation that led to these results has been confounded by the association between fitness and training load. In the present study, it is particularly the comparison between subjects with different training loads at equivalent fitness levels ( $Unfit_{LT}$  and  $Unfit_{MT}$ ) that brings out the nonconfounded positive effect of training load on HR recovery. We can advance that the cardiovascular and muscular adaptations usually accompanying physical training [e.g., increased muscle blood flow accompanied by elevated cardiac output and increased capillarization of muscle tissue (44), alterations in the balance between sympathoadrenal acceleratory activity and vagally mediated deceleration (45), and changes in substrate utilization (44)] all develop efficiency of metabolic regulation and thus help in improving the rate of HR recovery.

**HRV vs. HRR indexes.** Our results show that HRV indexes and postexercise HR recovery may not necessarily furnish similar insight into the link between physical fitness, aerobic training, and cardiovascular autonomic function, even though

they are both vagally mediated. Because we found that changes in training load are associated more with HR recovery and that high  $\dot{V}O_{2\max}$  is better related to increased vagal related indexes, we can suggest that  $HRR_t$  as well as  $HRR_{60s}$  would constitute accurate indexes for monitoring acute or short-term changes in cardiac autonomic function and that HRV indexes would be suitable for revealing chronic cardiovascular changes. Finally, we can advance that  $HRR_t$  might better reflect individual training load than  $HRR_{60s}$  because it is only for  $HRR_t$  that significant associations were observed.

**Study limitations.** Although mean age and BMI differences among our four groups were not significant and our statistical model sought to diminish the effect of these confounding factors on HRV, it could be that the age dispersion of our subjects (and on a more modest scale, the body weight dispersion) might have affected the results. Also, as aging (54) and increased percentage of body fat (15) have been associated with decreased autonomic function, we should also consider that the contribution of poor fitness and low training load to low cardiovascular indexes may have been overestimated in the  $Unfit_{LT}$  group by the slightly higher age and BMI of the subjects. Exercise protocol was not identical (treadmill vs. bicycle testing), so that even if subjects had remained similarly seated during the recovery period and even if there were no statistical effects of exercise mode on HRR indexes, we assume that using a single ergometer would have produced less ambiguous results. It should also be noted that the lack of significant differences between groups for certain indexes with a large standard deviation (e.g.,  $HRR_{60s}$ ) may be due to small sample sizes. As we recruited only healthy middle-aged men for whom autonomic dysfunction had not been detected during the screening visit, the present findings may be representative of only a small proportion of the population in this age range. This possibility should be considered when comparing individuals in certain disease states (e.g., obesity, hypertension, etc.). Finally, the Baecke sport score, which is the index we used to measure training load, suffers from several limitations. It does not accurately take physical activity intensity into account, even though intensity has been shown to be an important determinant of the HRV response to physical activity (7). The Baecke also evaluates the intensity of an activity for the whole reported activity duration, and periods of recovery or times-out are not considered. Motion measuring devices (e.g., accelerometers) would offer a more accurate alternative for determining intensities (7, 10).

**Conclusion.** In the current study, we have investigated for the first time the relative association of cardiorespiratory fitness and training load to different indexes of cardiovascular parasympathetic modulation. It first appears that the vagal-related indexes are significantly higher in fit subjects for all training loads. Second, it seems that postexercise HR recovery is faster in subjects with moderate training loads, whatever their fitness level. The results suggest that HRV indexes are linked more to  $\dot{V}O_{2\max}$ , whereas postexercise HR recovery is rather associated with training load levels.

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