

Effects of increased training load on vagal-related indexes of heart rate variability: a novel sleep approach

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Buchheit, M., C. Simon, F. Piquard, J. Ehrhart, and G. Brandenberger. Effects of increased training load on vagal-related indexes of heart rate variability: a novel sleep approach. *Am J Physiol Heart Circ Physiol* 287: H2813–H2818, 2004. First published August 12, 2004; doi:10.1152/ajpheart.00490.2004.—There is little doubt that moderate training improves cardiac vagal activity and thus has a cardioprotective effect against lethal arrhythmias. Our purpose was to learn whether a higher training load would further increase this beneficial effect. Cardiac autonomic control was inferred from heart rate variability (HRV) and analyzed in three groups of young subjects (24.5 ± 3.0 yr) with different training states in a period free of stressful stimuli or overload. HRV was analyzed in 5-min segments during slow-wave sleep (SWS, a parasympathetic state that offers high electrocardiographic stationarity) and compared with data collected during quiet waking periods in the morning. Sleep parameters, fatigue, and stress levels checked by questionnaire were identical for all three groups with no signs of overtraining in the highly trained (HT) participants. During SWS, a significant ($P < 0.05$) increase in absolute and normalized vagal-related HRV indexes was observed in moderately trained (MT) individuals compared with sedentary (Sed) subjects; this increase did not persist in HT athletes. During waking periods, most of the absolute HRV indexes indistinctly increased in MT individuals compared with controls ($P < 0.05$) but did not increase in HT athletes. Normalized spectral HRV indexes did not change significantly among the three groups. Heart rate was similar for MT and Sed subjects but was significantly ($P < 0.05$) lower in HT athletes under both recording conditions. These results indicate that SWS discriminates the state of sympathovagal balance better than waking periods. A moderate training load is sufficient to increase vagal-related HRV indexes. However, in HT individuals, despite lower heart rate, vagal-related HRV indexes return to Sed values even in the absence of competition, fatigue, or overload.

autonomic control; endurance; high-level athletes; cardioprotective

REGULAR EXERCISE IS A KEY component of cardiovascular risk prevention strategies, because it is associated with variety of beneficial metabolic and neurovegetative effects that reduce mortality and the incidence of adverse events due to coronary heart disease (2, 3, 54). However, the optimal exercise prescription in terms of intensity and duration remains undefined. Although activities of moderate intensity are commonly accepted to improve health and lower cardiovascular risks (3, 54, 61), some studies have argued that high-intensity training may have proportionally greater cardioprotective effect (2, 15, 62). Other studies have reported that both moderate and vigorous exercise could induce similar health benefits (6, 37).

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Studies on heart rate (HR) variability (HRV) hold increasing interest because of the predictive association between decreased HRV and mortality prognostics (32, 56). High HRV and high vagal-related HRV indexes have been related to low cardiovascular risks (32, 52, 56). Evidence from animal studies supports the hypothesis that the beneficial effect of exercise training might be in part mediated by the antiarrhythmic effect of increased cardiac parasympathetic control (24, 60). In humans, most studies have associated the cardioprotective effect of training to increased vagal tone as is commonly reported in athletes or trained individuals (4, 13, 29, 50). However, this increase in vagal-related HRV indexes has not been systematically observed (9, 14, 33, 39, 49) even after an intense training period (8, 21, 59). Others have reported a conversion from vagal to sympathetic predominance with increased training load (25, 42, 43, 45, 58), but in these latter investigations, subjects were confronted with fatigue and overload (42, 43, 45, 58) or the stress of a world competition (25), which can obscure the effect of training per se. The recent study by Iwasaki et al. (26) describes the graphic relationship between exercise load and HRV as a bell-shaped relation. The authors found that a moderate dose of exercise is sufficient to achieve a substantial change in vagal modulation of the heart, and that more prolonged intense training does not necessarily lead to greater enhancement of these changes or even lead to a return of HRV indexes to pretraining values. Nevertheless, the authors do not exclude a possible overtraining effect on HRV. These results need to be confirmed in conditions free of any stressful stimuli and overload.

HRV, which is evaluated by time and frequency domain indexes, is considered to reflect the activity of the autonomic nervous system (55). The standard deviation of normal R-R intervals (SDNN) reflects global variability, whereas the root mean square of successive normal R-R interval differences (RMSSD) and high-frequency (HF) power are reasonably linked to vagal activity (35, 55). Low-frequency (LF) power is considered mainly as an index of sympathetic activity with a parasympathetic component, but some studies provide evidence that it is a poor marker of sympathetic activity (16, 23). Despite controversy (16, 23), the normalized LF/(LF + HF) and HF/(LF + HF) ratios are considered to be the more reliable markers of sympathovagal balance at rest (35, 55).

In the literature, HRV indexes have been examined under training conditions during short-term supine (21, 45, 49, 59) or standing (27) ECG recordings, during exercise (57), or during 24-h Holter recordings (7, 14, 20) with sometimes distinct

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analysis of night and day (14) or during night only (8, 42, 43). Because it is known that environmental conditions and daily physical activity affect HRV (55), diurnal recordings could possibly be more sensitive to confounding factors. Nighttime recordings may thus present great interest, because sleep constitutes a condition free of external disruptive events. However, sleep is not a uniform state, but is characterized by large variations in the sympathovagal balance throughout the non-rapid eye-movement sleep and rapid eye movement (REM) sleep cycles (10, 63) with a switch from a pronounced vagal tone during slow-wave sleep (SWS) to a sympathetic dominance during REM sleep. Sleep is associated with spontaneous electroencephalographic arousals throughout the night and is also interrupted by short- or long-term awakenings, which all evoke a prominent increase in heart rate (HR). Then the marked modifications of autonomic control that appear across the different sleep stages and the quality of sleep could mask any other influence when all-night recordings are considered. SWS, however, which is the deepest stage of sleep involved in recovery processes (22), is characterized by pronounced vagal tone (41, 63), spontaneous regular respiratory patterns, and high electrocardiographic (ECG) stationarity, and thus may present optimal conditions for HRV analysis.

The present study was designed to investigate the effects of moderate and intensive physical exercise on HRV in young subjects with a distinct training load. To ensure the absence of overload or stressful stimuli in highly trained (HT) athletes, the results on HRV were examined with regard to sleep quality and the profile of mood states (POMS; Ref. 38) questionnaire for assessing fatigue and the psycho-emotional state of the subjects. Comparisons between R-R data collected during SWS and during quiet waking periods were made to clearly establish the relevance of SWS in discriminating the state of sympathovagal balance according to training state.

METHODS

Subjects. The 31 subjects (24.5 ± 3.0 yr) who took part in the study were assembled into three groups according to their weekly training load: sedentary subjects (Sed; $n = 12$) had a Baecke sport score of <4 and displayed <2 h/wk of physical activity; moderately trained subjects (MT; $n = 10$) had a sport score between 6 and 10 and pursued 4–6 h/wk of various aerobic activity such as running or university sports; and HT subjects ($n = 9$) had a sport score of >15 and followed >18 h/wk of intensive aerobic training such as running, cycling, or triathlon training. Subjects provided their informed consent to participate in this experiment, which was approved by the local Ethics Committee. The Baecke questionnaire sport score was based on activity duration (h/wk) and frequency (mo/yr) and the intensity at which the activity is normally performed. The intensity codes are unitless and were originally based on energy costs (5).

Protocol. After the subjects received two days of rest and one night for habituation, cardiac and polygraphic sleep recordings were made during an experimental night in a soundproof, air-conditioned sleep room from 2000 to 0700, with a sampling frequency of 256 Hz using an Astro-Med EEG system (Grass Instruments; West Warwick, RI). The continuous ECG signal was obtained with a modified C₅ lead connecting the electrodes to an analog preamplifier. The R-R sequence was then extracted from the ECG signal using an automated R-R extraction algorithm (R-R Interval Software, Astro-Med EEG System). Four EEGs (F₃, C₃, P₃ vs. A₂, and C₄ vs. A₁), one chin electromyogram, and one electrooculogram were recorded. Electrodes for sleep and cardiac recordings and the sensor straps for respiratory recording were applied between 2000 and 2100. Thoracic and abdom-

inal movements were recorded using a Crystal Trace piezo respiration sensor (Astro-Med EEG System) to determine breathing frequency. Because of the strong link between respiratory rates and HRV indexes, the subject presenting a nonstationary respiratory pattern or having a spontaneous breathing frequency of <10 cycles/min (equivalent to 0.17 Hz, which is superior to 0.15 Hz, the lower HF limit) during SWS were excluded ($n = 1$). Lights were switched off from 2300 to 0700, when the subjects were awakened and asked to empty their bladders. After 20 min of rest while lying down, subjects were asked to stay supine quietly for 10 min without speaking or making any movement. Subjects breathed at a rate of 12 cycles/min (0.20 Hz) by synchronizing their breathing pattern with an electronic metronome rhythm so that the respiratory rate would influence HRV in the same way for each subject.

Sleep analysis. Polygraphic sleep recordings were visually scored at 30-s intervals using standardized criteria (47) to obtain the overnight pattern of sleep stages. In particular, SWS included *stage 3* (20–50% of slow waves) and *stage 4* (at least 50% of slow waves). All variables characterizing sleep were derived from this scoring.

HRV analysis. The first 5-min stationary segment in the first SWS episode that lasted at least 15 min was used to determine HR and HRV indexes during sleep. During quiet wakefulness in the early hours, the last stationary 5-min segment of the controlled breathing period was analyzed. The R-R intervals, i.e., the length of time between the R peaks of consecutive QRS complexes, were calculated and checked for artifacts. Occasional ectopic beats were identified and replaced with interpolative R-R interval data. The time-domain indexes such as SDNN and RMSSD were determined. After the ectopic-free data were detrended and resampled, power spectral analysis was performed sequentially with a fast Fourier transform based on a nonparametric algorithm with a Welch window. A fixed linear resampling frequency of 1,024 equally spaced points per 5-min period was used. The power densities in the LF (0.04–0.15 Hz) and the HF (0.15–0.50 Hz) bands were calculated for each 5-min spectrum by integrating the spectral power density in the respective frequency bands. The LF/(LF + HF) and HF/(LF + HF) ratios were then calculated.

Fatigue and mood assessment. Once in bed (between 2200 and 2300 before the habituation night), the subjects were asked to fill in the POMS questionnaire to assist researchers in detecting eventual mood disturbances that are commonly associated with overload (40, 44). The POMS yields a measure of mood plus intermediate scores for tension, depression, anger, fatigue, vigor, and confusion.

Statistical analysis. The normal Gaussian distribution of the data was verified by the Kolmogorov-Smirnov goodness-of-fit test. A one-way ANOVA and post hoc Tukey tests were made on normally distributed data such as sleep and mood parameters, time, and normalized spectral HRV indexes. Because absolute spectral HRV indexes (LF and HF power) were skewed, the nonparametric Kruskal-Wallis test was used to check differences between the three groups.

RESULTS

Figure 1 presents two examples of 5-min Poincaré plots in one subject with regard to the power spectrum distribution of R-R intervals during SWS and quiet wakefulness. Distinct patterns characterize the two conditions. In the plot of the awake time, the points are scattered along the $x = y$ diagonal line with high values in the LF band. During SWS, the Poincaré plot is characterized by a tighter cluster of points, and a relatively higher HF band appears in the periodogram.

Respiratory analysis. During the awake time, breathing frequency was set at 12 cycles/min (corresponding to 0.20 Hz), and during SWS, as expected (31), very regular respiratory patterns were observed. Respiratory rate tended to diminish with increased training condition, but it always remained in the HF band (17.0 ± 0.6 , 15.7 ± 0.3 , and 14.9 ± 0.8 cycles/min,

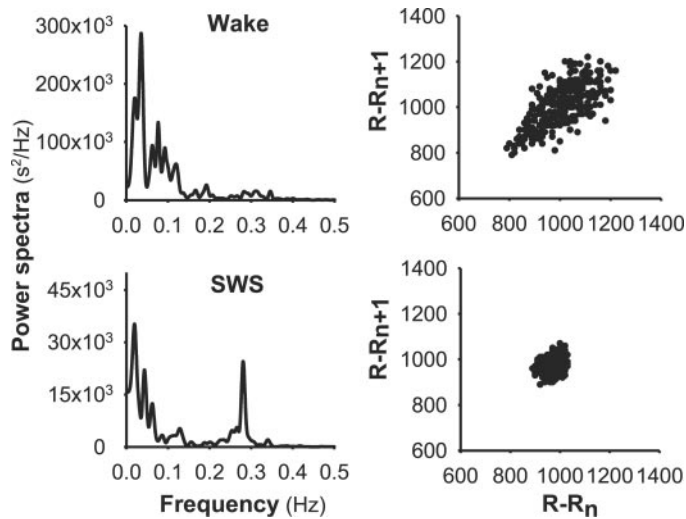


Fig. 1. Examples of 5-min Poincaré plots with regard to power spectra of R-R intervals (the length of time between the R peaks of consecutive QRS complexes) during a quiet awake period and slow-wave sleep (SWS). Note changes in y-axis scales among graphs.

equivalent to 0.28 ± 0.08 , 0.26 ± 0.09 , and 0.25 ± 0.04 Hz for Sed, MT, and HT subjects, respectively) with a significant difference between HT and Sed values ($P > 0.05$).

Absolute HRV indexes. Figure 2 illustrates the absolute HRV indexes obtained for each group for both recording conditions. During SWS, SDNN and LF power values were similar among the three groups, and only the absolute vagal-related indexes (RMSDD and HF power) were significantly higher in MT than in Sed and HT subjects. During awake time, temporal indexes (SDNN and RMSSD) were significantly higher in MT compared with Sed and HT subjects. Spectral LF and HF power values were significantly higher in MT than in HT individuals. Comparing HRV indexes during SWS and time awake revealed that all temporal and absolute spectral indexes were significantly lower during SWS than when subjects were awake in the three groups of subjects ($P < 0.005$).

HR and normalized HRV indexes. HR and normalized HRV indexes are shown in Fig. 3. During SWS, HR was significantly lower in HT athletes compared with MT and Sed subjects (45.1 ± 2.3 vs. 55.1 ± 2.1 and 55.0 ± 2.5 beats/min for HT, MT, and Sed subjects, respectively). Similarly, HR was significantly lower during quiet waking periods in HT athletes compared with MT subjects and controls (48.7 ± 2.7 vs. 53.7 ± 1.6 and 66.2 ± 5.1 beats/min for HT, MT, and Sed subjects, respectively). MT subjects also had significantly lower HR measurements than Sed individuals. The normalized HF/(LF + HF) index was significantly higher in MT compared with Sed and HT individuals during SWS, but it remained similar in the three groups when subjects were awake. Comparing HF/(LF + HF) during SWS and quiet wakefulness revealed that it was significantly ($P < 0.005$) higher during SWS for Sed and MT subjects, whereas the index remained unchanged for HT athletes.

Sleep and mood parameters. Sleep parameters, which are presented in Table 1, did not differ significantly among the three groups despite a tendency for total sleep time and sleep efficiency values to be higher for MT compared with Sed and HT individuals. Concerning the POMS questionnaire, no dif-

ferences in total mood score or in any intermediate score were noticed among the three groups of subjects (Table 2).

DISCUSSION

Several studies have reported that the cardioprotective effects of physical training might be in part mediated by the antiarrhythmic effect of increased vagal control of the heart (24, 60). Completing the conceptual background of Malliani and Montano (36), who emphasized the normalized spectral HRV indexes as a clinical tool for the investigation of autonomic activity, here we used for the first time SWS as an appropriate period of observation for revealing changes in parasympathetic activity. We found that SWS, which is characterized by high ECG stationarity, spontaneous regular respiratory patterns, and an inherent pronounced vagal tone, provides better discrimination between parasympathetic and sympathetic influences than awake periods. Our results show that the increase in vagal-related HRV indexes observed in MT subjects is not seen in HT athletes even in the absence of competition, fatigue, or overload.

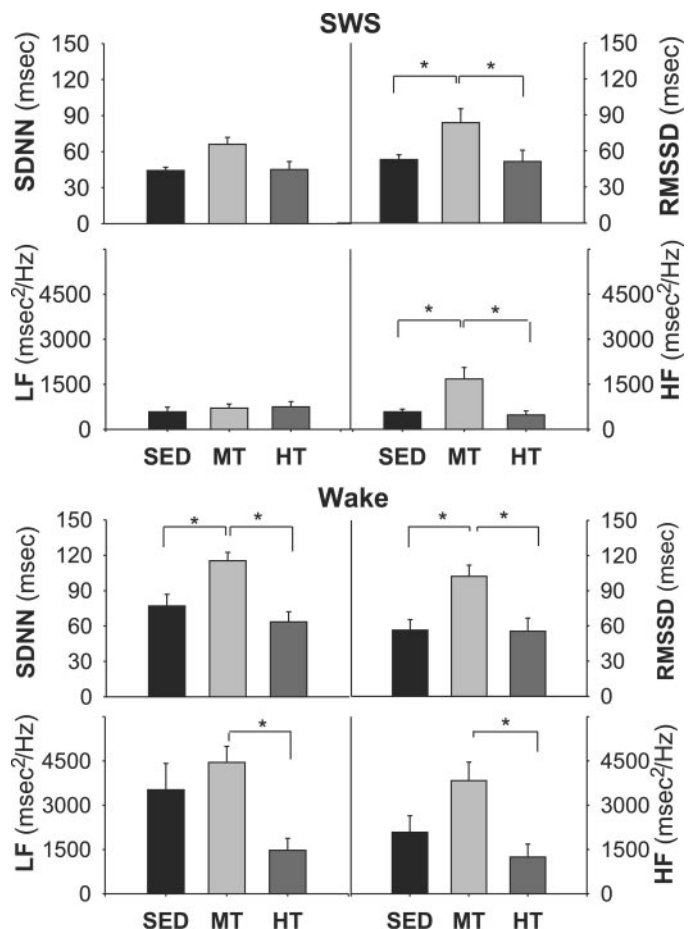


Fig. 2. Absolute heart rate variability (HRV) indexes recorded with subjects quietly awake in the morning and during SWS within the first sleep cycle in sedentary (Sed), moderately trained (MT), and highly trained (HT) subjects. SDNN, standard deviation of normal R-R intervals; RMSSD, root mean square of successive normal R-R interval differences; HF, high-frequency power; LF, low-frequency power. Values are means \pm SE. * $P < 0.05$, significant difference.

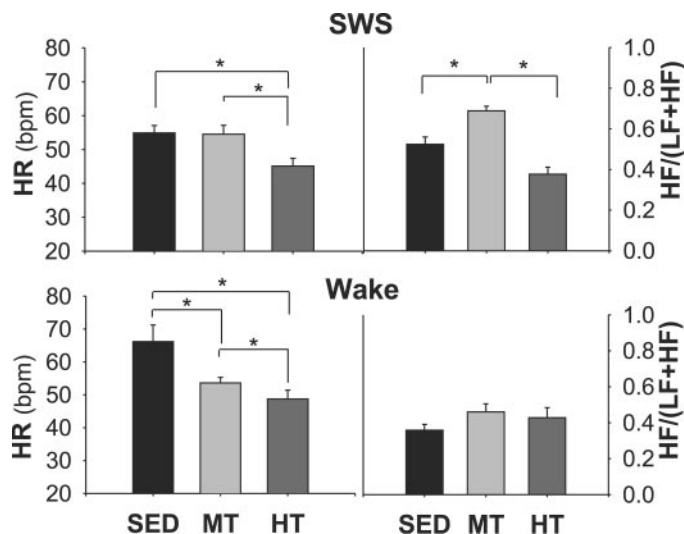


Fig. 3. HR and normalized HRV indexes [HF/(LF + HF)] recorded in subjects quietly awake in the morning and during SWS within the first sleep cycle in Sed, MT, and HT subjects. Bpm, beats/min. Values are means ± SE. *P < 0.05, significant difference.

Autonomic regulation during SWS vs. quiet wakefulness. Comparing HRV indexes from subjects in SWS vs. quietly awake revealed the importance of the R-R recording condition for assessing HRV. During SWS, subjects had lower global variability compared with the quietly awake period in the morning as inferred from lower SDNN, RMSSD, and LF and HF power values, whereas the normalized spectral indexes indicate higher parasympathetic control of the heart. Analysis of HRV indexes in three groups of subjects with different training states in SWS revealed a specific increase in both absolute and normalized vagal-related HRV indexes in MT subjects with no change in global variability (SDNN), whereas in HT subjects, all HRV indexes were similar to controls. In contrast, while subjects were awake in the early morning hours, we found that moderate training was associated with indistinct increases in most of the absolute HRV indexes, whereas the normalized indexes remained unaffected. Again, these increases did not persist in HT athletes

Table 1. Sleep parameter values

Sleep Parameter	Subjects' Activity Level		
	Sedentary	Moderately Trained	Highly Trained
Total recording period, min	480	480	480
Total sleep time, min	443.5±7.0	454.8±6.3	434.7±9.4
Sleep efficiency, %	91.9±1.2	94.1±1.1	90.3±1.9
Wake time after sleep onset, min	27.1±4.1	16.8±3.4	27.5±8.8
No. of sleep stage shifts	109.7±8.0	99.1±4.0	101.4±8.5
Sleep onset latency, min	19.3±7.0	16.1±3.3	20.3±4.9
SWS latency, min	34.2±6.9	30.4±3.8	32.2±6.0
REM sleep latency, min	103.0±11.7	90.5±7.2	94.8±14.3
Stage 2 duration, min	232.5±10.2	231.4±7.5	222.9±7.6
Stage 3 duration, min	41.5±4.1	36.8±3.8	42.6±4.7
Stage 4 duration, min	47.3±7.3	46.6±8.8	38.7±6.3
SWS duration, min	88.8±8.3	77.9±5.7	81.3±4.6
REM sleep duration, min	99.2±7.2	119.0±7.9	107.8±7.1

Values are means ± SE for sleep parameters (except time) of each group. Sleep efficiency is total sleep time divided by time in bed; SWS, slow-wave sleep; REM, rapid eye movement.

Table 2. Results of POMS questionnaire

POMS Variable	Subjects' Activity Level		
	Sedentary	Moderately Trained	Highly Trained
Tension	38.6±1.2	39.8±1.7	40.1±1.3
Depression	38.2±1.2	38.3±1.2	40.5±1.5
Anger	42.5±1.2	42.8±1.4	45.5±2.3
Vigor	58.4±1.7	57.4±3.4	58.6±1.6
Fatigue	43.7±1.6	44.5±1.3	45.6±1.4
Confusion	40.8±1.6	40.9±1.6	44.3±1.3
Total of mood states	145.5±5.1	148.8±6.5	157.4±6.1

Values are means ± SE for profile of mood states (POMS) questionnaire and include intermediate and total scores for each group.

who displayed similar HRV indexes to controls. These results demonstrate that the two recording conditions do not yield similar results in the same subjects despite careful control of both external confounding factors and breathing frequency, which largely influences HRV (12, 55). Because the low respiratory rate often seen in athletes (53) could increase LF power and thus artificially provoke a shift toward sympathetic predominance, HRV in the morning was assessed under controlled breathing (0.20 Hz) in the HF range (>0.15 Hz). SWS was characterized by spontaneous regular respiratory patterns with a mean breathing frequency of 0.25 ± 0.12 Hz in our subjects, which still remained in the HF band. Nevertheless, because breathing frequencies were not exactly similar, SWS and quiet wakefulness may be differently influenced by harmonic oscillations carrying over from slower rhythms, which may somewhat confound the comparison between SWS and awake periods.

Absolute vs. normalized HRV indexes. Our data indicate that conclusions about the influence of training on HRV may be in part dependent on the moment of observation and might explain some of the discrepancies among results reported in the literature. These inconsistencies also raise the question of the relevance of absolute HRV indexes vs. normalized spectral indexes. For example, studies on mortality prognostics and decreased HRV in patients or older persons concern mainly global variability (32, 52, 56). In contrast, as global HRV has been shown to vary markedly between subjects with identical physical capacity (30), the normalized indexes, which are independent of overall HRV level, may confer substantial interest in expressing the state of the sympathovagal balance (35). Thus in the present state of our knowledge, it appears that both absolute and normalized indexes have to be considered. We therefore propose that both SWS and a quiet awake period at the end of the night may be used to provide distinct but complementary information for investigating the effects of training on HRV: the awake period will yield more information about global HRV, whereas SWS is more likely to reveal the differences in vagal-related indexes and sympathovagal balance. Nevertheless, the relevance of SWS as an optimal period of observation for discriminating sympathovagal balance must be confirmed in other experimental situations, and simplified methods must be proposed for identifying SWS episodes on the basis of ECG recordings without the systematic need of polygraphic sleep recordings.

Effects of physical training on HRV. The main result of our study is that the increase in vagal-related HRV indexes observed in MT subjects is reversed in HT athletes who present similar HRV indexes as Sed controls. This agrees with a large

number of studies that generally report an increase in vagal tone with moderate training (4, 13, 29, 50). Some authors, however, did not observe this training effect (9, 14, 33, 39, 49), but these inconsistencies could mainly be related to insufficient training load (9, 14, 39). Then the small training effect has to overlap large interindividual HRV fluctuations; for example, there is no doubt about the strong effect of aging, which largely reduces global HRV and enhances normalized sympathetic indexes (11, 46). No cross-sectional studies have investigated the effects of high training load on HRV in conditions free of overload. Longitudinal studies during competition (25) or heavy training (42, 43, 45, 58) have reported a conversion from vagal to sympathetic dominance in athletes, whereas others failed to demonstrate any HRV modifications after an increased training load (8, 21, 59). However, competition or training overload could mask the effects of training per se. Therefore, in our study, care has been taken to minimize any stressful stimuli that might cause a shift from vagal activity to sympathetic predominance. In particular, the HT participants were not engaged in any competition, had suspended their usual training two days before the recordings, and did not show any sign of overtraining (mood disturbance or poor sleep quality), which have been reported to influence HRV indexes (13). Concerning the POMS, compared with Sed and MT individuals, no differences in mood or fatigue level were noticed in the HT subjects. Moreover, the sleep parameters did not show any alteration that could reveal a state of stress or overtraining. Our results in non overtrained athletes are in accordance with longitudinal investigations that report decreased vagal tone after training (1, 48), no difference in trained subjects compared with control subjects even after a sufficient training load of 5 mo (33), and higher vagal activity in detrained athletes (7). The bell-shaped graphic relationship between exercise load and HRV described by Iwasaki et al. (26) could then explain the return of HRV indexes to basal values when training is prolonged, which then leads to equivalent HRV indexes for athletes and Sed subjects.

HR and HRV relationships. Although the significantly lower HR observed in the HT subjects could be in part attributed to an alteration of the intrinsic HR consecutive to left ventricular remodeling (29), resting bradycardia has mainly been related to enhanced vagal tone (28, 51). Low HR with decreased vagal-related HRV indexes seems to reveal the limitation of HRV as a marker of cardiac vagal control when vagal activity is already high. Explanations have been advanced including loss of phasic vagal efferent discharge at high levels of vagal activity or saturation of acetylcholine receptors (18, 19, 34). Alternatively, the reduced sympathetic discharge reported in athletes (17) may have also reduced both HR and HRV (55). Understanding the mechanisms that underlie the return of vagal-related HRV indexes to Sed control levels in HT athletes requires additional research.

Limitations. Although we may advance that moderate physical training induces enhancement of vagal-related indexes, it can also be hypothesized that persons with genetically higher HRV indexes may be more predisposed to participate in physical activities. Thus care should be taken in determining whether exercise participation is the cause or the consequence of high HRV. Second, as our subjects were selected on a quantitative global training dose basis, the respective effects of exercise intensity and duration on HRV cannot be clearly

distinguished, which limits our interpretations to the effects on HRV of only the global training load.

Health implications. The optimal weekly training load in terms of intensity and duration of exercise for cardiovascular disease prevention is still a matter of debate. Surveys for maintaining cardiorespiratory fitness recommend either exercise of moderate intensity and long duration (3, 54) or shorter, more vigorous exercise (2, 62). Some studies suggest that subjects could receive greater cardioprotective benefit from sport activities than from a high level of routine physical activity (15), but an important all-day activity level (61) or brisk walking (37) have also been shown to reduce cardiovascular risks. The present results suggest that a moderate amount of exercise is sufficient to enhance vagal-related HRV indexes, and that higher training load does not cause additional increases despite a lower HR. It is tempting, then, to propose a moderate but not too high training load for promoting health; however, this recommendation needs to be supported by additional studies.

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