Effect of athletic training on heart rate variability

The time and frequency domain components of heart rate variability have been used to assess prognosis in patients with different types of heart disease. However, the effect of habitual exercise, which influences baseline parasympathetic tone, on heart rate variability has not been fully evaluated. To determine the effect of chronic exercise on heart rate variability, we studied 12 athletes and 18 control subjects. Time domain and frequency domain analysis was performed on 15-minute resting heart rate acquisitions. Athletes had evidence of increased vagal activity in the time domain compared with control subjects (eg, increased standard deviation of R-R intervals) but showed evidence of decreased power in variables reflecting vagal activity in the frequency domain (eg, total power and high-frequency power). Of note, there was good correlation between time and frequency domain variables, which reflected parasympathetic tone in the control group that was not seen in athletes. These data suggest that frequency domain analysis of heart rate variability may not be an accurate indicator of cardiac vagal tone in chronically trained endurance athletes and activity level may have to be considered when using heart rate variability to carry out prognostic stratification in patients with heart disease. (AM HEART J 1994;127:1275-8.)

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Heart rate variability is a simple, noninvasive technique that provides an index of cardiac autonomic tone through the measurement of instantaneous beat-to-beat variations in R-R interval length. Low heart rate variability, which probably results from increased sympathetic tone and diminished parasympathetic tone,[1] has been associated with increased mortality after myocardial infarction.[1-5] Other potential uses of heart rate variability include evaluation of patients with congestive heart failure, patients with ventricular arrhythmias, and survivors of sudden cardiac death.[5-12]

Trained endurance athletes have been noted to have profound bradycardia, which probably results from an increase in cardiac vagal tone.[13-15] Given this fact, it is possible that training may affect indexes of heart rate variability. If this is true, exercise level would have to be considered as a factor when using heart rate variability to evaluate patients with disease. This study was performed to determine whether the baseline values of a trained normal athlete vary from those of a sedentary person. We hypothesized that well-trained endurance athletes would have measurements of heart rate variability that would reflect increased parasympathetic tone when compared with untrained control subjects.

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Received for publication May 28, 1993; accepted Sept. 3, 1993.

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0002-8703/94/$3.00 + 0 **4/1/53385**

METHODS

Thirty healthy subjects were studied. Twelve were highly trained endurance athletes (nine men and three women; mean age 26 ± 1.6 years) who performed a minimum of 3 hours of aerobic activity per week (either bicycling or running or both) and had resting heart rates less than 60 beats/min. These subjects were compared with a control group of 18 untrained persons (17 men and one woman; mean age 30 ± 0.3 years).

Heart rate variability was measured by means of a Corozonix PREDICTOR ECG software package. All testing was performed between 8 and 10 AM, and subjects were requested to refrain from meals or caffeine for 12 hours before testing. R-R interval data were collected by using 15-minute measurements of supine resting heart rate obtained with standard orthogonal ECG monitoring leads. The raw R-R interval measurements were then edited such that any complexes not fitting the QRS template were removed from the calculations. Measurements of heart rate variability in both the time domain and frequency domain were calculated. In the time domain, measurements included average R-R interval, standard deviation of the R-R interval, per-

Table I. Heart rate variability parameters: Time domain

	Athletes	*Controls*	*p Value*
Mean R-R interval (msec)	1,227 (43)	890 (22)	<0.01
SDRR	92 (8)	73 (9)	<0.05*
pNN 50	45 (4)	25 (5)	<0.05
Coefficient of variation	7 (1)	8 (1)	NS

pNN 50, Percentage of R-R intervals >50 msec; *SDRR*, standard deviation of R-R interval; *NS*, not significant.
*Common log transformation.

Table II. Heart rate variability parameters: Frequency domain

	Athletes	*Controls*	*p Value*
Total power spectrum	8,198 (1,410)	17,196 (3,379)	<0.02*
Ultralow frequency (<0.04 Hz)	4,550 (1,064)	7,519 (1,725)	0.055*
Low frequency (0.04 to 0.1 Hz)	1,478 (275)	2,620 (363)	<0.05*
High frequency (0.11 to 0.4 Hz)	2,022 (240)	5,839 (1,839)	<0.02*

*Common log transformation.

centage of R-R intervals greater than 50 msec (pNN 50), and the coefficient of variation.

The power spectral plot of the heart rate variability was derived from the interval tachogram by means of a 2 Hz resampling algorithm described by Berger et al.[16] The resulting data were then passed through a Hanning window, and the fast Fourier transformation was computed with the data being smoothed once (spectral analysis performed by PREDICTOR Heart Rate Variability ECG software package). The power spectrum of three different frequency ranges was calculated. These were defined as ultralow (<0.04 Hz), low (.04 to 0.1 Hz), and high (0.1 to 0.4 Hz). The total power spectrum was also calculated.

Statistics. Comparisons between groups were then made by independent *t* tests. A logarithmic transformation of the data was performed in cases where there was a nonnormal distribution of the data. Data are presented as mean ± standard error of the mean.

RESULTS

Athletes had a significantly lower resting heart rate than control subjects (49.5 ± 1.6 vs 64.1 ± 3.6 beats/min, $p < 0.005$). This was reflected in the average R-R interval length, which was significantly longer in athletes. Time domain analysis (Table I) also showed that the standard deviation of R-R intervals was significantly greater in athletes as compared with control subjects (92.3 ± 7.7 vs 73 ± 9.3, $p < 0.05$), as was the percentage of successive R-R intervals greater than 50 msec (pNN 50) (45 ± 3.9 vs 25.3 ± 5.5, $p < 0.05$). The coefficient of variation was slightly larger in the control group, but this was not statistically significant.

Table III. Correlations between time domain and frequency domain variables: Control subjects

Frequency	*SDRR*	*p Value*	*pNN 50*	*p Value*
Total	0.86	0.001	0.76	0.001
Ultralow	0.33	NS	0.40	NS
Low	0.33	NS	0.22	NS
High	0.79	0.001	0.65	0.001

pNN 50, Percentage of R-R intervals >50 msec; *SDRR*, standard deviation of R-R interval; *NS*, not significant.

Table IV. Correlations between time domain and frequency domain variables: Athletes

Frequency	*SDRR*	*p Value*	*pNN 50*	*p Value*
Total	0.45	NS	−0.18	NS
Ultralow	0.53	NS	−0.095	NS
Low	0.42	NS	−0.34	NS
High	−0.15	NS	−0.17	NS

pNN 50, Percentage of R-R intervals >50 msec; *SDRR*, standard deviation of R-R interval; *NS*, not significant.

Analysis of the frequency domain variables (Table II) revealed that athletes had significantly less total power (8198 ± 1410 vs 17196 ± 3379, $p < 0.05$) when compared with control subjects. The power spectral density of the high-frequency (0.1 to 0.4 Hz.) and low-frequency (0.04 to 0.1 Hz) areas was also significantly less in athletes compared with control subjects. The ultralow-frequency (<0.04 Hz) power spectrum was less in athletes, but this did not reach statistical significance.

Correlations between time and frequency domain variables in the control group demonstrated that total power and high-frequency power showed significant correlation with the standard deviation of the R-R interval and the pNN 50 (Table III). No such relationships existed in the athletes (Table IV).

DISCUSSION

Analysis of heart rate variability is thought to permit differentiation of the autonomic tone of the heart into sympathetic and parasympathetic influences.[4-6, 10-12, 17-21] In particular, activity in the high-frequency component of the power spectrum (>0.1 Hz) has been correlated with parasympathetic activity. For example, pharmacologic blockade with atropine completely abolishes the high-frequency component of the heart rate power spectrum.[17-21] Endurance-trained athletes with a low resting heart rate would be expected to have heart rate variability findings that reflect increased parasympathetic activity. The results of this study show that endurance-trained athletes have indications of increased parasympathetic tone in the time domain analysis but not the frequency domain. Specifically athletes had di-

minished high-frequency activity compared with control subjects. In addition, we found a poor correlation between parameters that reflect parasympathetic tone in the time and frequency domain analyses in athletes but a strong correlation in control subjects.

There are two possible explanations for this finding. First, power spectral analysis of heart rate variability may not accurately reflect underlying autonomic tone in all patient populations.[21a] Autonomic tone in all areas (high, low, and ultralow) of the frequency power spectrum, as well as total power, was lower in athletes. This may represent a methodologic problem with the use of the technique of power spectral analysis for these subjects. The plot of instantaneous heart rate versus time describes a wave. Spectral analysis by means of fast Fourier transformation is a technique in which the periodic oscillations in R-R interval length are described as waves of discreet frequencies. However, if the R-R interval length is already close to maximally long, the corresponding changes in the R-R cycle length can only change in one direction, potentially limiting the amplitude of the resultant power spectrum. This would explain the uniformly lower values across the entire heart rate power spectrum and the decrease in total power found in the athletes.

Results of the time domain analysis may support this theory. Previous studies have shown that respiratory sinus arrhythmia is an accurate indicator of cardiac vagal tone.[22-24] The present study shows that measurements in the time domain analysis, essentially respiratory sinus arrhythmia, were significantly greater in the athletes. This includes the pNN 50, which some investigators consider the most accurate indicator of cardiac vagal tone.[25]

The results of this study show that the control group had a significant correlation between variables reflecting increased vagal tone in the time domain (increased standard deviation of R-R intervals and pNN 50) and the frequency domain (increased total power and high-frequency power). In contrast, the athletes had evidence of increased vagal tone from the time domain analysis but no significant correlation with the variables in the frequency domain, which reflect parasympathetic activity. Correlations between time and frequency domain variables have been described in other studies.[19, 25-26] Notably the standard deviation of R-R intervals has been reported as equivalent to total power, and the high-frequency power spectrum has been shown to correlate with the standard deviation of the R-R interval. The results of this study confirm these correlations in the control group but not in the athletes. This finding of evidence of increased vagal tone in the time domain, which does not correlate with evidence of increased vagal tone in the frequency domain variables in athletes may be further evidence of the inaccuracy of power spectral analysis to assess autonomic tone in a highly trained population.

An alternative explanation for the diminished high-frequency power spectral density in athletes is that the resting bradycardia of aerobically fit subjects may not be due to an increase in cardiac vagal tone. Maciel et al.[27] measured respiratory sinus arrhythmia in seven subjects both before and after aerobic training. Despite an increase in maximum oxygen consumption and a decrease in resting heart rate, there was no change in respiratory sinus arrhythmia. Similarly Elkblom et al.[28] studied autonomic tone by means of pharmacologic blocking of parasympathetic outflow with atropine and sympathetic outflow with propanolol in 14 subjects both before and after aerobic training. They found that autonomic activity was similar both before and after training. These investigators concluded that the resting bradycardia of athletes may be a result of a combination of reduced β-adrenergic receptor activity and an increase in parasympathetic activity.

In contrast to our results, Goldsmith et al.[29] recently reported that parasympathetic activity in both the time and frequency domains was greater in trained than in untrained men. That study is not comparable with the present study, however, inasmuch as the analysis was performed with a 24-hour recording (as opposed to our 15-minute recording). In addition, in the Goldsmith study[29] the mean R-R interval was subtracted from the sampled night and day data, a statistical treatment not used in our investigation.

Clinical significance. The significance of these findings lies in the application of heart rate variability analysis for stratifying patients. Prior stratification studies have not taken activity level into consideration. Our results indicate that trained athletes have baseline characteristics different from those of sedentary persons, with low activity in the high-frequency area, which may not reflect low vagal tone. The implications of this finding when heart rate variability is used as a prognostic tool have not been studied. For example, evidence of low parasympathetic tone in the power spectrum of a well-trained patient who is post myocardial infarction may have different significance from the same finding in a sedentary patient.

The clinical significance of this study may be affected by the fact that the athletes and control subjects were young (mean age 26 and 30 years, respectively). Heart rate variability is affected by aging,[30] and the results of the present study should be confirmed in an older population before being applied to such patients. Another consideration is that

the study assessed the effect of a dynamic form of exercise (running or cycling). Further work is needed to assess the effect of primarily static exercise, such as weight lifting, on heart rate variability. Finally, the heart rate variability studies were performed under baseline conditions. Whether provocative autonomic stimulation (such as deep breathing or tilt)[30] affects heart rate variability differently in athletes compared with control subjects is another area for further investigation.

Conclusion. Heart rate variability is affected by chronic exercise. Activity status may have to be evaluated when power spectrum analysis is used for prognostic stratification. The data show no influence of training on time domain analysis, which may continue to be used to evaluate patients regardless of training level. Additional studies are needed to evaluate the clinical impact of these findings and to clarify issues surrounding power spectral analysis such as standardization of data handling and acquisition.

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