

Reduced Room for Cardiac Vagal Modulation to Increase and Cardiac Sympathetic Modulation to Decrease by Resting in Football Players

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Abstract

Background: This study investigated the cardiac autonomic nervous modulation of football (soccer) players at rest, and whether resting can change it further.

Methods: The stationary state spectral heart rate variability (HRV) measures at rest and 60 and 90 min after baseline measurement were compared between football players and sedentary controls. The percentages of change in HRV measures 60 and 90 min after baseline measurement were also compared between football players and sedentary controls.

Results: Seventeen football players and 17 sedentary normal subjects were recruited in this study. The total power (TP), very low frequency power (VLFP), low frequency power (LFP), high-frequency power (HFP) and normalized high-frequency power (nHFP) of football players at rest were all significantly higher, while both normalized low-frequency power (nLFP) and low-/high-frequency power ratio (LHR) of football players at rest were significantly lower than those of normal controls. Sixty and 90 min after baseline measurement, though the heart rate (HR) was significantly decreased and the standard deviation of RR intervals (SD_{RR}), root mean squared successive difference ($rMSSD$), TP, VLFP, and HFP were all significantly increased in the control group, there were no significant changes in the HRV measures except that the HR was slightly decreased in the football players.

Conclusion: The football players have increased vagal modulation and decreased sympathetic modulation at rest as compared to sedentary non-athletes. Although vagal modulation can be increased significantly by resting in the sedentary non-athlete, it cannot be increased significantly in the football players. The football players at rest seem to be already in a relatively relaxed state which cannot be relaxed further by further resting, whereas the non-athletes are not relaxed fully at rest and more rest can lead to further relaxation. The room for cardiac vagal modulation to increase and cardiac sympathetic modulation to decrease by resting is reduced in the football players relative to that of normal subjects.

Keywords: Football; Sports; Heart rate variability; Autonomic nervous system

Introduction

Heart rate variability (HRV) refers to the fluctuation in the time intervals between successive heart beats. Power spectrum analysis of heart rate fluctuations provides a quantitative noninvasive means to assess the sympathetic and parasympathetic modulation of a subject [1]. It is already known that exercise can influence the HRV of the subjects. For instance, Jensen-Urstad et al. [2] reported that the runners had higher HRV in all spectral bands; the pNN50 and root mean squared successive difference ($rMSSD$) in the time domain can reflect the vagal tone and pronounced resting bradycardia in male elite runners which is associated with high HRV. Aubert et al. [3] suggested that HRV is affected by chronic exercise, especially in endurance trained athletes, suggesting that aerobic exercising can have beneficial effects on the cardiovascular risk profile. Melo et al. [4] pointed out that aging reduces HRV, and regular physical activity positively affects vagal activity on the heart and consequently attenuates the effects of

aging in the autonomic control of heart rate (HR). Lu and Kuo [5] demonstrated that short-term exercises can enhance vagal modulation, lower sympathetic modulation and arterial blood pressures in the Wai Tan Kung and Tai Chi Chuan practitioners. Amano et al. [6] showed that 12-week exercise training has significantly improved both the sympathetic and parasympathetic nervous activities of the obese individuals with markedly reduced autonomic nervous system activity. One study also indicated that endurance training results in enhanced vagal activities in athletes, and contributes in part to the resting bradycardia [7].

Football (soccer) is one of the most popular sports in the world; its popularity has massive financial implications, especially when considering professional football [8]. Although football sport is beneficial to the health, few studies have dealt with the autonomic nervous modulation of football players. This study intended to understand the autonomic nervous modulation of the football players relative to ordinary people at rest and the effect of resting on the autonomic nervous modulation of the football players by using HRV analysis.

Methods

Study subjects

Both football players and healthy sedentary subjects were included in this study. The football players were recruited from a nearby football club, and the healthy sedentary subjects were recruited from the community. Before the study, the healthy volunteers were asked whether they had exercise habits of any kind. The answer must be “no” except daily life activities before they were included in the study. The Institute Review Board of the hospital has approved this study. The procedure was fully explained to the subjects, and written informed consent was obtained from them before the study.

Instrument and measurement

The ECG signals of the subjects were recorded by using a multichannel recorder (Biopac MP150 with 16 channels, BIOPAC Systems, Inc., Goleta, CA, USA) from conventional lead II, and the blood pressure was measured by using an automatic blood pressure monitor (00615-MR3 Omron, Omron Healthcare Taiwan Co., Taipei, Taiwan) on each subject. The analog signals of ECG were transformed into digital signals by using an analog-to-digital converter with a sampling rate of 500 Hz. The systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MABP), and pulse pressure (PP) were obtained from each subject by using the sphygmomanometer. Spirometry (IQmark Digital ECG and Digital Spirometer, Brentwood Medical Technology Corp., Torrance, CA) were performed on each subject. Subject who had major cardiopulmonary disease or was on regular medicine for diabetes mellitus, hypertension, renal or liver disease was excluded from the study.

Study protocol

The subject included in this study was instructed not to take caffeinated or alcoholic beverages 24 hours prior to study. Each subject rested in the supine position for 5 min, and then a train of continuous electrocardiographic (ECG) signals was recorded for 15 min. After baseline ECG recording, the blood pressures and lung functions were measured. The ECG recording and blood pressure measurements were repeated 60 and 90 min after the baseline measurement in both groups of subjects. All procedures were performed in the afternoon in a bright and quiet room with a room temperature of 24 to 25°C and humidity of 54 to 55%.

HRV analysis

The R-wave detecting software was developed with the help of Matlab R13 software (MathWorks Inc., Natick, MA) and used to identify the peaks of the R waves in the ECG signals. The RR intervals (RRI) were calculated after eliminating the ectopic beats. If the percentage of ectopic beats were greater than 5%, then the subject was excluded from analysis. The last 512 stationary RRI were used for HRV analysis.

The mean (mRRI), standard deviation (SD_{RR}), coefficient of variation (CV_{RR}) and rMSSD of RRI were calculated using standard formula. The power spectra of the 512 RRI were obtained by means of fast Fourier transformation (Mathcad 13, Mathsoft Inc., Cambridge, MA). Direct current component was excluded before the calculation of the powers. The area-under-the-curve of the spectral peaks within the

range of 0.01-0.4 Hz, 0.01-0.04 Hz, 0.04-0.15 Hz, and 0.15-0.40 Hz were defined as the total power (TP), very low-frequency power (VLFP), low-frequency power (LFP), and high-frequency power (HFP), respectively.

Baseline characteristics	Control (n=17)	Football (n=17)	p value
Gender (M/F)	8/9	7/10	NS
Age (year)	23 ± 3	24 ± 5	NS
Height (cm)	168 ± 5	170 ± 7	NS
Weight (kg)	65 ± 15	66 ± 9	NS
BMI (kg/m ²)	23 ± 4	23 ± 3	NS
FVC (L)	3.96 ± 9.45	4.30 ± 0.84	NS
%FVC (%)	101 ± 13	106 ± 10	NS
FEV ₁ /FVC (%)	91 ± 5	92 ± 6	NS
FEV ₁ (L)	3.57 ± 0.64	3.95 ± 0.70	NS
%FEV ₁ (%)	107 ± 12	116 ± 12	0.046

Table 1: Baseline characteristics of normal controls and football players. All data are mean ± standard deviation. NS: Not Significant; BMI: Body Mass Index; FVC: Forced Vital Capacity; %FVC: FVC percent predicted; FEV₁: Forced Vital Capacity in the First Second; % FEV₁: FEV₁ percent predicted; FEV₁/FVC: Ratio of FEV₁ to FVC.

The Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology [9] has suggested that the power within the frequency range of 0.04-0.4 Hz be used for the normalization of LFP and HFP. Since this frequency range does not cover the frequency ranges of VLFP, it may not be suitable for the normalization of VLFP. Therefore, we used the power within the frequency range of 0.01-0.4 Hz to normalize VLFP, LFP and HFP in this study. The normalized VLFP ($nVLFP=VLFP/TP$) was used as the index of vagal withdrawal, renin-angiotensin modulation and thermoregulation [10-12], the normalized LFP ($nLFP=LFP/TP$) as the index of combined sympathetic and vagal modulation [13], the normalized HFP ($nHFP=HFP/TP$) as the index of vagal modulation, and the low-/high-frequency power ratio ($LHR=LFP/HFP$) as the index of sympathovagal balance [14].

Data analysis

The Mann-Whitney rank sum test (SigmaStat statistical software, SPSS Inc., Chicago, Illinois, USA) was employed to compare the baseline characteristics, hemodynamic data and HRV measures between football players and normal controls. Friedman repeated measures ANOVA on ranks was utilized to compare hemodynamic and HRV measure among the baseline measurement and 60 minutes and 90 minutes after baseline measurement in football group. Significant difference was further analyzed by pairwise comparison using the Student-Newman Keuls test. A $p<0.05$ was considered statistically significant. All data are presented as median (25% ~75%).

To quantify the effect of rest on the autonomic nervous modulation of the football players, the percentage changes in the HRV measures and blood pressures 60 and 90 minutes after the baseline measurement were calculated using the following formulae:

$$\%X_{0-60} = [(X_{60\text{min}} - X_{\text{baseline}}) / (X_{\text{baseline}})]$$

$$\%X_{0-90} = [(X_{90\text{min}} - X_{\text{baseline}}) / (X_{\text{baseline}})]$$

where "X" is the parameter to be evaluated.

Results

Baseline characteristics

Seventeen football players and 17 sedentary controls aged between 19 to 29 years were included in this study. The football players have practiced football for at least 1 hour each time, 3 times per week for 1-10 years. Table 1 tabulates the baseline characteristics of the control and football groups. There were no significant differences in the baseline characteristics between normal controls and football players except that the football players have significantly greater percent predicted FEV₁ (%FEV₁) than the normal controls.

Sequential change in blood pressures and HRV measures

Figure 1 shows the sequential change in blood pressures in both groups of subjects. The MABP 90 min after baseline measurement was significantly larger than that of 60 min after baseline measurement in the control group. In the football group, the baseline PP was significantly larger than that of the control group. Also, the DBP and MABP after a rest for 90 min were significantly larger than those of baseline measurement and 60 min after baseline measurement in the football group.

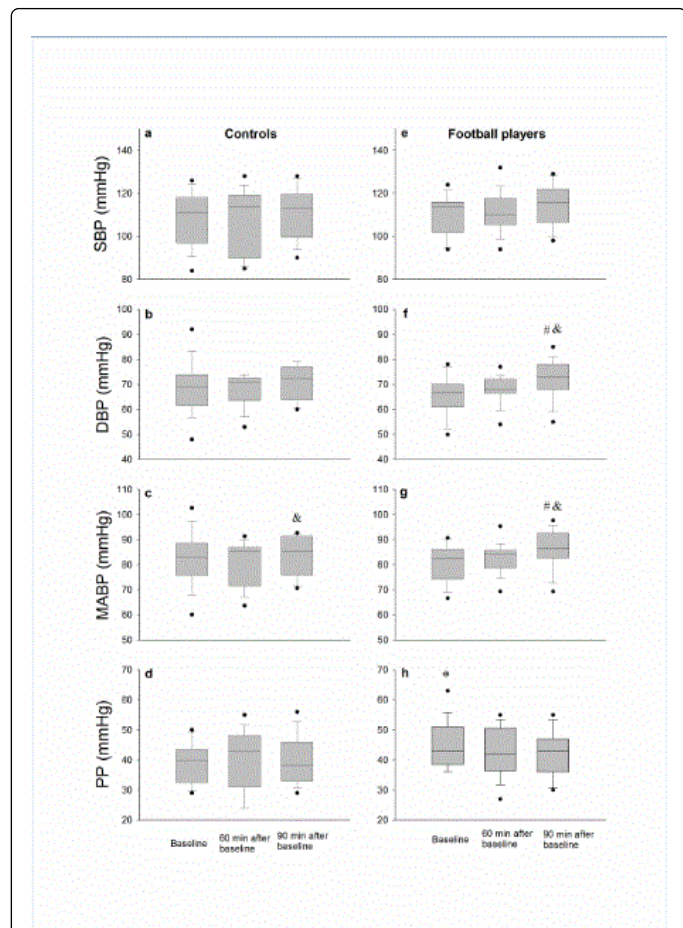


Figure 1: Sequential change in blood pressures of the control subjects and football players at rest. Data are presented as medians (25%~75%). #p<0.05 vs. baseline measurement; and p<0.05 vs. 60 minutes after baseline measurement; *p<0.05 vs. counterpart of the control (Friedman repeated measure ANOVA on ranks with post hoc Student-Newman-Keuls test). (a), (e) Systolic blood pressure (SBP); (b), (f) Diastolic blood pressure (DBP); (c), (g) Mean arterial blood pressure (MABP); (d), (h) Pulse pressure (PP).

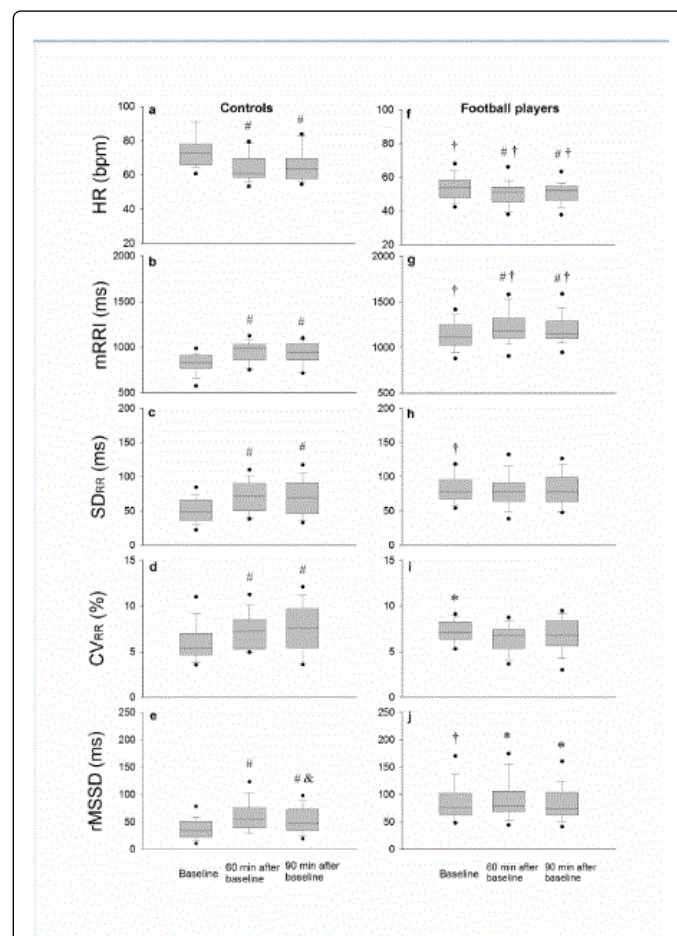


Figure 2: Sequential change in time domain HRV measures of the control subjects and football players at rest. BPM: Beats Per Minute; MS: Millisecond. Data are presented as medians (25%~75%). #p<0.05 vs. baseline measurement; *p<0.05 vs. counterpart of the control; †p<0.01 vs. counterpart of the control (Friedman repeated measure ANOVA on ranks with post hoc Student-Newman-Keuls test). (a), (f) Heart rate (HR); (b), (g) Mean RR interval (mRRI); (c), (h) Standard deviation of RR intervals (SD_{RR}); (d), (i) Coefficient of variation of RR intervals (CV_{RR}); (e), (j) Root mean squared successive difference (rMSSD).

Figure 2 shows the sequential change in time domain HRV measures in both groups of subjects. After a rest for 60 to 90 min, the HR was significantly decreased while the mRRI, SD_{RR}, CV_{RR}, and rMSSD were all significantly increased in the control group. In the football group, the baseline mRRI, SD_{RR}, CV_{RR}, and rMSSD were all

significantly larger, whereas the baseline HR was significantly smaller, than their counterparts in the control group. After a rest for 60 to 90 min in the football group, there were no significant changes in SD_{RR} , CV_{RR} , and $rMSSD$ except that the HR was significantly smaller and $mRRI$ was significantly larger than their counterparts in the control group. Within group comparison in the football group showed that a rest for 60 to 90 min could lead to a significant decrease in HR and a slight increase in $mRRI$ only, as compared to those in the baseline measurement. The $rMSSD$ after a rest for 60 to 90 min was significantly increased as compared with its counterpart in the control group.

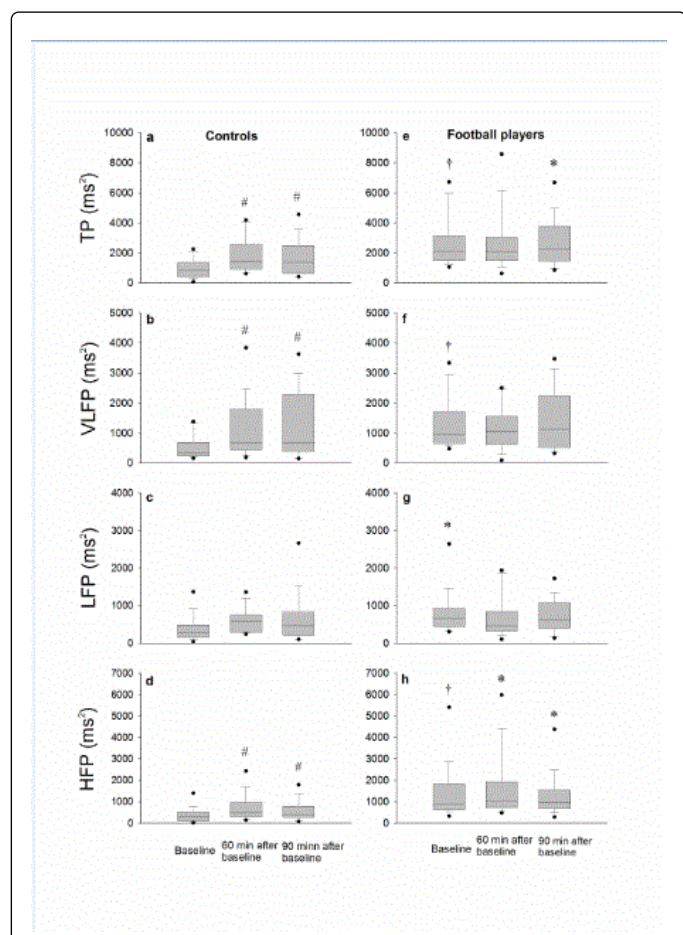


Figure 3: Sequential change in frequency domain of heart rate variability measures of the control subjects and football players at rest. Data are presented as medians (25%~75%). # $p < 0.05$ vs. baseline measurement; * $p < 0.05$ vs. counterpart of the control; † $p < 0.01$ vs. counterpart of the control (Friedman repeated measure ANOVA on ranks with post hoc Student-Newman-Keuls test). (a), (e) Total power (TP); (b), (f) Very low-frequency power (VLFP); (c), (g) Low-frequency power (LFP); (d), (h) High-frequency power (HFP).

Figure 3 shows the sequential change in the absolute powers of the frequency domain HRV measures in both groups of subjects. After a rest for 60 to 90 min in the control group, the TP, VLFP, and HFP were all significantly larger than those of the baseline measurement. In the football group, the baseline TP, VLFP, LFP, and HFP were all significantly larger than their counterparts in the control group. In

addition, the TP after a rest for 90 min and the HFP after a rest for 60 to 90 min were all significantly larger than their counterparts in the control group.

Figure 4 shows the sequential change in the relative powers of the frequency domain HRV measures in both groups of subjects. After a rest for 60 to 90 min, there were no significant changes in the nVLFP, nLFP, nHFP, and LHR, in both the control and the football groups. In the football group, the baseline nHFP was significantly larger, whereas the nLFP and LHR were significantly smaller, than their counterparts in the control group. After a rest for 60 min, the nHFP remained significantly larger while the nLFP and LHR remained smaller than their counterparts in the control group. After a rest for 90 min, the nHFP was still significantly larger, whereas the LHR was still significantly smaller than their counterparts in the control group.

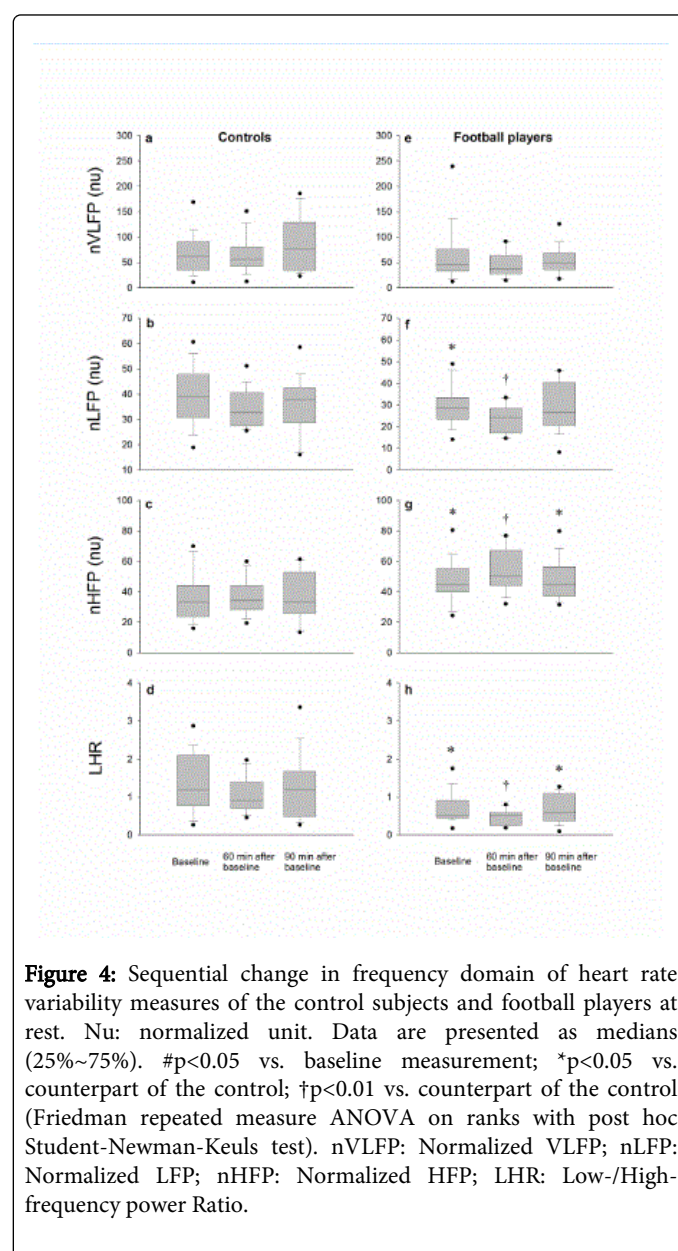


Figure 4: Sequential change in frequency domain of heart rate variability measures of the control subjects and football players at rest. Nu: normalized unit. Data are presented as medians (25%~75%). # $p < 0.05$ vs. baseline measurement; * $p < 0.05$ vs. counterpart of the control; † $p < 0.01$ vs. counterpart of the control (Friedman repeated measure ANOVA on ranks with post hoc Student-Newman-Keuls test). nVLFP: Normalized VLFP; nLFP: Normalized LFP; nHFP: Normalized HFP; LHR: Low-/High-frequency power Ratio.

Percentage changes in blood pressures and HRV measures

Table 2 shows the percentage changes in blood pressures and HRV measures in normal controls and football players after a rest of 60 and 90 minutes. The percentage changes in HR, mRRI, SD_{RR}, CV_{RR}, rMSSD, TP, VLFP, LFP, and HFP after a rest of 60 min were significantly different between normal controls and football players. A further rest to 90 min did not lead to further change in those HRV measures. This result indicated that the decrease in HR and the increase in mRRI, SD_{RR}, CV_{RR}, rMSSD, TP, VLFP, LFP, and HFP in the control group occurred mainly during the first 60 minutes' rest, and a further rest to 90 min did not lead to further change in these HRV measures. Similarly, the decrease in HR and the increase in mRRI in the football group occurred mainly after a rest of 60 min, and a further rest to 90 min did not lead to further change in the HR and mRRI. Comparison between these 2 groups of subjects shows that the magnitude of decrease in HR and the magnitude of increase in mRRI, SD_{RR}, CV_{RR}, rMSSD, TP, VLFP, LFP and HFP after a rest for 60 and 90 min in the football players were significantly smaller than those in the control subjects.

	Control	Football	p value
Blood pressures			
%SBP ₀₋₆₀ (%)	-2 (-5-3)	-2 (-4-6)	NS
%SBP ₀₋₉₀ (%)	3 (-2-6)	4 (-1-8)	NS
%DBP ₀₋₆₀ (%)	-1 (-6-5)	3 (-2-11)	NS
%DBP ₀₋₉₀ (%)	1 (-3-11)	6 (1-18)	NS
%PP ₀₋₆₀ (%)	0 (-10-11)	-4 (-20-3)	NS
%PP ₀₋₉₀ (%)	3 (-8-15)	-6 (-24-4)	NS
Time domain HRV measures			
%HR ₀₋₆₀ (%)	-12 (-19- -9)	-6 (-11-4)	0.009
%HR ₀₋₉₀ (%)	-10 (-16- -5)	-7 (-8-3)	NS
%mRRI ₀₋₆₀ (%)	13 (9-24)	6 (4-13)	0.009
%mRRI ₀₋₉₀ (%)	11 (6-19)	8 (3-9)	NS
%SD _{RR 0-60} (%)	32 (18-63)	0.4 (-19.0-13.1)	<0.001
%SD _{RR 0-90} (%)	28 (20-59)	3 (-20-20)	0.001
%CV _{RR 0-60} (%)	19 (5-33)	-8 (-19-2)	<0.001
%CV _{RR 0-90} (%)	15 (6-45)	-0.4 (-17.6-10.9)	0.005
%rMSSD ₀₋₆₀ (%)	38 (25-87)	18 (-12-32)	0.002
%rMSSD ₀₋₉₀ (%)	25 (4-49)	6 (-12-15)	0.033
Frequency domain HRV measures			
%TP ₀₋₆₀ (%)	70 (39-194)	4 (-31-31)	<0.001
%TP ₀₋₉₀ (%)	40 (16-87)	10 (-30-40)	0.03
%VLFP ₀₋₆₀ (%)	117 (20-225)	-17 (-56-40)	0.003
%VLFP ₀₋₉₀ (%)	146 (77-225)	0.2 (-48.0-89.0)	0.01
%LFP ₀₋₆₀ (%)	0.9 (0.2-1.8)	-0.2 (-0.6-0.3)	<0.001

%LFP ₀₋₉₀ (%)	0.5 (-0.1-1.0)	-0.2 (-0.4-0.4)	0.046
%HFP ₀₋₆₀ (%)	73 (29-226)	36 (-26-64)	0.033
%HFP ₀₋₉₀ (%)	29 (-9-125)	-6 (-23-56)	NS
%nVLFP ₀₋₆₀ (%)	0.09 (-0.31-0.51)	-0.2 (-0.5-0.6)	NS
%nVLFP ₀₋₉₀ (%)	0.3 (-0.2-1.1)	-0.2 (-0.4-0.8)	NS
%nLFP ₀₋₆₀ (%)	-16 (-30-12)	24 (-41-6)	NS
%nLFP ₀₋₉₀ (%)	-10 (-17-7)	-11 (-28-36)	NS
%nHFP ₀₋₆₀ (%)	6 (-10-20)	23 (-4-35)	NS
%nHFP ₀₋₉₀ (%)	-5 (-22-44)	-9 (-20-16)	NS
%LHR ₀₋₆₀ (%)	-0.1 (-0.5-0.1)	-0.2 (-0.5-0.1)	NS
%LHR ₀₋₉₀ (%)	-0.1 (-0.4-0.2)	0 (-0.4-0.7)	NS

Table 2: Comparison of the percentage change in blood pressures and HRV measures 60 and 90 minutes after baseline measurements between normal controls and football players. Data are presented as medians (25%~75%). NS: Not Significant; HR: Heart Rate; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; PP: Pulse Pressure; mRRI: Mean RR Interval; SD_{RR}: Standard Deviation of RR Intervals; CV_{RR}: Coefficient of Variation of RR Intervals; rMSSD: Root Mean Squared Successive Difference; TP: Total Power; VLFP: Very Low-Frequency Power; LFP: Low-Frequency Power; HFP: High-Frequency Power; nVLFP: Normalized VLFP; nLFP: Normalized LFP; nHFP: Normalized HFP; LHR: Low-/High-Frequency Power Ratio. Friedman repeated measure ANOVA on ranks with post hoc Student-Newman-Keuls test. *p<0.05 vs. %X₀₋₆₀; §p<0.05 vs. %X₀₋₉₀.

Discussion

For football sport, Fardy [15] has shown that soccer players made great strides in the cardiorespiratory endurance and VO_{2max}, and had about 15.5% increase in VO_{2max} after 10-week soccer training. Reilly and Thomas [16] indicated that the vital capacity was increased, while the body fat, systolic blood pressure (SBP), diastolic blood pressure (DBP), peaceful HR and maximum HR were all decreased after a programme of pre-season training on the soccer players. Ostojic and Mazic [17] suggested that soccer players should consume carbohydrate-electrolyte fluid throughout a game to help prevent deterioration in specific skill performance. Brites et al. [18] showed that soccer players under regular training show an improved plasma antioxidant status in comparison to sedentary controls. Hoff et al. [19] showed that HR monitoring during soccer specific exercise is a valid indicator of actual exercise intensity. Unfortunately, few studies have addressed the autonomic nervous modulation of the football players. We found in this study that almost all HRV measures in the football group were significantly larger or smaller than those of normal controls except the nVLFP in the baseline measurement. However, there were no significant differences in SD_{RR}, CV_{RR}, TP, VLFP, and LFP between the football and control groups 60 min after the baseline measurement, and there were no significant differences in the SD_{RR}, CV_{RR}, VLFP, LFP, and nLFP between the football and control groups 90 min after the baseline measurement. This result suggested that a rest for 60 min can narrow down the difference in autonomic nervous modulation between football players and normal controls. Despite of this effect of rest on the autonomic nervous modulation in the control

group, some HRV measures of the football players were still higher than those of normal controls after a rest for 60 to 90 min. This result suggested that even after a rest for 60 to 90 min, the autonomic nervous modulation of the sedentary controls still could not reach the same degree of relaxation as that of football players.

Ishida and Okada [20] showed that there were significant differences in the spectral components of HRV during exercise between athletes and control subjects. Their results showed that physical training could possibly increase the parasympathetic activity (or decrease the sympathetic activity). Costa et al. [21] indicated that higher power of both spectral bands (LF and HF) and higher amplitude of the respective peaks in athletes when compared with healthy sedentary subjects, with a clear predominance of the HF band in the total spectral power density, which suggested that the higher HRV observed in athletes reveals the predominance of parasympathetic activity, without reduction in the sympathetic tone. Shin et al. [22] indicated that endurance training induces autonomic imbalance (i.e., the enhanced vagal activities and the attenuated sympathetic tone). Shin and his colleagues also demonstrated that the HR of athletes was significantly lower than that of non-athletes, and the HF power of athletes was significantly higher than that of non-athletes during rest and post-exercise period, indicating that vagal activity was enhanced by the adaptive changes in neural regulation produced by long-term physical training [23]. Nagai and colleagues [24] suggested that the 12-month moderate exercise training has a positive effect on cardiac ANS activity in the children who initially had low HRV. In accordance with these studies, we found that the indices of vagal modulation such as HFP and nHFP in the football group were significantly higher than those of normal controls, and the indices of sympathetic modulation such as nLFP and LHR in the football group were significantly lower than those of normal controls. Even after a rest for 60-90 minutes, many HRV measures of the football players such as HR, mRRI, rMSSD, HFP, nHFP, and LHR were still significantly different from those of the normal controls. Our observation suggested that football sports can increase the vagal modulation and decrease the sympathetic modulation of the subjects, similar to other kinds of sports or physical trainings, as compared to the non-athletes. Persistent strenuous exercise in the football training and game should be the reason why the HRV measures of the football group were so different from those of the non-athlete high in the baseline measurement.

Sixty and 90 min after baseline measurement, the HR was decreased while the mRRI, SD_{RR} , CV_{RR} , rMSSD, TP, VLFP, and HFP were increased in the control group. In the football group, however, a rest for 60 to 90 min could lead to slight decrease in HR and slight increase in mRRI only; no changes in other HRV measures could be found in the football group after a rest for 60 to 90 min. These results suggested that a rest for 60 to 90 min could result in increased overall HRV and decreased HR in the control subjects, but only slightly decreased HR and not changed overall HRV in the football group. It seems that football players at rest were already in a nearly full relaxation state which could not be relaxed further by taking more rest, while the non-athletes were not relaxed fully at rest and more rest could lead to further relaxation. This result seems to be in accordance with the finding of Rebelo et al. [25] that in spite of the high intensity training period, there was no significant change in results from detraining condition to training condition in professional football players. Knoepfli-Lenzin et al. [26] showed that football training, consisting of high-intensity intermittent exercise, results in positive effects on blood pressure, body composition, stroke volume and supine heart rate

variability, and elicits at least the same cardiovascular health benefits as continuous running exercise in habitually active men with mild hypertension. Mandigout et al. [27] pointed out that an endurance training program had a positive effect on aerobic potential, morphological and functional cardiac parameters and on nocturnal global HRV in healthy prepubertal children without inducing sympathetic and parasympathetic. In parallel with the finding of these studies, we found the PP and %FEV₁ of the football players were larger than those of the controls. Our data suggested that the tracheobroncheal tree of the football players might be more competent to allow more air to pass through, and the vascular resistance of the football players might be smaller than that of the non-athletes to facilitate a better blood circulation.

The frequency range of 0.003-0.04 Hz defined for the VLFP by the Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology [9] is for the analysis of "entire 24 hours". This definition of frequency range for VLFP cannot be applied to the present study because the ECG signals were recorded for only 15 minutes or so in this study. For analysis of short-term recordings (5 min), a frequency range of ≤ 0.04 Hz was defined for the frequency range of VLFP by the Task Force [9]. Again, this definition of frequency range for VLFP cannot be applied to our study literally because the recording time of ECG in our study was 15 minutes or so which is longer than 5 minutes. In other words, there is currently "NO" standard definition for the frequency range of VLFP for ECG recording between 5 min and 24 hours. Therefore, we defined a frequency range of 0.01-0.04 Hz for VLFP in our studies. The upper limit of 0.04 Hz in our definition was the same as that of 24 hours and 5 min HRV analysis defined by the Task Force [9]. Since a lower limit of 0.003 Hz for VLFP is too small to be defined for an ECG recording of 15 minutes or so; therefore, a lower limit of 0.01 Hz was chosen so that the counterpart of ultralow frequency power (ULFP) for 24 hours recording (≤ 0.003 Hz) could be calculated in an ECG recording of around 15 min or longer.

It was suspected that the Task Force [9] might have not paid appropriate attention to the VLFP because the VLFP was not normalized by any power, and because the frequency range of the power used for the normalization of LFP and HFP did not cover that of VLFP in the definitions of the Task Force. We believe that this overlook of VLFP by the Task Force [9] is an insufficiency to spectral HRV analysis because Taylor et al. [11] have found that the VLF RR-interval oscillations are very much dependent on vagal tone so that the VLFP can be used as the indices of renin-angiotensin-aldosterone system activity and vagal withdrawal in spectral HRV analysis. The vagal withdrawal here means that the vagus nerve is inhibited from slowing the activity of the sinoatrial node and from buffering the degree of contraction throughout the myocardium. Thus, the VLFP and nVLFP cannot simply be downplayed. To remedy the insufficiency in the definitions of normalized powers by the Task Force, we used the power within the frequency range of 0.01-0.4 Hz (total power-ULFP) to normalize the VLFP, LFP and HFP in our studies. We believe that this way of definitions for nHFP, nLFP and nVLFP are improvements, rather than limitations, to spectral HRV analysis.

In this study, only 17 subjects per group were recruited for spectral HRV analysis. These are rather small groups, and thus the results obtained in this study should be interpreted with caution.

Conclusion

Football sports can result in increased vagal modulation and decreased sympathetic modulation, as compared to the non-athletes. A rest for 60 to 90 min can result in a significant decrease in vagal modulation in the control group, but little in the football players. It seems that the football players at rest are already in a nearly full relaxation state which cannot be relaxed further by more resting, whereas the non-athletes are not relaxed fully at rest and a rest can lead to further relaxation. The room for cardiac vagal modulation to increase and cardiac sympathetic modulation to decrease by resting in the football players is smaller than that of normal subjects.

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References

1. Akselrod S, Gordon D, Ubel FA, Shannon DC, Berger AC, et al. (1981) Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat-to-beat cardiovascular control. *Science* 213: 220-222.
2. Jensen-Urstad K, Saltin B, Ericson M, Storck N, Jensen-Urstad M (1997) Pronounced resting bradycardia in male elite runners is associated with high heart rate variability. *Scand J Med Sci Sports* 7: 274-278.
3. Aubert AE, Beckers F, Ramaekers D (2001) Short-term heart rate variability in young athletes. *J Cardiol* 37 Suppl 1: 85-88.
4. Melo RC, Santos MD, Silva E, Quitério RJ, Moreno MA, et al. (2005) Effects of age and physical activity on the autonomic control of heart rate in healthy men. *Braz J Med Biol Res* 38: 1331-1338.
5. Lu WA, Kuo CD (2006) Comparison of the effects of Tai Chi Chuan and Wai Tan Kung exercises on autonomic nervous system modulation and on hemodynamics in elder adults. *Am J Chin Med* 34: 959-968.
6. Amano M, Kanda T, Ue H, Moritani T (2001) Exercise training and autonomic nervous system activity in obese individuals. *Med Sci Sports Exerc* 33: 1287-1291.
7. Shin K, Minamitani H, Onishi S, Yamazaki H, Lee M (1997) Autonomic differences between athletes and nonathletes: spectral analysis approach. *Med Sci Sports Exerc* 29: 1482-1490.
8. Woods C, Hawkins R, Hulse M, Hodson A (2002) The Football Association Medical Research Programme: an audit of injuries in professional football-analysis of preseason injuries. *Br J Sports Med* 36: 436-441.
9. [No authors listed] (1996) Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Circulation* 93: 1043-1065.
10. Fleisher LA, Frank SM, Sessler DI, Cheng C, Matsukawa T, et al. (1996) Thermoregulation and heart rate variability. *Clin Sci (Lond)* 90: 97-103.
11. Taylor JA, Carr DL, Myers CW, Eckberg DL (1998) Mechanisms underlying very-low-frequency RR-interval oscillations in humans. *Circulation* 98: 547-555.
12. Thayer JF, Nabors-Oberg R, Sollers JJ 3rd (1997) Thermoregulation and cardiac variability: a time-frequency analysis. *Biomed Sci Instrum* 34: 252-256.
13. Koizumi K, Terui N, Kollai M (1985) Effect of cardiac vagal and sympathetic nerve activity on heart rate in rhythmic fluctuations. *J Auton Nerv Syst* 12: 251-259.
14. Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, et al. (1986) Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. *Circ Res* 59: 178-193.
15. Fardy PS (1969) Effects of soccer training and detraining upon selected cardiac and metabolic measures. *Res Q* 40: 502-508.
16. Reilly T, Thomas V (1977) Effects of a programme of pre-season training on the fitness of soccer players. *J Sports Med Phys Fitness* 17: 401-412.
17. Ostojic SM, Mazic S2 (2002) Effects of a carbohydrate-electrolyte drink on specific soccer tests and performance. *J Sports Sci Med* 1: 47-53.
18. Brites FD, Evelson PA, Christiansen MG, Nicol MF, Basílico MJ, et al. (1999) Soccer players under regular training show oxidative stress but an improved plasma antioxidant status. *Clin Sci (Lond)* 96: 381-385.
19. Hoff J, Wisløff U, Engen LC, Kemi OJ, Helgerud J (2002) Soccer specific aerobic endurance training. *Br J Sports Med* 36: 218-221.
20. Ishida R, Okada M (1997) [Spectrum analysis of heart rate variability for the assessment of training effects]. *Rinsho Byori* 45: 685-688.
21. Costa O, Freitas J, Puig J, Carvalho MJ, Freitas A, et al. (1991) [Spectrum analysis of the variability of heart rate in athletes]. *Rev Port Cardiol* 10: 23-28.
22. Shin K, Minamitani H, Onishi S, Yamazaki H, Lee M (1995) Assessment of training-induced autonomic adaptations in athletes with spectral analysis of cardiovascular variability signals. *Jpn J Physiol* 45: 1053-1069.
23. Shin K, Minamitani H, Onishi S, Yamazaki H, Lee M (1995) The power spectral analysis of heart rate variability in athletes during dynamic exercise--Part I. *Clin Cardiol* 18: 583-586.
24. Nagai N, Hamada T, Kimura T, Moritani T (2004) Moderate physical exercise increases cardiac autonomic nervous system activity in children with low heart rate variability. *Childs Nerv Syst* 20: 209-214.
25. Rebelo AN, Costa O, Rocha AP, Soares JM, Lago P (1997) [Is autonomic control of the heart rate at rest altered by detraining? A study of heart rate variability in professional soccer players after the pretraining period and after the preparatory period for competitions]. *Rev Port Cardiol* 16: 535-541, 508.
26. Knoepfli-Lenzin C, Sennhauser C, Toigo M, Boutellier U, Bangsbo J, et al. (2010) Effects of a 12-week intervention period with football and running for habitually active men with mild hypertension. *Scand J Med Sci Sports* 20 Suppl 1: 72-79.
27. Mandigout S, Melin A, Fauchier L, N'Guyen LD, Courteix D, et al. (2002) Physical training increases heart rate variability in healthy prepubertal children. *Eur J Clin Invest* 32: 479-487.