

The Effect of Tai Chi Chuan on the Autonomic Nervous Modulation in Older Persons

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ABSTRACT

LU, W-A., and C-D. KUO. The Effect of Tai Chi Chuan on the Autonomic Nervous Modulation in Older Persons. *Med. Sci. Sports Exerc.*, Vol. 35, No. 12, pp. 1972–1976, 2003. **Purpose:** This study evaluated the effect of Tai Chi Chuan (TCC) on the autonomic nervous modulation in older persons. **Methods:** Twenty TCC practitioners and 20 normal controls were included in this study. The stationary state spectral heart rate variability (HRV) measures between TCC practitioners and normal controls, and the sequential changes in HRV measures after classical Yang's TCC were compared. **Results:** The total power, very low-frequency power, low-frequency power, normalized low-frequency power, and low-/high-frequency power ratios in TCC practitioners were all significantly higher than those of normal controls, whereas the heart rate and systolic and diastolic blood pressures were not different between these two groups of subjects. After TCC, the normalized high-frequency power increased significantly from 22.8 ± 14.6 normalized units (nu) before TCC to 28.2 ± 16.1 nu 30 min after TCC and to 30.6 ± 18.4 nu 60 min after TCC. In contrast, the low-/high-frequency power ratio decreased significantly from 2.5 ± 2.4 before TCC to 1.8 ± 1.4 30 min after TCC and to 2.2 ± 2.9 60 min after TCC. The heart rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, and pulse pressure also decreased sequentially after TCC. **Conclusion:** The short-term effect of TCC was to enhance the vagal modulation and tilt the sympathovagal balance toward decreased sympathetic modulation in older persons. TCC might be good health-promoting calisthenics for older persons. **Key Words:** HEART RATE VARIABILITY, SPECTRUM, SYMPATHETIC, VAGAL, EXERCISE

Vagal modulation has been found to be decreased in various physiological and pathological conditions, such as aging (19), acute myocardial infarction (21), diabetes mellitus (20), chronic renal failure (1), and congestive heart failure (22). The reduction in the cardiac vagal function, as evaluated by baroreflex sensitivity or spectral heart rate variability (HRV) analysis, was found to be associated with poor prognosis (14). Because of the poor prognostic significance of reduced vagal modulation, any means that can increase the vagal modulation of subjects is potentially beneficial to those patients suffering from disease state with decreased vagal modulation. Pharmacological measures have been shown to increase the vagal modulation in normal subjects and in patients with acute myocardial infarction (4) and congestive heart failure (13). However, these pharmacologic interventions proved to be of

no clinical benefit to those patients. Physiological means are also welcome because of their safety, feasibility, and lack of complications. Among many possible physiological means, the right lateral decubitus position has been demonstrated to increase the vagal modulation of patients with coronary artery disease and acute myocardial infarction (11).

Handgrip exercise is one of the physiological means that can increase the vagal modulation of normal subjects (12). It was also suggested that supervised exercise in the elderly should emphasize aerobic, strengthening, and flexibility training (3). For patients after acute myocardial infarction referred for cardiac rehabilitation, low- and high-intensity exercise training have been shown to improve the functional capacity and produce relatively similar changes in cardiorespiratory variables during the initial 3 months of exercise training (2). For the elderly, some oriental conditioning exercises deserve special attention because they are easily accessible, of low cost, and hence are suitable for their implementation in the community.

Tai Chi Chuan (TCC) is a traditional Chinese conditioning exercise or calisthenics that can delay the decline of aerobic power (16). TCC training has been shown to be beneficial to the cardiopulmonary function (15,17), balance (25), and strength (17) of subjects. Study has demonstrated that a 1-yr TCC program for low-risk patients with coronary artery bypass surgery could favorably enhance cardiorespiratory function (18). One study also demonstrated that TCC could reduce tension, anxiety, and mood disturbance (10). Because there have been more than one hundred million TCC practitioners in

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the world (6), it is interesting to know the physiological effects of TCC on subjects.

To our knowledge, there were no studies concerning effects of TCC on the autonomic nervous modulation of subjects. Therefore, the aim of this study was to examine the effect of TCC on the autonomic nervous modulation in older persons by using spectral HRV analysis.

METHODS

Subject selection and study design. Both TCC practitioners and normal subjects without TCC experience were included in this study. The TCC practitioners were recruited from a TCC training center in Taiwan, and the normal subjects without TCC experience were recruited from the community. Medical and activity histories were obtained by questionnaire. All subjects had normal lifestyles and were capable of daily activities without limitations. The Institute Review Board of Taipei Veterans General Hospital approved this study (VGHIRB No. 91-04-13A). The procedure was fully explained to the subjects, and informed written consent was obtained from them before study. Subjects who had major cardiopulmonary disease or were on regular medicine for diabetes mellitus, hypertension, or renal or liver disease were excluded from the study.

A resting standard 12-lead ECG, a spirometry (IQmark Digital ECG and Digital Spirometer, Brentwood Medical Technology Corp., Torrance, CA), blood pressure measurement (Kenlu-model K-300 Sphygmomanometer, Di Tai Precision Ent. Co., Ltd., Taiwan), and arterial O₂ saturation (SpO₂) measurement (Biochem Vital Sign Monitor, BCI International, Waukesha, WI) were performed on each subject at each session.

The subjects included in this study were instructed not to take caffeinated or alcoholic beverages for at least 24 h before study. Before TCC, the subject was instructed to lie down and take a rest in supine position. After 5-min rest, a trend of conventional lead II electrocardiographic signal was picked up by the ECG monitor (Biochem Vital Sign Monitor) and transferred to a personal computer for storage and later analysis. After baseline recording, the subject was advised to exercise by doing classical Yang's TCC for 40 min. Each session of Yang's TCC included 10-min warm-up exercise (lower back and hamstring stretching, gentle calisthenics, and balance training), 20-min TCC exercise, and 10-min cool-down exercise. Each set of Yang's TCC included 64 postures (6). During TCC exercise, the subject kept the same pace in exercising the postures of TCC in sequence by performing these postures according to a pre-recorded tape to ensure that the same pace and sequence of postures were followed by the TCC practitioner. Thirty and 60 min after the completion of TCC exercise, the second and the third trends of ECG signals were recorded using the same methodology. All subjects took a rest in sitting position and watched the television between the end of the TCC session and HRV measurements at 30 and 60 min. All procedures were performed in a bright and quiet room with a constant temperature of 24–25°C.

Equipment and measurement. The sampling frequency for ECG recording was 200 Hz. R-wave detecting software was used to identify the peaks of the R waves in the recorded ECG signals and to measure the consecutive RR intervals. Sinus pause and arterial or ventricular arrhythmia were deleted, and the last 512 stationary RR intervals in each session were obtained for spectral HRV analysis.

The mean, standard deviation, and coefficient of variation of the 512 stationary RR intervals were calculated using standard formulae for each subject. The power spectra of RR intervals were obtained by means of fast Fourier transformation (Mathcad, Mathsoft Inc., Cambridge, MA). Zero-frequency component was excluded before the calculation of the power. The area-under-the-curve of the spectral peaks within the range of 0.01–0.4 Hz was defined as the total power (TP), the area underneath the spectral peaks within the range of 0.01–0.04 Hz was defined as the very low-frequency power (VLFP), the area underneath the spectral peaks within the range of 0.04–0.15 Hz was defined as the low-frequency power (LFP), and the area underneath the spectral peaks within the range of 0.15–0.40 Hz was defined as the high-frequency power (HFP). The normalized high-frequency power (nHFP = HFP/TP) was used as the index of vagal modulation, the normalized low-frequency power (nLFP = LFP/TP) as the index of sympathetic and vagal modulation, the low-/high-frequency power ratio (LFP/HFP) as the index of sympathovagal balance (22), and the normalized very low-frequency power (nVLFP = VLFP/TP) as the index of rennin-angiotensin-aldosterone system and vagal withdrawal of the subject (23). The interpretation of nLFP as an index of sympathetic and vagal modulation may be valid only when respiration is absolutely above the low-frequency band, which can be ascertained only by simultaneous measurement and analysis of respiration.

Data analysis. The Mann-Whitney rank sum test (SigmaStat statistical software, SPSS Inc., Chicago, IL) was employed to compare the baseline characteristics and the HRV measures in both time and frequency domains between TCC practitioners and normal controls. Friedman repeated measures analysis of variance on ranks was performed to compare the HRV measure among before TCC, 30 min after TCC, and 60 min after TCC, with statistical significance set as $P < 0.05$. Wilcoxon signed rank test with Bonferroni correction was employed in the three pairwise comparisons among before TCC, 30 min after TCC, and 60 min after TCC, where the statistical significance was set as $P < 0.017$. Linear regression analysis was used to assess the linear relationship between TCC experience and HRV measures. A $P < 0.05$ was considered statistically significant. All data are presented as mean \pm SD.

To quantify the effect of TCC on the autonomic nervous modulation, the percentage changes in the HRV measures after TCC in each subject were calculated using the following formula:

$$\%X_{30} = [(X_{30 \text{ min after TCC}} - X_{\text{before TCC}}) / (X_{\text{before TCC}})] \times 100$$

$$\%X_{60} = [(X_{60 \text{ min after TCC}} - X_{\text{before TCC}}) / (X_{\text{before TCC}})] \times 100$$

RESULTS

Twenty TCC practitioners and 20 normal controls were included in this study. The TCC practitioners had practiced TCC calisthenics at least 1 h each time, $3 \times \text{wk}^{-1}$ for an average of 1.9 ± 1.0 yr. Table 1 tabulates the baseline characteristics of normal controls and TCC practitioners. There were no significant differences in the age, gender, body weight, body height, body mass index (BMI), heart rate (HR), forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1), and the ratio of forced expiratory volume in the first second to the forced vital capacity (FEV_1/FVC) between normal controls and TCC practitioners except the length of TCC training. The length of TCC experience did not correlate with the measures of HRV. Among the TCC practitioners, there were 11 men and 9 women. There was no significant difference in HRV measures between men and women TCC practitioners.

The first two columns of Table 2 compare the hemodynamics and the HRV measures between normal controls and TCC practitioners before TCC. There were no significant differences in the HR, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, pulse pressure, and arterial O_2 saturation between TCC practitioners before TCC and normal controls. However, the SD_{RR} , CV_{RR} , TP, VLFP, LFP, nLFP, and LFP/HFP of the TCC practitioners before TCC were all significantly higher than those of normal controls.

The last three columns of Table 2 show the sequential hemodynamics and HRV measures after TCC in the TCC practitioners. After TCC, HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MABP), and pulse pressure (PP) all decreased gradually. In the frequency domain, the nHFP increased significantly from 22.8 ± 14.6 normalized unit (nu) before TCC to 28.2 ± 16.1 nu 30 min after TCC and to 30.6 ± 18.4 nu 60 min after TCC. In contrast, the LFP/HFP decreased significantly from 2.5 ± 2.4 before TCC to 1.8 ± 1.4 30 min after TCC and to 2.2 ± 2.9 60 min after TCC.

Table 3 shows the percentage changes in the hemodynamics and HRV measures in the TCC practitioners after

TCC. The percentage increases in nHFP 30 min and 60 min after TCC were $38.3 \pm 63.4\%$ and $46.7 \pm 61.4\%$, respectively, whereas the percentage decreases in LFP/HFP 30 min and 60 min after TCC were $19.6 \pm 36.8\%$ and $16.8 \pm 51.7\%$, respectively. The percentage changes in all hemodynamic measurements 30 min after TCC were not significantly different from those 60 min after TCC. However, the percentage changes in TP and HFP 60 min after TCC were significantly higher than those 30 min after TCC.

DISCUSSION

General features of TCC. TCC is a traditional Oriental mind-body calisthenics that is unique for its slow and graceful movements. TCC consists of a sequence of postures that are smooth, harmonic, and relaxing. TCC is performed in semi-squat postures with varying degrees of concentric and eccentric contractions (6). Thus, TCC can significantly enhance the strength of lower extremities, while the upper extremities are in a relaxed state during TCC practice (15–17). TCC has many advantageous features. First, it needs no special facility, equipment, or instruments. Second, it can be practiced almost anytime and anywhere. Third, it is low cost and low technology, and may be easily implemented in the community. Fourth, the orthopedic complications of TCC are minimal. Finally, TCC is effective for enhancing cardiorespiratory function and other fitness traits.

Channer and associates (5) showed that an 8-wk, low-intensity TCC program was effective for reducing blood pressure in patients with acute myocardial infarction. Lai et al. (15) studied 2-yr trends in cardiovascular function among elderly TCC practitioners and sedentary subjects, and found that regular TCC might delay the decline of cardiovascular function in elderly individuals and might be prescribed as a suitable aerobic exercise for the elderly. They also demonstrated that TCC training had benefits for health-related fitness and might be prescribed as a suitable conditioning exercise for the elderly (16). In another study, Lan et al. (17) demonstrated that a 12-month TCC program was effective in improving cardiorespiratory function, muscle strength, and flexibility in the elderly. Lan et al. (18) later showed that TCC could significantly increase the $\dot{\text{V}}\text{O}_{2\text{peak}}$ and peak work rate in patients with coronary artery bypass surgery. Similarly, Hong et al. (8) reported that long-term regular TCC exercise had favorable effects on the promotion of balance control, flexibility, and cardiovascular fitness in the elderly. From these studies, it is evident that TCC is a suitable conditioning exercise for elderly and patients with coronary artery disease.

Our study showed that the years of TCC experience did not correlate with the measures of HRV. It seems probable that many factors, such as the TCC practitioner's physical status, their level of devotion to TCC practice, and the sophistication of TCC done by the subject, can affect their autonomic nervous modulation, in addition to their length of TCC experience. This might be the reason why the length of

TABLE 1. Baseline characteristics of the TCC practitioners and the normal controls.

Baseline Characteristics	Normal Controls (N = 20)	TCC Practitioners (N = 20)
Age (yr)	56.3 ± 8.5	52.8 ± 7.5
Gender (M/F)	5/15	9/11
Body Weight (kg)	59.0 ± 8.8	59.3 ± 11.1
Body height (cm)	155.1 ± 7.4	160.5 ± 9.9
BMI ($\text{kg}\cdot\text{m}^{-2}$)	24.4 ± 2.1	22.9 ± 2.9
Length of TCC training (yr)	0.0	1.9 ± 1.0*
Heart rate (bpm)	70.1 ± 9.5	67.6 ± 9.5
FVC (L)	2.8 ± 0.7	3.4 ± 1.0
%FVC (%)	119.5 ± 19.0	115.5 ± 15.1
FEV_1 (L)	2.2 ± 0.6	2.7 ± 0.9
% FEV_1 (%)	116.8 ± 26.9	110.7 ± 23.2
FEV_1/FVC (%)	79.9 ± 7.4	78.8 ± 12.5

FVC, forced vital capacity; FEV_1 , forced expiratory volume in the first second; BMI, body mass index; bpm, beats per minute.

Data are listed as mean ± SD.

* $P < 0.05$ vs control.

TABLE 2. Sequential change in hemodynamics and heart rate variability after TCC.

Measures	Controls	Before TCC	30 min after TCC	60 min after TCC
Hemodynamics				
SBP (mm Hg)	121.8 ± 15.7	118.4 ± 12.8‡	110.8 ± 11.1†	109.9 ± 11.1†
DBP (mm Hg)	73.6 ± 10.3	75.8 ± 9.9‡	73.8 ± 8.2	73.7 ± 8.4
MABP (mm Hg)	89.6 ± 10.7	90.0 ± 10.4‡	86.1 ± 8.6†	85.8 ± 8.8†
PP (mm Hg)	48.3 ± 13.0	42.6 ± 7.6‡	37.1 ± 7.2†	36.2 ± 7.3†
SpO ₂ (%)	96.1 ± 1.4	96.0 ± 0.7	96.3 ± 0.8	96.3 ± 1.0
Time domain				
Mean RRI (ms)	855.9 ± 144	903.7 ± 119.9‡	921.6 ± 104.1	942.5 ± 118.4
Heart rate (bpm)	70.1 ± 9.5	67.6 ± 9.5‡	65.9 ± 7.8	64.6 ± 8.2
SD _{RR} (ms)	27.5 ± 11.3	37.0 ± 14.2*	33.8 ± 10.6	38.9 ± 16.8
CV _{RR} (%)	3.1 ± 1.2	4.1 ± 1.5*	3.8 ± 1.1	4.1 ± 1.6
Frequency domain				
TP (ms ²)	295.0 ± 255.0	596.8 ± 475.2*	513.6 ± 376.2	701.1 ± 733.3
VLFP (ms ²)	131.1 ± 102.2	218.6 ± 158.1*	179.4 ± 108.7	195.4 ± 146.3
LFP (ms ²)	69.5 ± 100.2	241.3 ± 263.5*	185.5 ± 244.9	297.6 ± 480.3#
HFP (ms ²)	94.4 ± 104.4	137.0 ± 150.5‡	148.7 ± 142.6	208.1 ± 201.1
nHFP (nu)	32.0 ± 19.1	22.8 ± 14.6‡	28.2 ± 16.1†	30.6 ± 18.4†
nLFP (nu)	21.1 ± 9.3	35.1 ± 11.6*	33.1 ± 12.2	35.1 ± 15.8
nVLFP (nu)	47.0 ± 19.1	42.1 ± 17.1	38.9 ± 15.4	34.2 ± 14.1
LFP/HFP	1.0 ± 0.9	2.5 ± 2.4*‡	1.8 ± 1.4†	2.2 ± 2.9

SBP, systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure; PP, pulse pressure; SpO₂, arterial O₂ saturation; RRI, RR intervals; SD_{RR}, standard deviation of RR; CV_{RR}, coefficient of variation of RR; ms, millisecond; bpm, beats per minute; nu, normalized unit; TP, total power; VLFP, very low-frequency power; LFP, low-frequency power; HFP, high-frequency power; nHFP, normalized high-frequency power; nLFP, normalized low-frequency power; nVLFP, normalized very low-frequency power; LFP/HFP, low/high-frequency power ratio.

Data presented are mean ± SD.

* $P < 0.05$ vs normal controls; ‡ $P < 0.05$ among before TCC, 30 min after TCC and 60 min after TCC; † $P < 0.017$ vs before TCC; # $P < 0.017$ vs 30 min after TCC.

TCC experience did not correlate with the measures of HRV.

Comparison of TCC practitioners and controls.

Because TCC has been shown to improve the cardiopulmonary function of the subjects, it is worthwhile comparing the cardiopulmonary function between TCC practitioners and normal controls. Though many traditional measurements of cardiopulmonary function were not significantly different between TCC exercisers before TCC and normal controls, many HRV measurements such as SD_{RR}, CV_{RR}, TP, VLFP,

LFP, nLFP, and LFP/HFP of the TCC practitioners before TCC were all significantly higher than those of normal controls. This observation suggested that the accumulated or long-term effect of TCC was to increase the low-frequency components without comprising the high-frequency components of HRV in TCC practitioners. At first sight, the higher low-frequency components (LF) (in relative terms) may suggest that TCC practitioners may have enhanced sympathetic modulation, but the evidence of a similar (or slightly lower) blood pressure, of a similar (or slightly lower) HR, and of a much greater HRV (SD_{RR}) and TP clearly argues against this concept.

The practice of TCC demands that the TCC practitioners synchronize their respiration with their body movements; therefore, the subjects likely learn how to control (and possibly slow down) their respiration. Because TCC practitioners occasionally take slow and deep breaths, the most likely reason why the TCC practitioners before TCC have a marked and significant predominance in the low-frequency region is that they occasionally take slow and deep breaths. To see whether the observed increase in the low-frequency components of HRV in TCC practitioners before TCC is indeed caused by their controlled slow respiration, it is necessary to compare the respiratory frequency of the TCC practitioners and that of the controls. Unfortunately, the respiratory frequency of both TCC practitioners and controls was not recorded in this study. This is a limitation of the present study.

Short-term effect of TCC on autonomic modulation. Hull et al. (9) demonstrated that chronic exercise not only increased high-frequency power but also prevented ventricular fibrillation during acute myocardial ischemia. Goldsmith et al. (7) indicated that physical fitness was highly correlated with vagal modulation as measured by

TABLE 3. The effects of TCC in hemodynamics and heart rate variability.

Measures	%X ₃₀	%X ₆₀
Hemodynamics		
SBP (mm Hg)	-6.1 ± 6.8	-6.9 ± 6.8
DBP (mm Hg)	-2.3 ± 6.4	-2.4 ± 6.1
MABP (mm Hg)	-4.0 ± 6.1	-4.4 ± 6.0
PP (mm Hg)	-12.1 ± 13.2	-14.2 ± 13.3
SpO ₂ (%)	0.3 ± 0.8	0.3 ± 1.1
Time domain		
Mean RRI (%)	2.5 ± 7.8	4.8 ± 9.7
Heart rate (%)	-1.9 ± 8.1	-1.5 ± 11.9
SD _{RR} (%)	-0.6 ± 27.4	10.5 ± 40.0
CV _{RR} (%)	-3.4 ± 23.5	5.0 ± 35.8
Frequency domain		
TP (%)	11.7 ± 70.0	45.1 ± 118.3†
VLFP (%)	14.4 ± 99.8	14.2 ± 85.8
LFP (%)	12.2 ± 82.8	56.4 ± 151.4
HFP (%)	39.2 ± 76.5	100.8 ± 185.0†
nHFP (%)	38.3 ± 63.4	46.7 ± 61.4
nLFP (%)	0.6 ± 42.9	3.1 ± 42.6
nVLFP (%)	-0.2 ± 36.9	-13.9 ± 34.6
LFP/HFP (%)	-19.6 ± 36.8	-16.8 ± 51.7

SBP, systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure; PP, pulse pressure; SpO₂, arterial O₂ saturation; RRI, RR intervals; SD_{RR}, standard deviation of RR; CV_{RR}, coefficient of variation of RR; ms, millisecond; bpm, beats per minute; nu, normalized unit; TP, total power; VLFP, very low-frequency power; LFP, low-frequency power; HFP, high-frequency power; nHFP, normalized high-frequency power; nLFP, normalized low-frequency power; nVLFP, normalized very low-frequency power; LFP/HFP, low/high-frequency power ratio.

Data are listed as mean ± SD.

† $P < 0.05$ vs %X₃₀.

high-frequency power. Kurita et al. (12) demonstrated that in normal subjects, the low-frequency power and low-/high-frequency power ratios were not significantly changed during handgrip exercise, but high-frequency power was significantly increased. Wilmore and Costill (24) also said that although the sympathetic system predominates during times of physical or emotional stress when the body demands are higher, the parasympathetic system again predominates after the stress subsides. In accordance with the above reports, we showed that the short-term effect of TCC exercise was to enhance the spectral vagal indices and to lower the sympathetic modulation. This is comprehensible, because TCC exercise might produce a nice relaxing effect as evidenced by the drop of HR from 67.6 ± 9.5 bpm before TCC to 65.9 ± 7.8 bpm 30 min after TCC, and to 64.6 ± 8.2 bpm 60 min after TCC, and by the drop of systolic blood pressure from 118.4 ± 12.8 mm Hg before TCC to 110.8 ± 11.1 mm Hg

30 min after TCC, and to 109.9 ± 11.1 60 min after TCC. Because aging and many disease states are associated with decreased vagal activity and increased sympathetic activity, and because TCC might improve the vagal modulation of these subjects, TCC is worthy of recommendation to older persons.

CONCLUSIONS

The short-term effect of TCC was to enhance the vagal modulation and tilt the sympathovagal balance toward decreased sympathetic modulation in older persons. TCC might be a good health-promoting calisthenics that could be recommended to older persons.

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