ORIGINAL RESEARCH

Foot Reflexology Can Increase Vagal Modulation, Decrease Sympathetic Modulation, and Lower Blood Pressure in Healthy Subjects and Patients With Coronary Artery Disease

Wan-An Lu, MD, PhD; Gau-Yang Chen, MD, PhD; Cheng-Deng Kuo, MD, PhD

Objective • Complementary and alternative medicine (CAM) has long been used by people to postpone the aging process and to reverse disease progression. Reflexology is a CAM method that involves massage to reflex areas in the feet and hands. This study investigated the effect of foot reflexology (FR) on the autonomic nervous modulation in patients with coronary artery disease (CAD) by using heart rate variability analysis.

Study Methods • Seventeen people with angiographically patent coronary arteries and 20 patients with CAD scheduled for coronary artery bypass graft surgery were recruited as the control and CAD groups, respectively. The normalized high-frequency power (nHFP) was used as the index of vagal modulation and the normalized very low-frequency power (nVLFP) as the index of vagal withdrawal and renin-angiotensin modulation.

Results • In both control and CAD groups, the nHFP was increased significantly whereas the nVLFP was decreased significantly 30 and 60 minutes after FR, as compared with those before FR. The systolic, diastolic, mean arterial, and pulse pressures were significantly decreased after FR in both groups of participants. In the CAD group, the percentage change in heart rate 30 and 60 minutes after FR was smaller than that in the control, and the percentage change in nVLFP 60 minutes after FR was smaller than that in the control. In conclusion, a higher vagal modulation, lower sympathetic modulation, and lower blood pressure can be observed following 60 minutes of FR in both controls and CAD patients. The magnitude of change in the autonomic nervous modulation in CAD patients was slightly smaller than that in the controls.

Conclusion • FR may be used as an efficient adjunct to the therapeutic regimen to increase the vagal modulation and decrease blood pressure in both healthy people and CAD patients. (*Altern Ther Health Med.* 2011;17(4):8-14.)

Wan-An Lu, MD, PhD, is an assistant professor in the Institute of Cultural Asset and Reinvention, Fo-Guang University, Ilan, Taiwan. Gau-Yang Chen, MD, PhD, is an associate professor in the Institute of Biomedical Engineering, National Yang-Ming University, Taipei, Taiwan. Cheng-Deng Kuo, MD, PhD, is the principal investigator in the Biophysics Laboratory in the Department of Research and Education, Taipei Veterans General Hospital, Taipei, Taiwan.

Corresponding author: Cheng-Deng Kuo, MD, PhD
E-mail address: cdkuo@vghtpe.gov.tw

Age-related changes in autonomic nervous system control of the circulation are a key feature of age-associated cardiovascular disease.[1,2] Vagal modulation was found to be decreased in various physiological and pathological conditions, such as aging,[3] acute myocardial infarction,[4] diabetes mellitus,[5] chronic renal failure,[6] congestive heart failure,[7] and coronary artery disease (CAD).[8-12] In addition, the reduction in cardiac vagal modulation, as evaluated by spectral heart rate variability (HRV) analysis, correlated with angiographic severity, independent of previous myocardial infarction, location of diseased coronary arteries, and left ventricular function.[12] Vagal stimulation has been shown to have an antiarrhythmic effect in animal models of acute ischemia[13,14] and can terminate ventricular tachycardia in humans.[15] A pharmacological method of using transdermal scopolamine as a cardiac vagal enhancer was tried in patients with acute myocardial infarction[16-19] and congestive heart failure[20] and proved to be effective in improving the autonomic indices that are associated with high mortality. However, the safety, tolerability, and efficacy of long-term transdermal scopolamine treatment remain to be clarified.[21]

Complementary and alternative medicine (CAM) has long been used to postpone the aging process and to reverse disease progression. CAM therapies have lasted because in some cases they work as well as or better than allopathic medicine. With growing public interest in CAM, it is important for medical professionals to examine the effectiveness of CAM techniques. Among many therapeutic modalities, reflexology is often used as an antiaging CAM technique.[22]

Reflexology is a form of complementary medicine that involves using massage to reflex areas in the feet and the hands.[23-26] By stimulating and applying pressure to certain areas, one can increase blood circulation and promote specific bodily and muscular functions. It has been estimated that more than 20 million Americans have seen reports of the effectiveness of reflexology on television and have read about this natural technique of healing in national magazines and newspapers.[23] Several books have been written to propagate the rejuvenation effects of reflexology.[24-26] Though Wang et al[27] reviewed five studies of reflexology in the literature and concluded that there is no evidence for any specific effect of reflexology in any conditions with the exception of urinary symptoms associated with multiple sclerosis, others have shown significant effects using reflexology. The feet are the most common areas treated with reflexology.[24] Sudmeier et al[28] showed that foot reflexology (FR) is effective in changing renal blood flow. Stephenson et al[29,30] have shown that FR can relieve pain in patients with metastatic cancer and decrease anxiety in patients with breast and lung cancer. Ergonomically created footwear also has been invented to provide relaxation, reduce swelling, induce blood flow, and rejuvenate the muscles and nerves in the ankle and foot area.[31]

Since patients with anxiety or pain are expected to have an elevated sympathetic and a depressed vagal modulation,[32,33] it is possible that treatment with FR can lower sympathetic modulation and raise vagal modulation. It is therefore worthy of investigating whether FR can have an effect on the autonomic nervous modulation in normal controls and in patients with CAD.

MATERIALS AND METHODS

Study Participants

Coronary arteriography was performed in patients with angina pectoris, unstable angina, previous myocardial infarction, or other evidence of myocardial ischemia. A panel of cardiologists interpreted the angiograms. The coronary arteries and branches were divided into 15 segments according to the Ad Hoc Committee for Grading of Coronary Artery Disease of the American Heart Association.[34] Only the luminal narrowing in the following segments was used in the final assessment: segment 1-3 for the right coronary artery, segments 6 and 7 for the left anterior descending branch, segments 11 and 12 for the circumflex branch, and segment 5 for the left main coronary artery. By confining the analysis to these segments alone, only those patients who had significant obstruction in the main epicardial coronary arteries were included in this study. Stenosis was considered to be significant if a luminal narrowing >50% was present. Patients without stenosis or with luminal narrowing <30% were classified as the control group. Coronary artery bypass graft surgery was suggested for patients who refused percutaneous coronary intervention or whose lesions were not suitable for it. Patients with CAD preparing for coronary artery bypass graft surgery were recruited as the study group. Patients with angiographically patent coronary arteries were recruited as the control group. Hypertension was defined as systolic blood pressure >140 mmHg or diastolic blood pressure >90 mmHg.[35] Hyperlipidemia was defined as total cholesterol >200 mg/dL or low density lipoprotein cholesterol >100 mg/dL.[36] Patients who had atrial fibrillation or coexisting valvular heart disease or were using class I antiarrhythmic medication were excluded from this study. All participants were requested to refrain from alcohol or caffeine ingestion 24 hours before the study. The hospital Institutional Review Board approved this study. The procedure was fully explained to the participants, and written informed consent was obtained from them before the study.

Equipment

The electrocardiogram (ECG) signals were recorded using a multichannel recorder (Biopac MP150 with 16 channels, MP150CE/UIM100C/ECG100C, BIOPAC Systems, Inc, Goleta, California) from conventional lead II, and blood pressure was measured by using a sphygmomanometer (Kenlu-model K-300 Sphygmomanometer, Di Tai Precision Ent Co Ltd, Kaohsiung City, Taiwan) on each participant lying in a supine position. The analog signals of ECG were transformed to digital signals by using an analog-to-digital converter with a sampling rate of 400 Hz. Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MABP), and pulse pressure (PP) were obtained from each participant before FR using the sphygmomanometer.

Study Protocol

Before FR, each participant rested in a supine position for 5 minutes, and then 10 minutes of continuous ECG signals and blood pressure data were recorded. After baseline ECG recording and blood pressure measurement, the participant received FR for 60 minutes. The ECG recording and blood pressure measurements were repeated 30 and 60 minutes after FR. All procedures were performed in a bright and quiet room with a room temperature of 24°C to 25°C and humidity of 54% to 55%.

FR was performed on participants lying in a comfortable supine position by a certified foot reflexologist from the Taiwan Association of Reflexology using the techniques of Father Josef's FR.[37] The reflexologist used the thumb and fingers to apply pressure to stimulate all reflex zones in both feet, which correspond to all organs, glands, and body parts. The technique of the thumb and fingers resembles a caterpillar-like action in reflexology.[38] Grapeseed oil is used during FR to prevent friction and possible discomfort because it is nonsticky and odorless and absorbs easily into the skin.[39,40]

Heart Rate Variability Analysis

R-wave–detecting software written with the help of Matlab R13 software (MathWorks Inc, Natick, Massachusetts) was used to identify the peaks of the R waves in the recorded ECG signals. The RR intervals (the time intervals between two consecutive R waves in the electrocardiogram, RRI) were then calculated after eliminating ectopic beats. If the percentage of ectopic beats was greater than 5%, then the participant was excluded from analysis. The last 512 stationary RRI were used for HRV analysis.

The mean, standard deviation (SD_{RR}) and coefficient of variation (CV_{RR}) of the 512 stationary RRI were calculated using a standard formula for each participant. The power spectra of RRI were obtained by means of fast Fourier transformation (Mathcad, Mathsoft Inc, Cambridge, Massachusetts). 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.

The Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology have suggested that the power within the frequency range of 0.04-0.4 Hz be used for the normalization of LFP and HFP.[41] Since this frequency range covers only the frequency ranges of LFP and HFP but not 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, which covers the frequency ranges of VLFP, LFP, and HFP, to normalize VLFP, LFP, and HFP in this study. The normalized very low-frequency power (nVLFP = VLFP/TP) was then used as the index of vagal withdrawal, renin-angiotensin modulation, and thermoregulation[42-44]; the normalized low-frequency power (nLFP = LFP/TP) was used as the index of combined sympathetic and vagal modulation[45]; the normalized high-frequency power (nHFP = HFP/TP) was used as the index of vagal modulation; and the low-/high-frequency power ratio (LFP/HFP) was used as the index of sympathovagal balance.[46]

Statistical Analysis

Values of HRV and blood pressure measures were presented as median (25 percentile-75 percentile). Friedman repeated measures analysis of variance on ranks (SigmaStat statistical software, Jandel Scientific, San Rafael, California) was employed to compare the HRV and blood pressure measures among before FR, 30 minutes after FR, and 60 minutes after FR. Significant difference was further analyzed by pairwise comparison using the Student Newman–Keuls test. The Mann-Whitney rank sum test was employed to compare the HRV and blood pressure measures between CAD patients and controls.

To correct for baseline differences on the comparison of HRV and blood pressure measures between CAD patients and controls, the percentage changes in HRV and blood pressure measures in each participant 30 and 60 minutes after FR were calculated using the following formulas:

$$\%X_{30} = [(X30_{min} - X_{before})/(X_{before})] \times 100$$
$$\%X_{60} = [(X60_{min} - X_{before})/(X_{before})] \times 100,$$

where X represents the variable to be compared. The Mann-Whitney rank sum test was used to compare $\%X_{30}$ and $\%X_{60}$ between controls and patients with CAD. Wilcoxon signed rank test was employed to compare $\%X_{30}$ with $\%X_{60}$ in both controls and patients with CAD. A $P < .05$ was considered statistically significant.

RESULTS

General Characteristics

The percentage of deletion of ectopic beats due to atrial or ventricular arrhythmia was >5% in two patients in the CAD group and three participants in the control group. Thus, only 20 out of 22 patients in the CAD group and 17 out of 20 patients in the control group were included in the final statistical analysis. Table 1 shows the baseline characteristics of the control and CAD groups. There were 17 men and 3 women (between 52.5 and 66.0 years of age with an average of 62.0 years) in the CAD group and 15 men and 2 women (between 52.0 and 66.0 years of age with an average of 56.0 years) in the control group.

Effect of Foot Reflexology on Blood Pressure

TABLE 1 Baseline Characteristics of the Control and Coronary Artery Disease (CAD) Groups*

	Control Group (n = 17)	CAD Group (n = 20)	*P* value
Age (y)	56.0 (52.0-66.0)	62.0 (52.5-66.0)	NS
Gender (M/F)	15/2	17/3	NS
Body height (cm)	161.0 (156.0-164.3)	163.5 (157.0-173.0)	NS
Body weight (kg)	59.0 (51.5-70.3)	64.0 (58.3-74.5)	NS
BMI (m^2/kg)	22.2 (21.1-25.0)	24.1 (22.2-26.4)	NS
History			
Previous MI	0	4	NA
Hypertension	12	15	NS
Diabetes mellitus	6	7	NS
Hyperlipidemia	6	7	NS
Current smoker	7	11	NS
Medication			
Beta-blocker	10	15	NS
Calcium antagonist	11	17	NS
Nitrates	13	19	NS
ACE inhibitor	6	10	NS
ARB	1	3	NS
Digitalis	1	1	NS
Aspirin	12	18	NS
Clopidogrel	5	10	NS
Ticlopidine	2	4	NS
Clinical status			
One-vessel disease	0	8	NA
Two-vessel disease	0	8	NA
Three-vessel disease	0	4	NA
Left main disease	0	5	NA
Left ventrical aneurysm	0	4	NA

*Values are numbers of patients or medians (25-75 percentile).
Abbreviations: BMI, body mass index; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; MI, myocardial infarction; NS, not significant ($P > .05$); NA, not assessed.

Table 2 shows the sequential changes in blood pressures after FR in both groups of participants. The SBP, DBP, and MABP decreased significantly after FR in both groups. The PP decreased 30 minutes after FR in both groups and elevated to pre-FR level 60 minutes after FR in the control group.

Table 3 shows the percentage changes in blood pressures after RF in both groups of participants. In the control group, the percentage decrease in SBP and PP 30 minutes after FR was larger than that 60 minutes after FR. In the CAD group, the percentage decrease in PP 30 minutes after FR was larger than that of 60 minutes after FR. No significant difference in the percentage change in blood pressures after FR was found between the two groups.

Effect of Foot Reflexology on Heart Rate Variability

Table 4 shows the effects of FR on the time and frequency domain HRV measures in patients with CAD and control group.

TABLE 2 Effect of Foot Reflexology (FR) on Blood Pressure*

	Before FR	30 Min After FR	60 Min After FR
Control group (n = 17)			
SBP (mmHg)	136.0 (129.8-141.8)	115.0 (109.8-123.3)‡	124.0 (114.0-133.5)‡§
DBP (mmHg)	75.0 (72.8-80.3)	67.0 (63.0-73.3)‡	67.0 (62.8-75.0)‡
MABP (mmHg)	95.0 (87.5-101.3)	83.0 (78.3-88.0)‡	84.0 (79.8-86.8)‡
PP (mmHg)	60.0 (54.3-65.0)	48.0 (45.5-52.3)‡	54.0 (47.8-61.3)§
CAD group (n = 20)			
SBP (mmHg)	153.5 (139.5-163.5)†	134.5 (123.0-148.0)†‡	134.0 (125.5-147.0)†‡
DBP (mmHg)	84.5 (78.0-93.0)†	80.5 (70.5-84.5)†	75.5 (69.5-85.0)†‡
MABP (mmHg)	102.0 (95.5-113.0)†	96.5 (88.5-104.0)†	94.0 (85.0-99.0)†‡
PP (mmHg)	64.0 (60.0-72.0)	50.5 (46.5-66.5)‡	57.0 (53.0-64.0)‡

*Values presented are medians (25-75 percentile).
†$P < .05$ between controls and patients with CAD.
‡$P < .05$ vs before FR.
§$P < .05$ vs 30 min after FR.
Abbreviations: CAD, coronary artery disease; SBP, systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure; PP, pulse pressure.

TABLE 3 Percentage Changes in Blood Pressure After Foot Reflexology*

	%X30	%X60
Control group (n = 17)		
SBP; %	−15.2 (−18.5 to −8.3)	−8.3 (−12.5 to −2.8)†
DBP; %	−5.5 (−8.2 to −3.7)	−8.1 (−14.3 to −4.1)
MABP; %	−11.0 (−16.8 to −6.9)	−9.3 (−16.7 to −3.8)
PP; %	−15.9 (−26.7 to −10.8)	−8.5 (−16.1 to −1.2)†
CAD group (n = 20)		
SBP; %	−10.2 (−14.6 to −3.8)	−6.6 (−14.1 to −3.1)
DBP; %	−8.1 (−12.4 to 0.0)	−8.1 (−9.7 to −4.4)
MABP; %	−5.6 (−14.0 to −0.1)	−7.3 (−12.5 to −4.1)
PP; %	−13.5 (−23.7 to −5.3)	−9.1 (−16.7 to 1.7)†

*Values presented are medians (25-75 percentile).
†$P < .05$ vs $\%X_{30}$.
Abbreviations: CAD, coronary artery disease; SBP, systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure; PP, pulse pressure.

Thirty and 60 minutes after RF, the nHFP was significantly increased, whereas the nVLFP was significantly decreased in both groups of participants s as compared with those before RF. The VLFP and TP after FR were significantly decreased in the control group. The mean RRI was significantly increased, and the heart rate was significantly decreased after RF in CAD patients. Although the SD_{RR}, TP, VLFP, LFP, and HFP of the CAD patients before FR were not significantly different from those of the controls, they were significantly larger than those of the controls 60 minutes after FR. However, the relative HRV measures including nVLFP, nLFP, nHFP and LFP/HFP were not significantly different between the controls and the CAD patients both before FR and 60 minutes after FR.

Table 5 shows the percentage changes in HRV measures after RF in both groups of participants. The percentage decrease in SD_{RR}, TP, VLFP, and LFP and the percentage increase in heart rate 60 minutes after FR were larger than those of 30 minutes after FR in controls. There were no significant differences in the percentage changes in all HRV measures between 30 minutes and 60 minutes after FR in the CAD patients.

The percentage increase in mean RRI 30 minutes after FR in the CAD group was larger than that in the control group, whereas the percentage decrease in heart rate 30 minutes after FR in the CAD group was smaller than that in the control group. Similarly, the percentage increase in mean RRI, SD_{RR}, TP, VLFP, LFP, and HFP in the CAD group 60 minutes after FR was larger than those in the control group, whereas the percentage decrease in heart rate and nLFP in the CAD group 60 minutes after FR was smaller than those in the control group.

Effect of Beta-blockers on Heart Rate Variability

Table 6 shows that there were no significant differences in all HRV measures between participants using or not using beta-blockers in either control or CAD group and between the control and CAD groups whether they were using beta-blockers or not. There are no differences in the effects of FR on patients whether they were using or not using beta-blocker medication.

DISCUSSION

Ludwig has defined aging as a time-dependent, irreversible shift from environmental to intrinsic causation of disease.[47] This intrinsic pathogenesis has two components: the first one is genetic and beyond the reach of contemporary health care and the second one entails the growing number of degenerative lesions due to viral agents as well as carcinogenesis. Bonnemeier et al have shown that normal aging is associated with a constant decline of cardiac vagal modulation due to a significant decrease of nocturnal parasympathetic activity.[48] It has also been shown that depressed vagal modulation is associated increased risk of sudden death in patients with CAD, and the experimental evidence also suggests a causal relationship.[49-52] With the adverse prognostic implication of reduced cardiac vagal activity in its susceptibility to life-threatening arrhythmia,[51-53] any intervention that can enhance the vagal modulation will be beneficial to patients, especially for those at high risk for life-threatening arrhythmia. Exercise and medication have been found to increase the vagal modulation of the study participants,[16-20,53-55,]

TABLE 4 Effect of Foot Reflexology (FR) on Heart Rate Variability Measures (HRV)*

	Before FR	30 Min After FR	60 Min After FR
Control group (n = 17)			
Mean RRI (ms)	861.9 (781.3-948.7)	839.5 (776.0-981.0)	827.0 (765.7-921.6)
Heart rate (bpm)	69.6 (63.2-76.8)	71.5 (61.2-77.3)	72.6 (65.1-78.4)
SD_{RR} (ms)	52.3 (48.5-60.3)	48.1 (45.6-57.7)	44.8 (42.3-49.5)
CV_{RR} (%)	6.0 (5.4-6.6)	5.9 (5.4-6.4)	5.4 (5.3-6.0)
TP (ms^2)	946 (784-1428)	808 (642-1242)	640 (552-866)‡§
VLFP (ms^2)	312 (186-497)	226 (135-385)‡	135 (106-179)‡§
LFP (ms^2)	251 (172-406)	206 (152-390)	188 (153-276)
HFP (ms^2)	369 (311-547)	390 (286-532)	344 (268-428)
nVLFP (nu)	33.1 (28.4-41.9)	27.8 (19.0-33.8)‡	20.2 (14.5-26.1)‡
nLFP (nu)	28.3 (20.5-29.4)	26.9 (23.7-29.0)	29.0 (25.6-32.5)
nHFP (nu)	38.6 (33.4-46.2)	47.2 (40.9-55.0)‡	50.7 (43.7-54.5)‡§
LFP/HFP	0.71 (0.53-0.82)	0.61 (0.51-0.66)	0.59 (0.49-0.67)
CAD group (n = 20)			
Mean RRI (ms)	831.1 (762.3-903.0)	917.5 (849.7-928.6)‡	911.0 (861.6-922.4)‡
Heart rate (bpm)	72.2 (66.5-78.7)	65.4 (64.6-70.6)‡	65.9 (65.1-69.6)‡
SD_{RR} (ms)	52.6 (45.2-62.5)	53.5 (49.4-66.6)	55.2 (49.6-60.7)†
CV_{RR} (%)	6.3 (5.5-7.2)	6.0 (5.5-7.1)	5.8 (5.4-6.5)
TP (ms^2)	981 (694-1526)	979 (886-1630)	1134 (931-1499)†
VLFP (ms^2)	257 (174-545)	233 (163-398)	212 (153-346)†
LFP (ms^2)	271 (208-491)	293 (278-407)	322 (259-449)†
HFP (ms^2)	470 (277-555)	515 (413-648)	527 (457-732)†
nVLFP (nu)	30.6 (24.9-32.9)	23.5 (17.7-29.0)‡	20.0 (14.2-29.0)‡
nLFP (nu)	28.4 (25.8-31.5)	29.2 (28.1-31.7)	29.5 (27.4-30.7)
nHFP (nu)	42.8 (34.8-46.3)	46.4 (40.9-50.9)‡	52.1 (42.9-55.4)‡
LFP/HFP	0.66 (0.53-0.83)	0.62 (0.52-0.77)	0.57 (0.52-0.66)

*Values presented are medians (25-75 percentile).
†$P < .05$ between normal controls and patients with CAD.
‡$P < .05$ vs before FR.
§$P < .05$ vs 30 min after FR.
Abbreviations: CAD, coronary artery disease; RRI, RR intervals; SD_{RR}, standard deviation of RR; CV_{RR}, coefficient of variation of RR; TP, total power; VLFP, very low-frequency power; LFP, low-frequency power; HFP, high-frequency power; nVLFP, normalized very low-frequency power; nLFP, normalized low-frequency power; nHFP, normalized high-frequency power; LFP/HFP, low-/high-frequency power ratio.

but this study showed that foot reflexology can also increase vagal modulation.

Reflexology is the study of working on the specific reflex points (areas) on the hands, feet, and ears that mirror the whole body in order to relax and relieve stress and pain.[23-26] In clinical terms, reflexology is the application of pressure, primarily but not limited to the feet, hands, or ears, that causes a physiological response in the body. Many studies have examined the efficacy of reflexology. However, controversy existed regarding efficacy of reflexology.[27,56-60] Frankel found that the frequency of sinus arrhythmia after reflexology and FM was increased by 43.9% and 34.1%, respectively; he suggested a "neuro theory" whereby reflexology and foot massage alter the baroreceptor reflex sensitivity by stimulating the sensory nervous system in the feet.[56] Hattan et al have investigated the impact of foot massage and guided relaxation on the well-being of patients who had undergone coronary artery bypass graft surgery and demonstrated that these interventions appear to be effective noninvasive techniques for promoting psychological well-being in this patient group.[57] Some studies have pointed out that reflexology possesses the potential to provide relief of pain and symptoms and induce relaxation.[25,58,59] Hayes and Cox[60] also demonstrated that a 5-minute foot massage had the potential effect of increasing relaxation as evidenced by a significant decrease in heart rate, blood pressure, and respiration during the brief foot massage intervention administered to critically ill patients in intensive care.

In this study, we found that FR results in positive effects on blood pressure and autonomic nervous modulation in both control group and patients with CAD. The nHFP was significantly increased after FR, whereas the nVLFP and LFP/HFP were significantly decreased after FR, in both control and CAD groups. This result suggested that a higher vagal modulation, lower sympathetic modulation, renin-angiotensin modulations and thermoregulatory activity can be observed following 60 minutes of FR in both angiographically patent controls and CAD patients. However, the increase in mean RRI, SD_{RR}, TP, VLFP, LFP, and HFP in the CAD group 60 minutes after FR was still present, whereas these measures were decreased in the control group 60 minutes after FR (Table 4). It seems that the beneficial effect of FR on HRV measures, especially on those measures

TABLE 5 The Percentage Changes in Heart Rate Variability Measures After Foot Reflexology*

	%X30	%X60
Control group (n = 17)		
Mean RRI; %	–0.6 (–4.0 to 4.8)‡	–4.2 (–9.1 to 0.9)
Heart rate; %	0.6 (–4.6 to 4.0)‡	4.3 (–0.8 to 10.0)‡
SD_{RR}; %	0.0 (–10.4 to 6.5)	–13.3 (–17.6 to –1.2)‡
CV_{RR}; %	–0.5 (–8.7 to 8.1)	–4.6 (–12.5 to 1.1)
TP; %	–5.7 (–21.9 to 18.4)	–28.0 (–36.0 to –8.4)‡
VLFP; %	-12.2 (–38.6 to 3.6)	–56.0 (–69.8 to –33.9)‡
LFP; %	0.0 (–15.7 to 19.1)	–23.2 (–39.6 to 13.3)‡
HFP; %	3.6 (–9.1 to 30.2)	–6.9 (–30.0 to 13.0)
nVLFP; %	–9.3 (–42.5 to 1.1)	–37.3 (–49.6 to –26.5)
nLFP; %	1.0 (–9.2 to 24.7)	14.1 (–2.9 to 23.5)
nHFP; %	9.9 (–0.0 to 41.3)	18.0 (7.8 to 52.0)
LFP/HFP; %	–14.3 (–28.5 to 16.6)	–5.7 (–23.2 to 7.9)
CAD group (n = 20)		
Mean RRI; %	6.6 (3.1 to 11.1)†	10.9 (3.5 to 17.7)†
Heart rate; %	–6.2 (–10.0 to –3.1)†	–9.9 (–15.0 to –3.4)†
SD_{RR}; %	4.3 (–8.7 to 23.7)	3.8 (–6.6 to 11.0)†
CV_{RR}; %	0.0 (–12.8 to 12.4)	–6.0 (–10.4 to –2.0)
TP; %	11.3 (–25.1 to 58.9)	14.5 (–13.3 to 38.5)†
VLFP; %	–0.3 (–39.9 to 21.6)	–14.5 (–40.6 to 10.3)†
LFP; %	7.6 (–22.3 to 72.8)	13.8 (–6.7 to 40.7)†
HFP; %	10.0 (–13.2 to 53.3)	32.4 (6.0 to 59.2)†
nVLFP; %	–16.4 (–28.2 to –2.9)	–18.4 (–40.6 to –4.5)
nLFP; %	0.5 (–6.7 to 7.1)	2.0 (–5.1 to 11.7)†
nHFP; %	8.9 (0.0 to 25.9)	15.1 (1.3 to 33.1)
LFP/HFP; %	–11.2 (–23.3 to 2.1)	–9.1 (–31.5 to 3.7)

*Values presented are medians (25-75 percentile).
†$P < .05$ between normal controls and patients with CAD.
‡$P < .05$ vs before FR.
§$P < .05$ vs 30 min after FR.
Abbreviations: CAD, coronary artery disease; RRI, RR intervals; SD_{RR}, standard deviation of RR; CV_{RR}, coefficient of variation of RR; TP, total power; VLFP, very low-frequency power; LFP, low-frequency power; HFP, high-frequency power; nVLFP, normalized very low-frequency power; nLFP, normalized low-frequency power; nHFP, normalized high-frequency power; LFP/HFP, low-/high-frequency power ratio.

TABLE 6 Effect of Beta-blockers on Heart Rate Variability in the Control and Coronary Artery Disease (CAD) Groups*

	Control Group (n = 17)	CAD Group (n = 20)
Without beta-blockers (n = 12)		
TP (ms2)	861.6 (696.0-9876.9)	1415.0 (467.0-2036.0)
VLFP (ms2)	312.0 (176.2-406.9)	218.0 (165.3-733.0)
LFP (ms2)	174.5 (158.5-255.1)	382.0 (127.7-641.2)
HFP (ms2)	373.3 (228.2-493.5)	460.5 (194.4-927.8)
nVLFP (nu)	36.0 (24.3-41.9)	31.2 (26.2-37.7)
nLFP (nu)	23.8 (20.2-29.2)	28.2 (26.4-31.3)
nHFP (nu)	42.9 (35.4-48.1)	37.7 (33.2-48.6)
LFP/HFP	0.53 (0.48-0.81)	0.82 (0.52-0.90)
With beta-blockers (n = 25)		
TP (ms2)	1083.1 (827.2-2104.0)	962.5 (7484.1-1375.0)
VLFP (ms2)	311.9 (197.5-514.1)	288.4 (173.8-444.4)
LFP (ms2)	337.1 (177.9-413.1)	256.8 (219.4-437.9)
HFP (ms2)	354.1 (318.7-625.3)	480.2 (318.4-546.6)
nVLFP (nu)	33.0 (30.3-41.3)	30.5 (24.1-32.7)
nLFP (nu)	28.3 (21.5-30.1)	28.6 (25.2-32.4)
nHFP (nu)	38.5 (32.6-44.0)	43.2 (35.5-46.2)
LFP/HFP	0.74 (0.59-0.81)	0.65 (0.53-0.82)

*Values presented are medians (25-75 percentile).
Abbreviations: TP, total power; VLFP, very low-frequency power; LFP, low-frequency power; HFP, high-frequency power; nVLFP, normalized very low-frequency power; nLFP, normalized low-frequency power; nHFP, normalized high-frequency power; LFP/HFP, low-/high-frequency power ratio.

related to vagal modulation, last longer in patients with CAD than in the controls. The mechanism responsible for this differential effect was not clear at present because it was not investigated in this study. We speculate that the FR-related autonomic nervous effect of increasing vagal and decreasing sympathovagal balance may be more evident in those patients who have depressed vagal modulation and enhanced sympathetic modulation, such as patients with CAD. Further studies are needed to disclose the underlying mechanism.

Other considerations include that the manipulation of FR on a participant did not allow him or her to rest uninterruptedly. Thus, a participant not receiving FR is not a good control to contrast the effect of FR on that person. If a control is going to be used to contrast the effect of FR, manipulating some area other than the foot that has no reflex points on it for the same period of time may be a better choice than a participant not receiving FR. According to the traditional Oriental medicine, no area over the whole body can be found that can be stimulated by pressure and massage without causing a physiological response in the body. Therefore, the participant not receiving FR in either group was not designed as a control to contrast the effect of FR in this study.

To know the differences in the effects of FR on patients who are on different medications and the effects of those medications on FR, we chose to compare the effect of beta-blockers on the HRV measures in both control and CAD groups. We found that there were no significant differences in all HRV measures between participants using or not using beta-blockers in either control or CAD group and between the control and CAD groups whether they were using beta-blockers or not (Table 6). Thus, there are no differences in the effects of FR on CAD patients whether or not they are on beta-blocker medication, and the beta-blockers do not significantly influence the effect of FR on the autonomic nervous modulation of the participants.

In conclusion, a higher vagal modulation, lower sympathetic modulation, and lower blood pressures can be observed following 60 minutes of FR in both angiographically patent controls and CAD patients. Though the magnitude of change in the autonomic nervous modulation of the CAD patients was slightly smaller than that of the controls, FR is a complementary therapeutic method to allopathic medical care that is simple and safe for almost everyone. FR requires very little time and expense, no special equipment, and no medication and can be performed practically anywhere. Since the mortality risk due to acute myocardial infarction is lower in patients with higher vagal modulation and is higher in patients with higher sympathetic modulation, our research suggests that FR is a safe, low-cost adjunct treatment that can be used as an effective physiological vagal enhancer and sympathetic suppressor in both control and CAD patients to benefit cardiovascular health.

Acknowledgments

This study was supported by the project VGHUST93-P1-08 of the Joint Research Program of Veterans General Hospital and University System, Taiwan, and the project CCMP97-RD047 of the Committee on Chinese Medicine and Pharmacy, Department of Health, Taipei, Taiwan.

REFERENCES

1. Ferrari AU, Radaelli A, Centola M. Invited review: aging and the cardiovascular system. *J Appl Physiol.* 2003;95(6):2591-2597.
2. Seals DR, Esler MD. Human ageing and the sympathoadrenal system. *J Physiol.* 2000;528(Pt 3):407-417.
3. Lipsitz LA, Mietus J, Moody GB, Goldberger AL. Spectral characteristics of heart rate variability before and during postural tilt. Relations to aging and risk of syncope. *Circulation.* 1990;81(6):1803-1810.
4. Lombardi F, Sandrone G, Pernpruner S, et al. Heart rate variability as an index of sympathovagal interaction after acute myocardial infarction. *Am J Cardiol.* 1987;60(16):1239-1245.
5. Lishner M, Akselrod S, Avi VM, Oz O, Divon M, Ravid M. Spectral analysis of heart rate fluctuations. A non-invasive, sensitive method for the early diagnosis of autonomic neuropathy in diabetes mellitus. *J Auton Nerv Syst.* 1987;19(2):119-125.
6. Axelrod S, Lishner M, Oz O, Bernheim J, Ravid M. Spectral analysis of fluctuations in heart rate: an objective evaluation of autonomic nervous control in chronic renal failure. *Nephron.* 1987;45(3):202-206.
7. Saul JP, Arai Y, Berger RD, Lilly LS, Colucci WS, Cohen RJ. Assessment of autonomic regulation in chronic congestive heart failure by heart rate spectral analysis. *Am J Cardiol.* 1988;61(15):1292-1299.
8. Ryan C, Hollenberg M, Harvey DB, Gwynn R. Impaired parasympathetic responses in patients after myocardial infarction. *Am J Cardiol.* 1976;37(7):1013-1018.
9. Tristani FE, Kamper DG, McDermott DJ, Peters BJ, Smith JJ. Alternations of postural and Valsalva responses in coronary heart disease. *Am J Physiol.* 1977;233(6):H694-H699.
10. Bennett T, Wilcox RG, Hampton JR. Cardiovascular reflexes in patients after myocardial infarction. Effect of long-term treatment with beta-adrenoceptor antagonists. *Br Heart J.* 1980;44(3):265-270.
11. Airaksinen KE, Ikäheimo MJ, Linnaluoto MK, Niemelä M, Takkunen JT. Impaired vagal heart rate control in coronary artery disease. *Br Heart J.* 1987;58(6):592-597.
12. Hayano J, Sakakibara Y, Yamada M, et al. Decreased magnitude of heart rate spectral components in coronary artery disease. Its relation to angiographic severity. *Circulation.* 1990;81(4):1217-1224.
13. Kent KM, Smith ER, Redwood DR, Epstein SE. Electrical stability of acutely ischemic myocardium. Influences of heart rate and vagal stimulation. *Circulation.* 1973;47(2):291-298.
14. Vanoli E, De Ferrari GM, Stramba-Badiale M, Hull SS Jr, Foreman RD, Schwartz PJ. Vagal stimulation and prevention of sudden death in conscious dogs with a healed myocardial infarction. *Cir Res.* 1991;68(5):1471-1481.
15. Waxman MB, Wald RW. Termination of ventricular tachycardia by an increase in cardiac vagal drive. *Circulation.* 1977;56(3):385-391.
16. Casadei B, Pipilis A, Sessa F, Conway J, Sleight P. Low doses of scopolamine increase cardiac vagal tone in the acute phase of myocardial infarction. *Circulation.* 1993;88(2);353-357.
17. Pedretti R, Colombo E, Sarzi Braga S, Carù B. Influence of transdermal scopolamine on cardiac sympathovagal interaction after acute myocardial infarction. *Am J Cardiol.* 1993;72(5):384-392.
18. Vybiral T, Glaeser DH, Morris G, et al. Effects of low dose transdermal scopolamine on heart rate variability in acute myocardial infarction. *J Am Coll Cardiol.* 1993;22(5):1320-1326.
19. De Ferrari GM, Mantica M, Vanoli E, Hull SS Jr, Schwartz PJ. Scopolamine increases vagal tone and vagal reflexes in patients after myocardial infarction. *J Am Coll Cardiol.* 1993;22(5):1327-1334.
20. La Rovere MT, Mortara A, Pantaleo P, Maestri R, Cobelli F, Tavazzi L. Scopolamine improves autonomic balance in advanced congestive heart failure. *Circulation.* 1994;90(2):838-843.
21. Sneddon JF, Bashir Y, Ward DE. Vagal stimulation after myocardial infarction: accentuating the positive. *J Am Coll Cardiol.* 1993;22(5):1335-1337.
22. Gammack JK, Morley JE. Anti-aging medicine—the good, the bad, and the ugly. *Clin Geriatr Med.* 2004;20(2):157-177.
23. Carter M, Weber T. *Body Reflexology: Healing at Your Fingertips.* New York, NY: Parker Publishing Co; 1994.
24. Palma RP. *Hand and Foot Reflexology: Do It Yourself Treatment and Rejuvenation.* Concepcion, Tarlac: R.P. Palma, 1986.
25. Chia M. *Chi Self-Massage: The Taoist Way of Rejuvenation.* New York, NY: Healing Tao Books; 1986.
26. Weber KK. *Healing Self-Massage: Over 100 Simple Techniques for Re-energizing Body and Mind.* New York, NY: Sterling Publishing; 2005.
27. Wang MY, Tsai PS, Lee PH, Chang WY, Yang CM. The efficacy of reflexology: systematic review. *J Adv Nurs.* 2008;62(5):512-520.
28. Sudmeier I, Bodner G, Egger I, Mur E, Ulmer H, Herold M. Changes of renal blood flow during organ-associated foot reflexology measured by color Doppler sonography [article in German]. *Forsch Komplementarmed.* 1999;6(3):129-134.
29. Stephenson N, Dalton JA, Carlson J. The effect of foot reflexology on pain in patients with metastatic cancer. *Appl Nurs Res.* 2003;16(4):284-286.
30. Stephenson NL, Weinrich SP, Tavakoli AS. The effects of foot reflexology on anxiety and pain in patients with breast and lung cancer. *Oncol Nurs Forum.* 2000;27(1):67-72.
31. Ballentine K. Ergonomic ped-hydro cavity agitation for therapeutic assistance and relaxation use. US patent 2006/0149318 A1.
32. Dishman RK, Nakamura Y, Garcia ME, Thompson RW, Dunn AL, Blair SN. Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. *Int J Psychophysiol.* 2000;37(2):121-133.
33. Thompson JJ, Elsenbruch S, Harnish MJ, Orr WC. Autonomic functioning during REM sleep differentiates IBS symptom subgroups. *Am J Gastroenterol.* 2002;97(12):3147-3153.
34. Austen WG, Edwards JE, Frye RL, et al. A reporting system on patients evaluated for coronary artery disease. Report of the Ad Hoc Committee for Grading of Coronary Artery Disease, Council on Cardiovascular Surgery, American Heart Association. *Circulation.* 1975;51(4 Suppl):5-40.
35. Chobanian AV, Bakris GL, Black HR, et al; Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood Institute; National High Blood Pressure Education Program Coordinating Committee. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension.* 2003;42(6):1206-1252.
36. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of National Cholesterol Education Program (NCER) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in adults (Adult Treatment Panel III) final report. *Circulation.* 2002;106(25):3143-3421
37. J Eugster, Cheng EYC. *Father Josef's New Foot Reflexology.* Taipei, Taiwan: Cosmax Publishing, 2007:136-145.
38. Botting D. Review of literature on the effectiveness of reflexology. *Complement Ther Nurs Midwifery.* 1997;3(5):123-130.
39. Enzer S. *Reflexology: A Tool for Midwives.* Sydney, Australia: S Enzer; 2000.
40. Hulme J, Waterman H, Hillier VF. The effect of foot massage on patients' perception of care following laparoscopic sterilization as day case patients. *J Adv Nurs.* 1999;30(2):460-468.
41. No authors listed. 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.* 1996;93(5):1043-1065.
42. Taylor JA, Carr DL, Myers CW, Eckberg DL. Mechanisms underlying very-low-frequency RR-interval oscillations in humans. *Circulation.* 1998;98(6):547-555.
43. Thayer JF, Nabors-Oberg R, Sollers JJ 3rd. Thermoregulation and cardiac variability: a time-frequency analysis. *Biomed Sci Instrum*. 1997;34:252-256.
44. Fleisher LA, Frank SM, Sessler DI, Cheng C, Matsukawa T, Vannier CA. Thermoregulation and heart rate variability. *Clin Sci (Lond).* 1996;90(2):97-103.
45. Koizumi K, Terui N, Kollai M. Effect of cardiac vagal and sympathetic nerve activity on heart rate in rhythmic fluctuations. *J Auton Nerv Syst.* 1985;12(2-3):251-259.
46. Pagani M, Lombardi F, Guzzetti S, et al. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. *Circ Res.* 1986;59(2):178-193.
47. Ludwig FC. Senescence. Pathology facing medicine's ultimate issue. *Arch Pathol Lab Med.* 1981;105(9):445-451.
48. Bonnemeier H, Richardt G, Potratz J, et al. Circadian profile of cardiac autonomic nervous modulation in healthy subjects: differing effects of aging and gender on heart rate variability. *J Cardiovasc Electrophysiol.* 2003;14(8):791-799.
49. Kleiger RE, Miller JP, Bigger JT Jr, Moss AJ. Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *Am J Cardiol.* 1987;59(4):256-262.
50. La Rovere MT, Specchia G, Mortara A, Schwartz PJ. Baroreflex sensitivity, clinical correlates, and cardiovascular mortality among patients with a first myocardial infarction: a prospective study. *Circulation.* 1988;78(4):816-824.
51. Farrell TG, Paul V, Cripps TR, et al. Baroreflex sensitivity and electrophysiological correlates in patients after acute myocardial infarction. *Circulation.* 1991;83(3):945-952.
52. Schwartz PJ, La Rovere MT, Vanoli E. Autonomic nervous system and sudden cardiac death. Experimental basis and clinical observations of post-myocardial risk stratification. *Circulation.* 1992;85(1Suppl):I77-I91.
53. Goldsmith RL, Bigger JT Jr, Steinman RC, Fleiss JL. Comparison of 24-hour parasympathetic activity in endurance-trained and untrained young men. *J Am Coll Cardiol.* 1992;20(3):552-558.
54. Dibner-Dunlap ME, Eckberg DL, Magid NM, Cintrón-Treviño NM. The long-term increase of baseline and reflexly augmented levels of human vagal-cardiac nervous activity induced by scopolamine. *Circulation.* 1985;71(4):797-804.
55. Vybiral T, Bryg RJ, Maddens ME, et al. Effects of transdermal scopolamine on heart rate variability in normal subjects. *Am J Cardiol.* 1990;65(9):604-608.
56. Frankel BS. The effect of reflexology on baroreceptor reflex sensitivity, blood pressure and sinus arrhythmia. *Complement Ther Med.* 1997;5(2):80-84.
57. Hattan J, King L, Griffiths P. The impact of foot massage and guided relaxation following cardiac surgery: a randomized controlled trial. *J Adv Nurs.* 2002;37(2):199-207.
58. Degan M, Fabris F, Vanin F, et al. The effectiveness of foot reflexotherapy on chronic pain associated with a herniated disk [article in Italian]. *Prof Inferm.* 2000;53(2):80-87.
59. Oleson T, Flocco W. Randomized controlled study of premenstrual symptoms treated with ear, hand, and foot reflexology. *Obstet Gyn.* 1993;82(6):906-911.
60. Hayes J, Cox C. Immediate effects of a five-minute foot massage on patients in critical care. *Intensive Crit Care Nurs.* 1999;15(2):77-82.