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Effects of arm swing exercise training on cardiac autonomic modulation, cardiovascular risk factors, and electrolytes in persons aged 60–80 years with prehypertension: A randomized controlled trial

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ABSTRACT

Background/Objective: This study aimed to investigate the effect and carry-over effect of arm swing exercise (ASE) training on cardiac autonomic modulation, cardiovascular risk factors, and blood electrolytes in older persons with prehypertension.

Methods: Subjects were 50 individuals with prehypertension (aged 66.90 ± 5.50 yr, body mass index 23.84 ± 3.65 kg/m^2). They were randomly assigned into ASE group and control group. Subjects in the ASE group underwent an ASE training program for 3 months at a frequency of 30 min/day, 3 days/week. Subjects in the control group maintained their daily routine activities minus regular exercise. Blood pressure, heart rate variability (HRV), cardiovascular risk factors including blood glucose, lipid profile, high-sensitive C-reactive protein (hsCRP), and electrolytes were evaluated on 3 occasions: before and after the 3-month intervention, and 1 month after intervention ended.

Results: Following the 3-month intervention, systolic blood pressure (SBP) and serum hsCRP concentration were significantly lower, while serum high-density lipoprotein (HDL)-cholesterol, potassium (K^+), magnesium (Mg^{2+}) concentrations, standard deviation of normal R-R intervals (RMSSD) and high frequency (HF) power values were higher in the ASE group when compared with the control group ($p < 0.05$). At the 1-month follow-up interval, SBP and serum hsCRP concentration remained lower while serum HDL-cholesterol and K^+ concentrations remained higher in the ASE group as compared to the control group ($p < 0.05$).

Conclusion: ASE training decreased SBP and serum hsCRP concentration, increased serum HDL-cholesterol, K^+, and Mg^{2+} concentrations and increased RMSSD and HF power values in older persons with prehypertension. In addition, there were carry-over effects of ASE training i.e. decreased SBP and serum hsCRP concentration as well as increased serum HDL-cholesterol and K^+ concentrations.

Introduction

Prehypertension, an earlier stage in the continuum of hypertension,[1] affects ~25–50% of adults worldwide and increases the future risk of hypertension incidence.[2] NHANES II 1999–2000 data showed that 64% of individuals with prehypertension had at least one other cardiovascular risk factor; the percentage increased to 94% in those aged 60 years or more.[3] The Jichi Medical Cohort study reported that prehypertension was associated with a 45% higher risk of cardiovascular events as opposed to normotension after consideration of traditional cardiovascular risk factors.[4] Moreover, data also revealed that prehypertension was associated with an increased 5-year risk of hypertension and 10-year risk of cardiovascular disease (CVD).[2,3]

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The relative risk of hypertension incidence declines by ~20% with an intensive lifestyle intervention.[2] Exercise therapy is recommended in all the main clinical guidelines concerning CVD. It also produces beneficial effects amid risk factors for CVD, including hypertension, dyslipidemia, insulin resistance, obesity, and inflammation.[5] Arm swing exercise (ASE) is a traditional Chinese exercise. ASE is uncomplicated and easy to practice for older individuals because it is categorized as a low-intensity exercise based on ~23% of maximal oxygen consumption or ~45% of maximal heart rate.[6] Some studies have shown favorable effects of ASE training in subjects with type 2 diabetes as a result of reducing body fat percentage, glycated hemoglobin, low-density lipoprotein (LDL)-cholesterol, malondialdehyde and increasing reduced glutathione and oxidized glutathione.[7,8] The above parameters have been shown to be major influencing factors on blood pressure.[9–13] To date, data linked to effects of ASE training on blood pressure are limited, particularly amid prehypertension. The purpose of this study therefore, was to investigate the effect and carry-over effect of ASE training on blood pressure in older individuals with prehypertension. Importantly, cardiac autonomic modulation, cardiovascular risk factors, and blood electrolytes were together investigated in order to understand the underlying mechanisms causing alterations in blood pressure.

Methods

Study design and screening of subjects

Study design was a randomized controlled trial. Sedentary persons aged 60–80 years with prehypertension were announced to participate in the study with fifty subjects including accordingly (11 males and 39 females, aged 66.90 ± 5.50 yrs, body mass index; BMI 23.84 ± 3.65 kg/m^2). Subjects' systolic and diastolic blood pressure (SBP and DBP) ranged from 121 to 139 mmHg (mean 127.02 ± 5.39 mmHg) and 68–88.33 mmHg (mean 75.69 ± 5.88 mmHg) respectively. Prehypertension classification was defined by the Updated JNC-8 Guideline Recommendations.[14] This definition classifies prehypertension as resting SBP between 120 and 139 mmHg or a DBP between 80 and 89 mmHg. Subjects were invited to confirm their BP classification by measuring BP in the morning at the laboratory over 2 visits, 1 week apart. In addition to BP level, prehypertensive subjects were also classified as those without a history, diagnosis, or prior treatment of hypertension, and currently taking antihypertensive medication. A Heath Questionnaire Form was used to assess underlying diseases, prior drug treatments, and history of illness. Physical examinations including vital signs (BP and heart rate; HR) were also taken. Subjects who regularly smoked or consumed alcohol, had diabetes mellitus, thyroid disease, renal disease or cardiovascular disease i.e., arrhythmias were excluded from the study.

Power calculation

A statistical formula for comparing the mean read-outs of two groups of subjects was applied to calculate sample size. Leelayuwat et al.[7] reported that ASE training over the course of 8 weeks yielded mean HDL-cholesterol difference and SD at 2 ± 2.8 mg/dL. The decision was made to require 80% power with a significance level of 0.05. Accordingly, the proposed sample size of this study was 22 subjects per group with a total of 50 subjects, including a 10% drop-out rate.

Ethics statement

A consent form was obtained from all subjects before screening. Prior to being offered and signing the participation consent form, subjects were informed of their role in the study - both verbally and in writing. The consent form provided information regarding collection, use, and storage of subject data. Furthermore, the consent form and study protocols were in accordance with the ethical standards of the Human Ethics Committee of Burapha University (approval no. 61/2559 and 178/2559), as well as with the 1964 Helsinki declaration and its later amendments.

Recruitment of subjects

Fifty sedentary subjects aged 60–80 years with prehypertension were recruited from an Aging Society in Mueang District, Chonburi Province, Thailand from June to September 2016. Leaflets containing details of the study were sent to staff of the relevant institution. Subjects who were interested in participating in the study called the researchers and research assistant directly. Then, subjects were invited to the laboratory for screening. After screening, based on the inclusion and exclusion criteria and provision of informed consent, subjects were enrolled in the study.

Randomization and blinding

A random number table was generated using IBM SPSS Statistics 21 (IBM, Armonk, NY, USA). Subjects were numbered according to the sequence of enrollment. Eligible subjects with an odd-number were assigned into the ASE group (25 subjects), while those with an even-number were assigned to the control group (25 subjects). This study was an open trial; all subjects were aware of grouping and assessment.

Procedures

Fifty subjects were equally randomized into 2 groups: control group and ASE group. Subjects in the control group were asked to maintain their daily routine activities such as housekeeping, gardening, and cooking without any additional regular exercise i.e. arm swing exercise for 3 months. Subjects in the ASE group were requested to maintain their daily routine activities plus practicing ASE training for 3 months at a frequency of 30 min/day, 3 days/week. This intervention was designed as home-based exercise training. The subjects were trained on and practiced ASE correctly prior to performing training by themselves. In the process of carrying out the ASE training, subjects were instructed to stand with their feet shoulder width apart and have both arms, forearms, and hands relaxed in the pronation position. They were instructed to keep their trunk straight while relaxing the head and neck. The forefeet were pushed down to the floor allowing the muscles of the foot, leg, thigh, and abdomen to remain tight. Then, both arms were to be swung forward to around 30° and backward to around 60° - slowly and continuously. Moving the arms fully forward and backward was counted as 1 cycle, and subjects were instructed to swing at a rate of around 42 cycles/minute.[15] The subjects who could not complete the intervention (e.g., those exhibiting a health problem or requesting to stop) were asked to inform a researcher. If in the unlikely instance subjects were injured as a result of practicing ASE training, they would be compensated for care costs by the research team. Consequently, any adverse events associated with the study intervention would be recorded. Compliance to exercise training was determined via self-reporting. Subjects were asked to record their ASE training schedule in the record sheet provided by researchers during the training period. They were also closely followed weekly via telephone communication by researchers.

After the 3-month intervention period, subjects in the control

group were asked to carry on with their daily routine activities and food consumption behaviors. While subjects in the ASE group were asked to temporarily quit the ASE training program and restore their daily routine activities. This follow-up period lasted for 1 month, and subjects were also closely followed weekly via telephone communication by researchers in the same manner as during the intervention period with the goal of encouraging completion of the study. Cardiac autonomic function, cardiovascular risk factors, and blood electrolytes were evaluated prior to and post intervention. HR, BP, and body measurements were also conducted. These outcomes were also evaluated at 1 month post intervention.

Anthropometric measurements

Height and body mass measurements (Health O meter Pro Series, USA) were yielded which then enabled subject BMI to be determined. Body composition was measured in the standing position using a body fat monitor (Tanita UM076, Japan). Measurements of waist and hip circumferences using a standard measuring tape and their ratio were gained to assess fat distribution. Waist circumference was measured at the end of a normal expiration and at the mid-point between the bottom rib and the superior iliac spine. Hip circumference was measured on a horizontal plane at the line of femur heads.

Heart rate and blood pressure measurements

HR and BP measurements were gained after resting in the supine position for 15 min using a digital automatic BP monitor (Microlife BP 3AQ1, Switzerland). HR and BP were measured 3 times, 5 min apart.[16] The mean among the 3 readings was then reported as the HR and BP for each subject. Pulse pressure (PP), mean arterial pressure (MAP), and rate pressure product (RPP) were calculated from the mean HR, SBP, and DBP. All measurements were performed at the same time of the day and under noise-free environmental conditions.

Assessment of cardiac autonomic function

Cardiac autonomic function was assessed via HRV analysis following HR and BP measurements. HRV measurement was attained for 10 min from a Lead II electrocardiogram employing PowerLab 4/30 (ADInstruments, Australia). Analysis of HRV parameters incorporated time domain and frequency domain. Time domain consisted of the standard deviation of normal beat-to-beat (R-R) intervals (SDNN) and the root-mean-square of successive R-R (RMSSD). In the frequency domain, analysis included the values of total power (TP): very low, low, and high frequency powers (VLF: DC to 0.04 Hz, LF: 0.04–0.15 Hz, and HF: 0.15–0.4 Hz), and LF/HF ratio.

Assessment of cardiovascular risk factors and electrolytes

In the morning after 12-h overnight fasting, subjects' blood was drawn from an antecubital vein and collected in glucose and clot activator tubes. Blood samples of 2 ml in a glucose tube were analyzed for plasma glucose concentration. Blood samples of 4 ml in a clot activator tube were analyzed for concentrations of serum hsCRP, lipid profile (HDL-cholesterol, LDL-cholesterol, triglyceride; TG, and total cholesterol; TC), and electrolytes (K^+, Mg^{2+}, sodium; Na^+, chloride; Cl^-, calcium; Ca^{2+}, and phosphorus). Plasma glucose concentration and serum TC, in addition to TG concentrations were measured via enzymatic methods. Serum HDL-cholesterol concentration was measured via the accelerator selective detergent method which then enabled LDL-cholesterol concentration to be calculated. Serum Na^+, K^+, and Cl^- concentrations were measured utilising the indirect ion-selective electrode method. Serum Mg^{2+} concentration was measured by employing the calorimetric method. Serum Ca^{2+} concentration was measured via the arsenazo III dye method and serum phosphorus concentration was measured using the phosphomolybdate method. These parameters were assayed utilising standard automated laboratory method (Architect c8000, Abbott, USA). Serum hsCRP concentration was measured via the immunoturbidimetric method using standard automated laboratory method (Architect ci8200, Abbott, USA). Atherosclerogenic index (AI) as an indicator for risk of coronary heart disease and metabolic risks[17,18] was calculated by applying the values of serum TC and HDL-cholesterol concentrations from the following equation:[19]

AI = (TC - HDL-cholesterol) / HDL-cholesterol

Statistical analysis

All statistical analysis were performed with IBM SPSS Statistics 21 (IBM, Armonk, NY, USA). All data were expressed as mean ± SD. The Shapiro-Wilk test was applied to examine normal distribution of data. Two-way repeated measures analysis of variance (ANOVA) was used to examine the differences in each parameter and group before and after intervention. Independent *t*-test was used to examine the differences between groups before intervention. A value of $p < 0.05$ was considered as significant.

Results

Physical characteristics

Baseline data of physical characteristics of subjects are shown in Table 1. There were no significant differences between the control and ASE groups in terms of age, gender, height, body mass, BMI, body fat percentage, fat mass, fat-free mass, body water, visceral fat

Table 1
Baseline data of physical and physiological characteristics of subjects.

	Control group	ASE group	p value
Age (yrs)	67.32 ± 6.89	67.24 ± 5.29	0.96
Gender (M/F)	4/21	7/18	0.32
Height (m)	1.56 ± 0.06	1.56 ± 0.10	1.00
Body mass (kg)	58.46 ± 10.74	58.24 ± 8.96	0.94
BMI (kg/m^2)	23.87 ± 4.13	23.81 ± 3.19	0.96
Body fat (%)	31.76 ± 8.76	31.02 ± 7.74	0.75
Fat mass (kg)	19.10 ± 7.97	18.04 ± 5.22	0.58
Fat-free mass (%)	64.85 ± 8.71	65.27 ± 7.36	0.85
Fat-free mass (kg)	37.36 ± 5.69	38.04 ± 7.75	0.72
Body water (%)	49.94 ± 5.00	50.58 ± 4.17	0.63
Water mass (kg)	28.95 ± 4.80	29.49 ± 5.64	0.72
Visceral fat level	8.44 ± 4.01	9.32 ± 3.86	0.43
Waist circumference (cm)	82.72 ± 12.24	80.76 ± 9.01	0.52
Hip circumference (cm)	96.02 ± 10.53	94.92 ± 6.12	0.65
W/H ratio	0.86 ± 0.08	0.85 ± 0.06	0.55
HR (beats/min)	63.30 ± 7.91	66.68 ± 11.05	0.25
SBP (mmHg)	127.42 ± 6.57	124.82 ± 8.10	0.11
DBP (mmHg)	72.43 ± 7.36	72.47 ± 6.96	0.99
PP (mmHg)	47.75 ± 8.48	52.11 ± 7.65	0.08
MAP (mmHg)	88.35 ± 7.29	89.84 ± 6.37	0.47
RPP (mmHg/min)	7606.19 ± 1123.35	8298.19 ± 1474.67	0.08

Data are mean ± SD, n = 25 in each group.
ASE, arm swing exercise; BMI, body mass index; W/H, waist to hip circumference ratio; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MAP, mean arterial pressure; RPP, rate pressure product.

level, and waist and hip circumferences, nor their ratio prior to intervention, following intervention, and at the 1-month follow-up period.

Heart rate and blood pressure

There were no significant differences between the control and ASE groups in regards HR and BP parameters prior to intervention (Table 1).

Post intervention, HR and SBP were significantly decreased in the ASE group ($p < 0.05$). There were no changes in the control group (Table 2). The results showed a significantly lower SBP in the ASE group as compared with the control group ($p < 0.05$) (Fig. 1).

At the 1-month follow-up interval, decreased SBP was sustained in the ASE group when compared with before intervention ($p < 0.05$). There were no changes in the control group. The results also revealed a significantly lower SBP amid the ASE group compared with the control group ($p < 0.05$).

Cardiac autonomic function

There were no significant differences between the control and ASE groups in terms of HRV parameters prior to intervention (Table 3).

After intervention, RMSSD and HF power values had significantly increased in the ASE group ($p < 0.05$). There were no changes to speak of in the control group. The results showed significantly greater RMSSD and HF power values in the ASE group as compared with the control group ($p < 0.05$) (Figs. 2 and 3).

At the 1-month follow-up period, there were no significant differences between the control and ASE groups.

Cardiovascular risk factors

There were no significant differences between the control and ASE groups in the parameters of cardiovascular risk factors in advance of the intervention (Table 2).

Following intervention, serum HDL-cholesterol concentration had significantly increased and serum hsCRP concentration had significantly decreased in the ASE group ($p < 0.05$). There were no changes in the control group. The results showed a significantly higher serum HDL-cholesterol concentration and a significantly lower serum hsCRP concentration in the ASE group when compared with the control group ($p < 0.05$).

At 1-month follow-up interval, there were no changes in both the control and ASE groups. However, statistical analyses showed a significantly greater serum HDL-cholesterol concentration and a significantly lower serum hsCRP concentration in the ASE group as compared with the control group ($p < 0.05$) (Figs. 4 and 5).

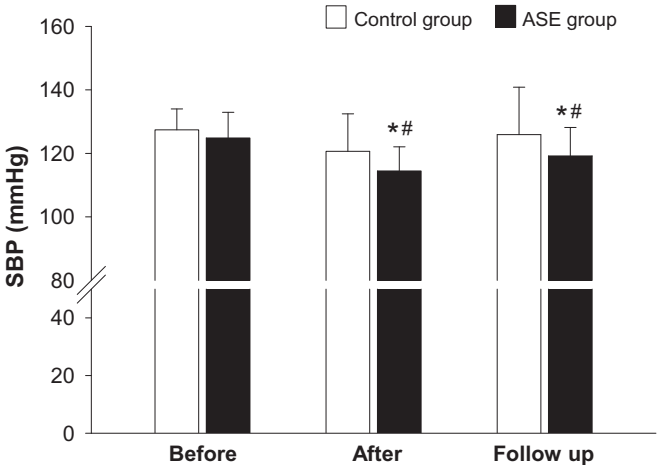

Fig. 1. Systolic blood pressure (SBP) in the control and arm swing exercise (ASE) groups before and after intervention, and at 1-month follow-up period. Values are expressed as mean ± SD, n = 25 per group. *Significantly different from prior to intervention ($p < 0.05$). #Significantly different from control group ($p < 0.05$).

Blood electrolytes

There were no significant differences between the control and

Table 2
Heart rate, blood pressure, cardiovascular risk factors, and electrolytes of subjects before and after the 3-month intervention, and 1-month follow-up period.

	Control group			ASE group		
	Before intervention	After intervention	Follow-up period	Before intervention	After intervention	Follow-up period
HR (beats/min)	63.30 ± 7.91	62.36 ± 7.36	61.85 ± 6.93	66.68 ± 11.05	62.36 ± 7.36*	63.25 ± 10.26
SBP (mmHg)	127.42 ± 6.57	120.62 ± 11.83	125.92 ± 14.91	124.82 ± 8.10	114.47 ± 7.57*#	119.20 ± 8.97*#
DBP (mmHg)	72.43 ± 7.36	71.72 ± 6.87	72.47 ± 8.00	72.47 ± 6.96	69.93 ± 5.93	71.15 ± 6.24
PP (mmHg)	47.75 ± 8.48	46.67 ± 9.86	48.32 ± 11.10	52.11 ± 7.65	45.81 ± 7.30	46.81 ± 7.89
MAP (mmHg)	88.35 ± 7.29	87.28 ± 8.40	88.58 ± 9.92	89.84 ± 6.37	85.20 ± 5.93	86.75 ± 6.30
RPP (mmHg/min)	7606.19 ± 1123.35	7353.72 ± 1016.27	7426.05 ± 960.91	8298.19 ± 1474.67	7422.05 ± 1270.03	7464.98 ± 1377.39
FBG (mg/dL)	88.87 ± 7.08	92.17 ± 10.81	91.67 ± 13.38	94.00 ± 13.07	91.63 ± 10.37	91.39 ± 12.42
TG (mg/dL)	129.87 ± 82.07	110.21 ± 51.02	128.71 ± 53.42	122.64 ± 46.56	123.29 ± 62.28	111.61 ± 35.48
LDL-cholesterol (mg/dL)	138.30 ± 43.01	146.50 ± 36.59	136.24 ± 32.77	137.36 ± 36.01	136.38 ± 37.97	134.96 ± 33.38
HDL-cholesterol (mg/dL)	49.52 ± 9.68	50.42 ± 9.08	48.86 ± 9.12	49.56 ± 12.69	56.16 ± 10.28*#	54.47 ± 9.71#
TC (mg/dL)	213.83 ± 47.42	219.04 ± 37.01	210.90 ± 36.89	211.56 ± 36.89	212.46 ± 40.54	206.87 ± 36.00
Atherosclerogenic index	3.47 ± 1.29	3.62 ± 1.31	3.45 ± 1.12	3.49 ± 1.17	3.40 ± 1.32	3.35 ± 1.11
hsCRP (mg/L)	1.84 ± 0.27	1.98 ± 0.31	2.07 ± 0.26	1.99 ± 0.45	1.51 ± 0.37*#	1.68 ± 0.49#
Na^+ (mmol/L)	141.87 ± 1.14	141.04 ± 1.63	141.71 ± 1.85	141.91 ± 1.08	141.00 ± 1.65*	141.39 ± 1.70
Cl^- (mmol/L)	108.22 ± 1.95	107.38 ± 1.93	107.71 ± 2.28	108.17 ± 1.99	107.35 ± 1.97*	107.83 ± 2.85
K^+ (mmol/L)	4.20 ± 0.32	4.13 ± 0.28	4.00 ± 0.28	4.29 ± 0.34	4.29 ± 0.30#	4.20 ± 0.37#
Ca^{2+} (mg/dL)	9.07 ± 0.58	9.11 ± 0.50	9.17 ± 0.73	9.05 ± 0.57	9.24 ± 0.51*	9.23 ± 0.34*
Mg^{2+} (mg/dL)	2.42 ± 0.12	2.33 ± 0.16	2.34 ± 0.21	2.44 ± 0.16	2.43 ± 0.18#	2.40 ± 0.15
Phosphorus (mg/dL)	3.70 ± 0.48	3.83 ± 0.43	3.95 ± 0.53	3.73 ± 0.41	3.82 ± 0.49	3.73 ± 0.47

Data are mean ± SD, n = 25 in each group.
ASE, arm swing exercise; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; MAP, mean arterial pressure; RPP, rate pressure product; FBG, fasting blood glucose; TG, triglyceride; LDL, low-density lipoprotein; HDL, high-density lipoprotein; TC, total cholesterol; hsCRP, high-sensitive C-reactive protein; Na^+, sodium; Cl^-, chloride; K^+, potassium; Ca^{2+}, calcium; Mg^{2+}, magnesium.
*Significantly different from prior to intervention ($p < 0.05$), #Significantly different from control group ($p < 0.05$).

Table 3
Heart rate variability of subjects before and after the 3-month intervention, and 1-month follow-up period.

	Control group			ASE group		
	Before intervention	After intervention	Follow-up period	Before intervention	After intervention	Follow-up period
HR (beats/min)	61.83 ± 6.36	62.90 ± 6.99	62.31 ± 6.54	64.97 ± 10.28	64.93 ± 10.52	63.63 ± 10.50
SDNN (ms)	43.97 ± 16.97	44.80 ± 23.31	42.80 ± 18.89	35.43 ± 14.45	43.15 ± 21.20	43.30 ± 23.40
RMSSD (ms)	34.47 ± 17.34	32.07 ± 18.59	37.77 ± 31.92	30.65 ± 17.92	48.17 ± 23.74*#	41.98 ± 26.12
TP (ms^2)	2015.18 ± 1689.41	2087.13 ± 1812.09	1810.10 ± 1219.95	1386.40 ± 1163.07	2350.92 ± 2335.04	2451.93 ± 2975.80
VLF (ms^2)	1069.89 ± 1336.07	1035.87 ± 757.77	961.99 ± 611.57	746.58 ± 644.04	970.17 ± 987.27	1392.72 ± 1587.86
LF (ms^2)	317.53 ± 272.28	329.80 ± 321.81	263.41 ± 166.26	231.90 ± 240.88	342.16 ± 521.92	388.67 ± 608.60
LF (nu.)	38.06 ± 17.59	42.61 ± 19.43	40.77 ± 19.34	44.47 ± 21.86	37.43 ± 21.21	43.21 ± 17.68
HF (ms^2)	500.40 ± 536.89	564.97 ± 807.59	413.45 ± 499.38	351.14 ± 537.26	673.36 ± 823.65	536.09 ± 940.55
HF (nu.)	51.50 ± 14.29	48.91 ± 16.35	48.37 ± 17.70	47.54 ± 17.10	59.58 ± 15.16*#	52.94 ± 11.51
LF/HF ratio	0.95 ± 0.93	1.20 ± 1.16	1.21 ± 1.35	1.57 ± 1.70	1.05 ± 0.98	1.34 ± 1.68

Data are mean ± SD, n = 25 in each group.
ASE, arm swing exercise; HR, heart rate; SDNN, the standard deviation of normal beat-to-beat (R-R) intervals; RMSSD, the root-mean-square of successive R-R; TP, total power; VLF, very low frequency power; LF, low frequency power; HF, high frequency power.
*Significantly different from prior to intervention ($p < 0.05$), #Significantly different from control group ($p < 0.05$).

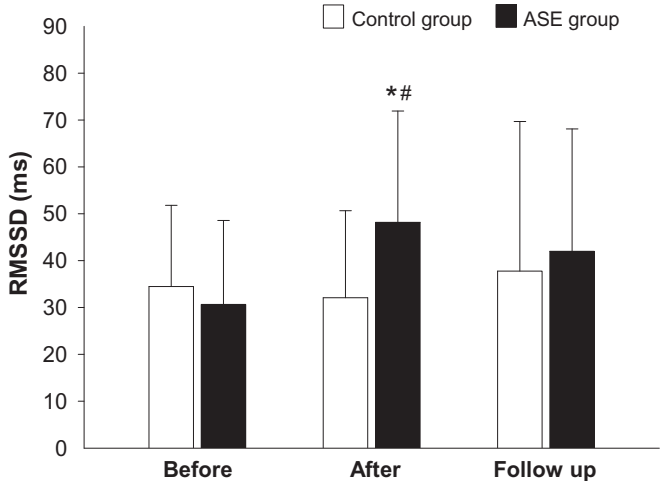

Fig. 2. The root-mean-square of successive R-R (RMSSD) values in the control group and arm swing exercise group (ASE) before and after intervention, and at 1-month follow-up interval. Values are expressed as mean ± SD, n = 25 per group. *Significantly different from before intervention ($p < 0.05$). #Significantly different from control group ($p < 0.05$).

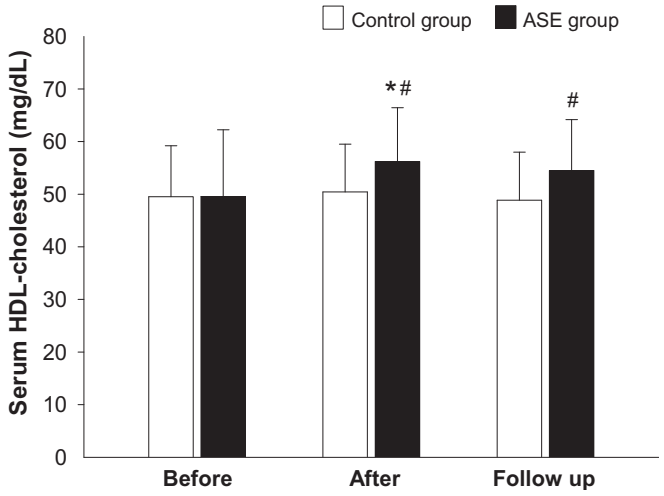

Fig. 4. Serum high-density lipoprotein (HDL)-cholesterol concentration in the control group and arm swing exercise group (ASE) before and after intervention, and at 1-month follow-up period. Values are expressed as mean ± SD, n = 25 per group. *Significantly dissimilar to before intervention ($p < 0.05$). #Significantly different from control group ($p < 0.05$).

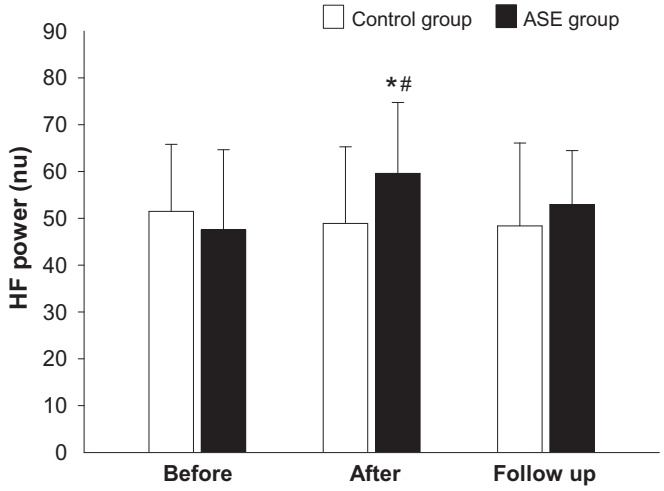

Fig. 3. High frequency (HF) power in the control group and arm swing exercise (ASE) group prior to intervention, post intervention, and at 1-month follow-up juncture. Values are expressed as mean ± SD, n = 25 per group. *Significantly different from before intervention ($p < 0.05$). #Significantly different from control group ($p < 0.05$).

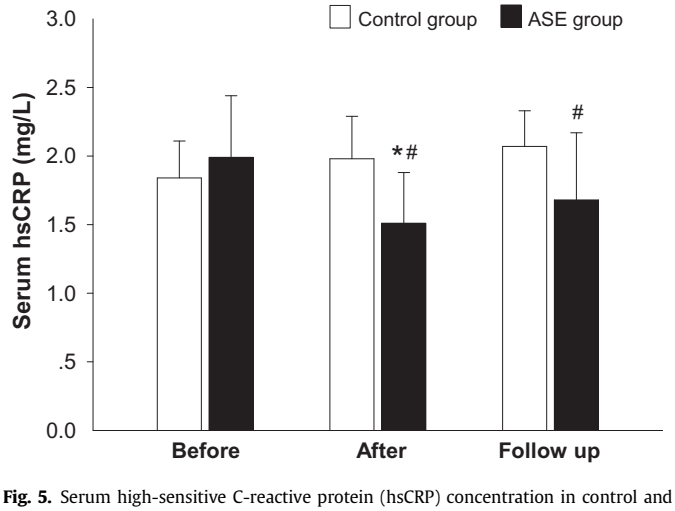

Fig. 5. Serum high-sensitive C-reactive protein (hsCRP) concentration in control and arm swing exercise (ASE) groups before as well as after intervention, and at 1-month follow-up juncture. Values are expressed as mean ± SD, n = 25 per group. *Significantly dissimilar to before intervention ($p < 0.05$). #Significantly different from control group ($p < 0.05$).

ASE groups in terms of serum electrolyte concentrations prior to intervention (Table 2).

After intervention, serum Ca^{2+} concentration had significantly increased, and serum Na^{+} and Cl^{-} concentrations had significantly decreased in the ASE group ($p < 0.05$). There were no changes in the control group. The results displayed significantly greater serum K^{+} and Mg^{2+} concentrations in the ASE group when compared with the control group ($p < 0.05$).

At the 1-month follow-up period, serum Ca^{2+} concentration had significantly increased in the ASE group when compared with before intervention ($p < 0.05$). There were no variations in the control group. The results revealed a significantly greater serum K^{+} concentration in the ASE group as compared with the control group ($p < 0.05$) (Figs. 6 and 7).

Discussion

The present study investigated the effect and carry-over effect of ASE training at an average intensity of 55–60% of maximal HR for 30 min a day, 3 days a week, throughout 3 months. Our findings show that ASE training for 3 months decreased resting SBP and DBP by 10.35 and 2.94 mmHg (accounting for −8.29% and −4.07%, respectively) compared with the non-exercising control group. Decreased BP was further observed at the 1-month follow-up juncture of 5.62 and 1.48 mmHg for SBP and DBP (−4.50% and −2.05%). Our results are consistent with previous literature: for instance, a study conducted in elderly individuals with essential hypertension revealed that regular low-intensity exercise training for 9 months significantly reduced SBP and DBP by 7 and 9 mmHg respectively as compared with a control group not exercising.[20] In prehypertensive and stage I hypertensive patients exhibiting an average age of 55 years, regular walking for 24 weeks significantly reduced SBP by 11 mmHg (8%) and MAP by 5 mmHg (5%).[21] Moreover, in normal healthy subjects aerobic exercise training for 4 weeks decreased resting SBP and DBP by 10 and 6 mmHg; such a reduction can persist for a period of 7 days, thus incorporating a reduced total peripheral resistance index of 14%, and reduced plasma noradrenaline of 21%. In that study, during detraining, BP remained low for 1–2 weeks after cessation of exercise.[22] It has been estimated that a 2-mmHg reduction of SBP results in a 6% reduction in stroke mortality and a 4% reduction in mortality attributable to coronary heart disease; such percentage reductions

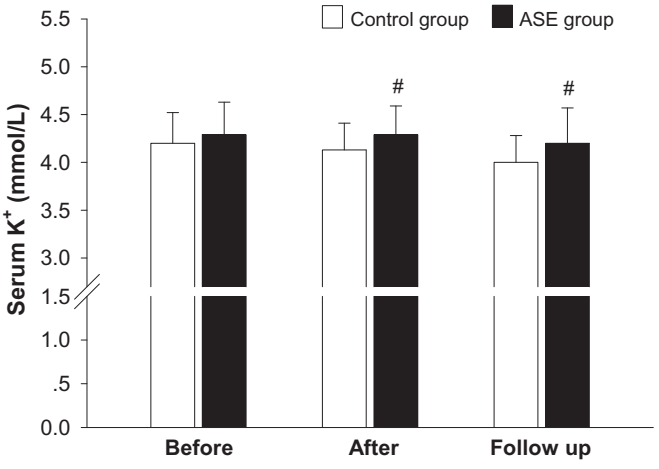

Fig. 6. Serum potassium (K^{+}) concentration in the control group and arm swing exercise (ASE) group before and after intervention, and at 1-month follow-up point. Values are expressed as mean ± SD, n = 25 per group. #Significantly different from control group ($p < 0.05$).

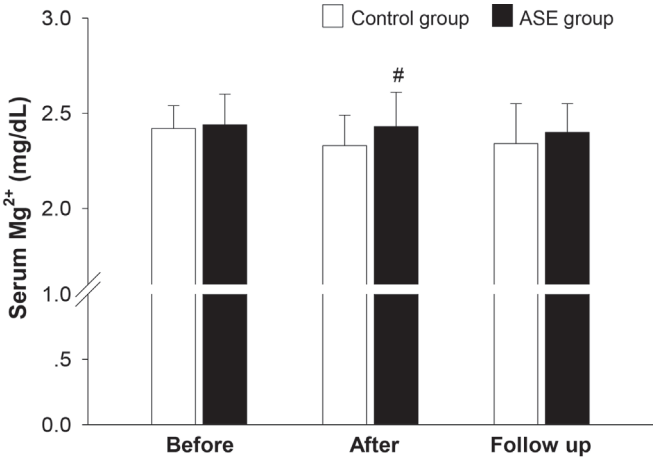

Fig. 7. Serum magnesium (Mg^{2+}) concentration in the control and arm swing exercise groups (ASE) before and after intervention, and at 1-month follow-up point. Values are expressed as mean ± SD, n = 25 per group. #Significantly different from control group ($p < 0.05$).

amount to 14% and 9%, respectively for a 5-mmHg decrease of BP.[23]

A 6% increase in cardiac index and decrease in vascular resistance at rest were reported in subjects given exercise training.[24] Meta-analysis revealed a significant reduction of systemic vascular resistance without change in cardiac output. Data also exhibited that the decrease of HR was counter-balanced by an increase in stroke volume with unchanged cardiac output reflecting the beneficial effect of aerobic exercise training on resting hemodynamics.[23] The above data suggest that exercise training is an important contributor to chronic adaptations of cardiovascular function, which may prevent or delay the development of hypertension in prehypertensive individuals.[25]

Cardiac autonomic function has been evidenced to be one influential factor regulating BP.[26] The present study showed that RMSSD and HF power values were significantly increased after ASE training compared to control subjects. The increases in RMSSD and HF power values reflect the increase of parasympathetic nervous system activity, which has been reported to be a potent factor in the reduction of BP post-exercise training.[27,28] Our data did not indicate a significant alteration in the parameters reflecting a reduction of sympathetic nervous system activity, such as decreased values of LF power or ratio of LF/HF.[29,30] Nevertheless, a decrease in sympathetic nervous system activity was reported to be involved in the training-induced reduction of BP as evidenced by lower plasma norepinephrine and renin levels when compared with untrained subjects.[23] This data suggests that the lessening in sympathetic nervous system activity also affects the kidneys, which is a powerful factor in long-term regulation of BP.[23]

The reduction of cardiovascular risk factors may also contribute to the favorable effect on BP. Decreased LDL-cholesterol and TG, and increased HDL-cholesterol are potentially important mechanisms.[31] Improvements to insulin sensitivity and endothelial function are also concerted in this circumstance.[23] Our results showed that serum HDL-cholesterol level significantly increased, whereas serum hsCRP level significantly decreased after the 3-month ASE training program. The ASE training also exerted carry-over effects on increased serum HDL-cholesterol and decreased serum hsCRP levels at the 1-month follow-up point. HDL-cholesterol stimulates structural changes under pathological conditions such as oxidative stress, inflammation and diabetes, (which may exert antiatherogenic, anti-inflammatory, antiapoptotic, and antithrombotic effects in the endothelial cells). Hence, increased serum HDL-cholesterol is related to a decreased risk concerning

both coronary artery disease and myocardial infarction.[32] HsCRP, a biomarker of inflammation, is recognized as a major CVD risk factor.[33] Reduced serum hsCRP level contributes to reduced vascular inflammation, atherosclerosis, and CVD risk.[34–36] Our data indicates the cardiovascular benefits of ASE training; and these benefits are more apparent when systemic inflammation is reduced in addition to increased serum HDL-cholesterol level. What's more, our results are compatible with several other findings which reveal improvements concerning CVD risk factors in elderly subjects post exercise training.[32,37–39]

A number of studies suggest that there is close association between electrolyte and BP levels.[40,41] The present study revealed that serum Na^+, K^+, Ca^{2+}, and phosphorus levels among subjects were in the clinically normal range, while serum Cl^- and Mg^{2+} levels were slightly higher than the normal range. Moreover, there is substantial evidence regarding the adverse effects of high Na^+ intakes on BP and cardiovascular health.[42] Several studies have shown that intracellular and plasma or serum Na^+ and Cl^- concentration are significantly higher in hypertensives than normotensives; and these ions act cooperatively to induce BP rise.[43] Whereas, K^+ is an important predictor of MAP.[43] Hypertensives have been reported to present lower plasma or serum and total body K^+ as well as lower urinary K^+ excretion levels than normotensives.[41,43] Considerable evidence has shown that K^+ deficiency plays a key role in hypertension and its sequelae. The possible mechanism by which reductions in serum K^+ affect BP is that they lead to reduced urinary excretion of Na^+, retention of Na^+ by the body and an increase in the volume of extracellular fluid.[41] Mg^{2+} is also involved in regulation of BP.[44] That is to say, it may play a role in BP regulation through directly stimulating prostacyclin and nitric oxide formation, modulating endothelium-dependent and endothelium-independent vasodilation, reducing vascular tone and reactivity, and finally, preventing vascular injury via its antioxidant and anti-inflammatory functions.[45] The supplementation of Mg^{2+} decreases arterial BP and attenuates the development of hypertension-induced complications.[46] We observed that serum K^+ and Mg^{2+} levels were significantly higher in our subjects given ASE training compared to non-exercise control subjects. Furthermore, in the ASE training group a higher serum K^+ level remained at the 1-month follow-up juncture. Although prior evidence has indicated that low circulating Mg^{2+} may be related to the development of hypertension due to its Ca^{2+} antagonist and endothelial effects, the present study found that Mg^{2+} levels were slightly higher than the normal range of 1.5–2.3 mg/dL. A higher Mg^{2+} level may be partially due to a mechanism of preservation of Mg^{2+} in the balance of both Na:K and Mg:Ca ratios at both the cellular and whole body levels in addition to a regulation of intracellular Ca^{2+} ion concentration producing healthy BP.[44,47] One of the mechanisms by which Mg^{2+} lowers BP, is by acting like a natural Ca^{2+} channel blocker. Mg^{2+} competes with Na^+ for binding sites on vascular smooth muscle cells, increases prostaglandin E, binds to K^+ in a cooperative manner, induces endothelial-dependent vasodilation and improves endothelial dysfunction.[48] High levels of extracellular Mg^{2+} were associated with the improvements in hemodynamic status, such as blood flow, vascular resistance, and capacitance function of vessels.[45]

Our findings support the broad recommendation that physical exercise - even low-intensity exercise - is imperative in yielding a plethora of health benefits, not only for the prevention of cardiovascular disease but also in the management of hypertension.

Conclusions

Our study results suggest that ASE training for 3 months at a frequency of 30 min/day, 3 days/week decreased SBP and serum hsCRP concentration, increased serum HDL-cholesterol, K^+, and Mg^{2+} concentrations and increased RMSSD and HF power values in elderly persons with prehypertension. In addition, there were carry-over effects of ASE training on decreased SBP and serum hsCRP concentration as well as increased serum HDL-cholesterol and K^+ concentrations amid the follow-up period.

Abbreviations

AI, Atherosclerogenic index; ANOVA, Analysis of variance; ASE, Arm swing exercise; BP, Blood pressure; Ca^{2+}, Calcium; Cl^-, Chloride; CVD, Cardiovascular disease; DBP, Diastolic blood pressure; HDL, High-density lipoprotein; HF, High frequency; HR, Heart rate; HRV, Heart rate variability; hsCRP, High-sensitive C-reactive protein; K^+, Potassium; LDL, Low-density lipoprotein; LF, Low frequency; MAP, Mean arterial pressure; Mg^{2+}, Magnesium; Na^+, Sodium; PP, Pulse pressure; RMSSD, Root-mean-square of successive R-R; RPP, Rate-pressure product; SBP, Systolic blood pressure; SD, Standard deviation; SDNN, Standard deviation of normal R-R intervals; TC, Total cholesterol; TG, Triglyceride; TP, Total power; VLF, Very low frequency.

Conflicts of interest

No conflict of interest was reported by the authors of this paper.

CRediT authorship contribution statement

Piyapong Prasertsri: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Writing - original draft, Writing - review & editing. **Sanita Singsanan:** Formal analysis, Funding acquisition, Investigation, Writing - original draft. **Chirapond Chonanant:** Funding acquisition, Investigation, Writing - original draft. **Orachorn Boonla:** Investigation, Writing - original draft. **Petcharat Trongtosak:** Funding acquisition, Investigation, Supervision, Writing - original draft.

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