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The effect of a weight loss program on mental stress-induced cardiovascular responses and recovery

**Abbreviated title:** Effect of weight loss on stress response

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Susan Torres (ST) and Caryl Nowson (CN) planned the research and ST conducted the experiments. ST completed the statistical data analysis and wrote the draft manuscript. ST and CN contributed to the data interpretation and final approval of the manuscript. None of the authors had any conflicts of interest.
ABSTRACT

Objective: To assess the effect of weight loss on BP and pulse rate during rest, psychological stress and recovery after stress.

Methods: In a prospective parallel design, 33 men underwent weight loss (intervention group) (on 2 different diets: high fruit, vegetables and dairy DASH (WELL) or low fat (LF)), and 31 men maintained weight (controls). Both groups completed a mental stress test (13 min resting, 7 min stress, 36 min recovery) at baseline and 12-wk.

Results: Fifty-five men with a baseline BP of 125.9 ± 6.9/83.6 ± 7.1 mmHg (mean ± SD) completed the study [intervention (n=28), controls (n=27)]. The intervention group compared to the controls lost weight (-4.3 ± 0.3 kg versus +0.4 ± 0.4 kg, \(P = 0.001\)) (mean ± SEM) and had a greater fall in resting systolic BP (SBP) (-2.0 ± 1.1 % versus +2.0 ± 1.1 %, \(P < 0.05\)). Using repeated measures anova, there was a significant decrease in SBP (\(P < 0.05\)) and pulse rate (\(P < 0.05\)) at all time points during the stress test between the groups. After treatment, the intervention group returned to resting SBP levels in less time than the controls (15.2 ± 1.8 versus 20.9 ± 1.9 min, \(P < 0.05\)). In the WELL (n=14) versus LF (n=14) groups, there was an overall greater fall in DBP (\(P < 0.05\)) and DBP during recovery, up to 27 min post stress (\(P < 0.05\)).

Conclusion: A 5% loss of weight resulted in a reduction in BP during rest and assisted in returning SBP to resting levels faster, which is likely to reduce the risk of cardiovascular disease.

Key words: blood pressure, hypertension, stress, men, obesity, diet
INTRODUCTION

Hypertension is an important public health issue and well established risk factor for coronary heart disease [1]. Psychological stress is one of many mental health disorders that contributes to the global burden of disease [2]. Exposure to acute psychological stressors can activate the sympathetic nervous system (SNS), followed by pronounced cardiovascular responses including increases in blood pressure (BP)\(^1\) and pulse rate [3]. There is evidence that unfavorable cardiovascular responses during stress and cardiovascular recovery after stress are risk factors for the development of hypertension. Prospective studies have demonstrated that individuals with heightened cardiovascular reactivity (defined as the difference between stress and resting values) during psychological stress are more likely to have higher resting BP levels at follow-up [4-8], although one study has not supported this finding [9]. Higher absolute levels of BP during psychological stress have also been associated with higher resting BP levels at follow-up [4]. Delayed cardiovascular recovery from stress may also be related to increased risk for future hypertension [10-12].

Obesity is a global epidemic and is increasing at an alarming rate [13]. There is a strong positive association between body weight and resting BP, and prospective studies have shown that obesity is a risk factor for hypertension. BMI has also been positively associated with impaired post-stress recovery of BP [14]. Weight loss programs have favorable results on BP and pulse rate, with reductions reported for resting [15-17], and absolute levels during psychological stress [16, 17], although the effect on cardiovascular reactivity during stress and recovery after stress is not well understood. Furthermore, a comprehensive dietary approach (DASH: Dietary Approaches to Stop Hypertension), which included a diet high in

\(^1\) BP indicating both systolic blood pressure (SBP) and diastolic blood pressure (DBP)
fruit, vegetables and low-fat dairy foods, demonstrated large falls in resting BP [18]. The aim of this study was to determine the effect of a weight loss intervention, induced by dietary deficiency and increased modest physical activity (walking), on cardiovascular indices (BP and pulse rate) during rest, acute psychological stress and recovery after stress. An additional aim was to compare the effect on BP and pulse rate a DASH-type weight-loss diet (WELL) with a usual low-fat weight-loss diet (LF). This was part of a larger investigation examining the effects of a lifestyle intervention on BP in free-living overweight and obese individuals [15].

SUBJECT AND METHODS

Subjects

Subjects for the stress study weight loss intervention group 2 were recruited from a larger study investigating the effect of two dietary interventions on home BP- the WELL and LF diet, which were both combined with increased physical activity to achieve weight loss. Details of subject recruitment for the main dietary intervention are reported elsewhere [15]. Advertisements in newspapers and workplaces, and mail outs were used to recruit subjects for the stress study control group.

Subjects were eligible for the stress study if they were male, had a BMI between 24 and 36 kg/m² and a seated office SBP ≥118 mmHg or an office DBP ≥78 mmHg. A trained researcher obtained screening office BP measurements using a Vital Care 506DX automated BP monitor (Criticare Systems Inc., US) or the TM 2551 portable bedside monitor (A&D Co. Ltd, Tokyo, Japan). Seated office BP was measured 4 times at 2 min intervals after an initial

2 “weight loss intervention group” will be referred to as “intervention group”
rest period of 5 min, and the mean office BP was calculated from the last 3 measurements. Subjects who were taking antihypertensive medication were included, provided they were willing to maintain their current medication type and dosage. Subjects were excluded if they had a cardiovascular event in the past 6 months, had insulin dependent diabetes or a stress related disorder, were on medications such as warfarin, dilantin or antidepressants, were planning to change smoking habits, and drank more than 30 standard alcoholic drinks (10g alcohol) per wk.

All subjects provided written informed consent prior to starting the study, which was approved by the Deakin University Human Research Ethics Committee.

Study design

Intervention group

After the baseline stress test, subjects participating in the main dietary study were randomized to either the WELL or LF diet for 12 wk. The WELL diet had specific daily targets including ≥4 serves of fruit, ≥4 serves of vegetables and ≥3 of low fat dairy products. The LF diet had general guidelines on increasing fruit and vegetable intake, and reducing total fat intake. Both diet groups were instructed to participate in moderate intensity exercise (60–79% of maximum heart rate), which consisted of walking, for at least 30 min on all or most days of the week. The diet and exercise program have both been described in further detail [15]. The stress test was completed at the end of the study.

Control group
Subjects completed a baseline stress test and were instructed to maintain their usual lifestyle practices with no changes in their diet and physical activity levels for 12 wk. The stress test was completed at the end of the study.

**Anthropometry Measurements**

Weight was recorded at baseline and 12-wk in kilograms to the nearest 0.1 kg with the UC-300 digital scales (A&D Co. Ltd, Tokyo, Japan) on a firm surface. Height was measured at baseline to the nearest millimetre using a free standing stadiometer. Subjects wore similar clothing without shoes for each measurement. BMI was calculated as weight (kg) divided by height² (m²).

**Stress test**

The mental arithmetic stress test protocol for the present study was adapted from Earle et al [19]. Mental stress testing has been extensively used in cardiovascular stress research [5, 6, 19-21]. In the 12 hours preceding the stress test, subjects were instructed to avoid alcohol, caffeine, smoking and vigorous exercise. All stress tests were conducted by the same researcher starting at 0800 h and concluding by 1230 h. Weight was measured and then subjects rested in the sitting position for 5 min. The TM-2421 automated ambulatory BP monitor (A&D Co. Ltd, Tokyo, Japan), attached to the subject's left arm, measured BP and pulse rate every 3 min commencing with a 13 min resting period. At minute 14, subjects commenced a standardised arithmetic stress test, which consisted of serial subtractions of a prime number from a high number given aloud. Subjects were informed they had to start from the beginning if they made a mistake. In addition, subjects had to complete a set number of subtractions per minute, and if they met this target, then the number of subtractions to aim for increased incrementally every minute. The arithmetic stress test
continued for 7 min, concluding at minute 21. BP and pulse rate readings continued until minute 57. The stress test was divided into 3 parts: resting (minute 0 to 13, mean of 5 BP and pulse rate readings); stress (minute 14 to 21, mean of 3 BP and pulse rate readings) and recovery (minute 22 to 57, divided into 4 phases with each phase being the mean of 3 BP and pulse rate readings). Time to recovery was calculated as the time taken to return to mean resting SBP, DBP and pulse rate levels from the conclusion of stress.

**Statistical analysis**

Data were analyzed using SPSS for WINDOWS (version 11.5.2.1; SPSS Inc, Chicago). All data are reported as means ± SEMs unless otherwise indicated. Paired Student’s t tests were used to determine the difference within groups. Differences between groups were investigated using Student’s t test for independent samples. Two-factor (group x time) repeated measures ANOVA, followed by linear contrasts, were used to test between group differences in the percentage change in BP and pulse rate at every time point during the stress test. The interaction between “group” and “time” was also tested. \( P < 0.05 \) was considered to be statistically significant.
RESULTS

Subject Characteristics

Intervention group

From the 63 subjects enrolled in the main dietary study, 59 were not taking anti-depressants and were invited to participate in the intervention group of the stress study. Thirty-three completed the baseline stress test and 29 completed the post-treatment stress test. Four subjects dropped out of the stress study (3 withdrew from the main dietary study and 1 subject refused). Due to technical difficulties with the BP monitor during the stress test, data from 1 subject was excluded from the study. Twenty-eight subjects completed baseline and post-treatment stress tests and were included in the final analysis. Of these 28 subjects, 14 followed the WELL diet and 14 followed the LF diet.

Control group

Forty-three attended a screening appointment and of these, 31 were eligible for the stress study and completed the baseline stress test. Twenty-eight of these subjects completed the post-treatment stress test. Three subjects dropped out of the study (1 commenced anti depressants, 1 moved interstate and 1 gave no reason). Data from 1 subject were excluded, as there was a change in antihypertensive medication. Twenty-seven subjects completed baseline and post-treatment stress tests and were included in the final analysis.

Of the 55 subjects who completed the stress study, 21 were taking anti-hypertensive medication (12 intervention group, 9 control group). In the intervention group there were 6 subjects on single BP therapy [angiotensin-converting enzyme inhibitor (ACE), angiotensin II receptor antagonists (AT₁), calcium channel blocker], and 6 on combination therapy. In
the control group there were 7 subjects on single therapy [(ACE), (AT₁), calcium channel blocker], and 2 subjects on combination therapy. Within the intervention and control groups, there were no significant differences in the change in BP and pulse rate (baseline versus post-treatment) between subjects taking antihypertensive medication and those not taking antihypertensive medication, therefore data within each group were pooled (results not shown).

Table 1 shows the screening office BP’s and baseline age, weight, BP and pulse rate stress test data. At screening, the mean office DBP was significantly lower in the control group as compared to the intervention group ($P < 0.001$). However, at baseline, the intervention and control groups did not differ significantly from each other in age, BMI, resting BP and resting pulse rate levels. The mean time between the baseline and post-treatment stress tests was $11.3 \pm 0.2$ wk for the intervention group and $10.3 \pm 0.2$ wk for the control group ($P < 0.05$).

**Effects of treatment on body weight- between group analysis**

Compared with subjects in the control group (+0.4 ± 0.4 kg), subjects in the intervention group lost significantly more weight (-4.3 ± 0.3 kg; $P < 0.001$).

**Effect of treatment on cardiovascular indices during rest and stress- within group analyses**

The level of SBP and DBP during the mental stress task was significantly lower after weight loss in the intervention group [(baseline versus post-treatment) SBP: 142.0 ± 2.6 mmHg versus 136.3 ± 2.6 mmHg, $P = 0.008$; DBP: 93.9 ± 1.5 mmHg versus 90.2 ± 1.5 mmHg, $P = 0.018$]. There was an indication of a 2 bpm fall in resting pulse rate with weight loss.
[(baseline versus post-treatment): 69.6 ± 2.3 versus 67.5 ± 2.4 bpm, \( P = 0.082 \)]. A significant difference in baseline and post-treatment measurements was not seen in the control group.

**Effect of treatment on cardiovascular indices during the stress test- between group analyses**

Figure 1 shows SBP, DBP and pulse rate at every time point during the stress test at baseline and post-treatment. Resting SBP was significantly lower (\( P < 0.05 \)) in the intervention group as compared to the control group after treatment, and there was an indication of a lower resting DBP (\( P = 0.055 \)). Using repeated measures anova, there was a significant decrease in SBP (\( P < 0.05 \)) and pulse rate (\( P < 0.05 \)) at all time points during the stress test in the intervention group as compared to the control group, with no difference between the groups with respect to DBP (\( P = 0.245 \)). None of the group by time interactions were significant (SBP: \( P = 0.853 \); Pulse rate: \( P = 0.777 \)) indicating that SBP and pulse rate reactivity was not altered by weight loss. However, following weight loss in the intervention group, SBP was significantly lower in the first 24 min post stress (recovery phases 1-3), compared to the control group (\( P < 0.05 \)) (Figure 1). In each of the 4 recovery phases (36 min post stress), DBP tended to be lower, although not significantly different from the control group (Figure 1).

Table 2 shows the percentage change in BP and pulse rate at each time point during the stress test after 12 wk. There was a greater fall in resting SBP in the intervention compared to the control group, but no between group differences for resting DBP and pulse rate, absolute BP and pulse rate levels during stress, and BP and pulse rate reactivity (difference between stress and resting levels). The intervention group compared to the control group had a significantly
greater fall in SBP during recovery phase 2 (9-18 min post stress) and a greater fall in pulse rate during recovery phases 2-4 (9-36 min post stress).

At baseline, 22 (78.6%) subjects in the intervention group and 17 (63.0%) subjects in the control group recovered to resting SBP levels with a mean time of 20.5 ± 2.1 min and 20.5 ± 2.3 min, respectively, with no difference between the groups (P = 0.989). Post-treatment, both groups had a similar number of subjects who recovered, 23 (82.1%) subjects in the intervention group and 23 (85.2%) subjects in the control group; however, the mean recovery time for the intervention group was significantly less than the control group (15.2 ± 1.8 min versus 20.9 ± 1.9 min, (P < 0.05).

**Effect of treatment on body weight, energy intake, physical activity and cardiovascular indices during the stress test- between group analyses (diet groups versus control)**

Following treatment, there were no significant differences between the WELL and LF diet groups in percentage weight change (-5.3 ± 0.8 % versus -3.9 ± 0.5 %, P = 0.151), change in daily total energy intake (-3.0 ± 1.5 Mj/day versus -4.0 ± 0.9 Mj/day, P = 0.614) and change in daily walking (1.9 ± 1.1 hours/day versus 2.5 ± 1.2 hours/day, P = 0.717). Using repeated measures anova, there was a significant between group effect for the overall percentage change in SBP, DBP and pulse, (P < 0.05 for all). In the WELL versus LF group, there were no significant differences in the overall change in SBP (P = 0.085) and pulse rate (P = 0.794), but there was a greater fall in DBP (P < 0.05). Figure 2 shows the percentage change in BP and pulse rate at every time point during the stress test for the two diet groups (WELL and LF) and control group following treatment. The WELL group compared with the LF group, had a significantly greater fall in DBP during recovery phases 1-3 (27 min post stress), which equated to a mean difference between the groups of 5.5-7.5 mmHg.
DISCUSSION

The present study investigated in overweight and obese men the effect of a weight loss intervention, induced by dietary restriction and increased modest physical activity (walking), on BP and pulse rate during rest, acute psychological stress and recovery after stress. Lifestyle modifications incorporating weight loss are known to reduce resting BP [15], and we found that a 4.3 kg loss of weight (4.6%) was sufficient to significantly reduce resting SBP by 3 mmHg. The reduction of 3 mmHg absolute resting SBP corresponds to the expected SBP reduction with weight loss in the range of 4% to 8% of body weight [22]. We found that our weight loss intervention had no effect on absolute BP and pulse rate levels during stress or BP and pulse rate reactivity (difference between stress and resting levels). We did find that the weight loss intervention facilitated faster recovery of SBP back to resting levels.

The absolute level of BP during the stress task has been positively associated with the development of hypertension, although this study did not adjust for resting BP levels [4]. In 44 men who lost on average 7.8 kg, there was a reduction in absolute BP (11/7 mmHg) and pulse rate (11 bpm) levels during acute mental stress [16]. With a weight loss of 4.3 kg in the present study, we found a smaller but significant reduction in absolute stress levels of DBP (4 mmHg) within the intervention group.

Evidence from several studies have indicated that heightened cardiovascular responses to psychological stress are associated with future increases in BP [9, 23, 24]. We found that BP and pulse rate reactivity was not altered by our intervention program. There is some evidence to suggest that cardiovascular reactivity may be under genetic control. In a study with twins,
heritability estimates were significant for BP reactivity but not pulse rate reactivity during mental stress [25]. Tonacio et al. [26] assessed the impact of body weight reduction on muscle sympathetic nerve activity during mental stress and found that weight loss resulted in a significant reduction in the absolute levels but no alteration in the magnitude. Our results suggest the weight loss intervention shifts the whole stress response curve downward, rather than having a specific effect on BP and pulse rate reactivity. When compared to the control group, the intervention group tracked lower at every time point during the one hour procedure after treatment, with a significant decrease in resting SBP, and the level of DBP achieved during the mental stress test was lower after weight loss in the intervention group.

We did find the weight loss intervention facilitated a faster recovery of SBP to resting levels after the conclusion of a stressful event, as SBP levels were significantly lower in the intervention group compared to the control group, in the first half of the recovery phase (9-18 min post stress). DBP and pulse rate followed the same trend during recovery; however this was only significant for pulse rate, which was 5% lower in the weight loss group at the end of recovery. Delayed BP recovery from stress has been associated with higher BP levels after 3 y [5, 11, 12] and 5 y of follow-up [12], and we found that after treatment, SBP reached resting levels 5 min earlier.

As part of the weight loss program, subjects in the intervention group were advised to decrease their energy intake and increase their physical activity levels, which consisted of walking at a moderate intensity for 30 minutes on all or most days of the week. There is the possibility that some of the observed improvements in BP and pulse rate during rest, mental stress and recovery after stress may be due to the effect of walking in the weight loss group. Tonacio et al [26] assessed the BP and pulse rate responses to stress of an energy deficient
diet alone versus energy deficient diet combined with aerobic exercise, in two groups matched for weight loss (~8-9 kg). They found a greater reduction in absolute stress levels of SBP, but not DBP or pulse rate with the combined approach. In a meta-analysis that examined the effect of walking on resting BP, decreases of 3/2 mmHg were observed after an average of 25 weeks of treatment with no change in body weight and a fall in resting pulse rate of 6 bpm [27]. In the present study, it is possible that walking contributed to the observed fall in resting SBP of 3 mmHg, although we found no significant fall in resting pulse rate, and the intensity of walking was less than that of the supervised aerobic exercise reported by Tonacio et al [26].

Psychosocial stressors (e.g. mental stress) are known to activate the ‘fight or flight response” with the sympathetic nervous system (SNS) predominating [28]. Hypertension in obesity is associated with increased SNS activity [29]. One measure of SNS activity during stress is an increase in BP and pulse rate. Fagerberg et al. [30] found reductions in pulse rate, but not BP, following weight loss in the range of 5-12 kg were associated with decreases in indices of SNS activity. In our study, weight loss may have resulted in a reduction in SNS activity, as there was a significant fall of pulse rate of 5 bpm during stress in the intervention group, although this did not differ from the reduction of 0.4 bpm in the control group.

Subjects in the intervention group were recruited from a larger study which investigated the effect of two weight loss diets on BP: WELL (moderate-sodium, high-potassium, high-calcium, low-fat DASH-type diet) or LF diet [15]. This study demonstrated that for a similar weight loss, there was greater reduction in home BP for the WELL diet. In the present study, there was a trend for an overall lower SBP during the stress procedure in the group allocated to the WELL diet, although this was not significantly different from the LF diet. However,
overall DBP fell 4.8 mmHg more in the WELL compared to LF group, and DBP was 5% lower 9-36 min after stress. These findings are of interest; however, further studies are required to confirm these results.

A weight loss of 5% reduced SBP during rest, SBP and pulse rate during recovery after mental stress, and the time taken to reach resting SBP levels following stress. This modest weight loss is likely to have a favorable effect on cardiovascular risk as the length of time that BP and pulse rate is raised during the day would be significantly reduced.
REFERENCES


[10] Schneider GM, Jacobs DW, Gevirtz RN, O'Connor DT. Cardiovascular haemodynamic response to repeated mental stress in normotensive subjects at genetic risk of


Figure legends

**Fig.1.** Mean ± SEM systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse rate values for the intervention and control groups at baseline and post-treatment stress tests. A, B and C represent baseline levels for SBP, DBP and pulse rate. D, E and F represent post-treatment levels for SBP, DBP and pulse rate. Solid and dashed lines represent data for the intervention and control group, respectively. *Significant (P < 0.05) between group differences, (independent samples t tests).

**Fig.2.** Mean ± SEM percentage changes (post-treatment – baseline) in blood pressure and pulse rate for the WELL, Low Fat (LF) and control groups during each part of the stress test. asignificantly different from control; bsignificantly different from LF. SBP = Systolic blood pressure; DBP = Diastolic blood pressure.
**TABLE 1.** Baseline characteristics of the intervention and control groups\(^1\)

<table>
<thead>
<tr>
<th></th>
<th>Intervention group n = 33</th>
<th>Control group n = 31</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening office SBP (mmHg)</td>
<td>135.9 ± 13.0</td>
<td>130.3 ± 9.1</td>
</tr>
<tr>
<td>Screening office DBP (mmHg)</td>
<td>90.2 ± 8.4*</td>
<td>81.7 ± 8.2</td>
</tr>
<tr>
<td>Age (y)</td>
<td>48.7 ± 9.9 (29.5-66.0)</td>
<td>49.9 ± 11.8 (27.4-76.3)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>92.7 ± 8.3</td>
<td>89.6 ± 11.8</td>
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<tr>
<td>BMI (kg/m(^2))</td>
<td>30.0 ± 2.1</td>
<td>28.8 ± 3.1</td>
</tr>
<tr>
<td>Resting SBP (mmHg)</td>
<td>126.8 ± 8.5</td>
<td>125.4 ± 7.6</td>
</tr>
<tr>
<td>Resting DBP (mmHg)</td>
<td>83.0 ± 5.8</td>
<td>84.3 ± 8.3</td>
</tr>
<tr>
<td>Resting pulse rate (bpm)</td>
<td>69.4 ± 11.9</td>
<td>63.9 ± 10.5</td>
</tr>
</tbody>
</table>

\(^1\)All values are mean ± SD; range in parentheses.

\(^2\)Data were missing for one subject, final n=32

\(*P < 0.05,\) intervention versus control group
TABLE 2
BP and pulse rate percentage changes for the intervention and control groups after 12 wk of treatment

<table>
<thead>
<tr>
<th>Phase</th>
<th>Intervention group</th>
<th>Control group</th>
<th>P-value</th>
</tr>
</thead>
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<tr>
<td></td>
<td>n = 28</td>
<td>n = 27</td>
<td></td>
</tr>
<tr>
<td>Resting SBP</td>
<td>-2.0 ± 1.1</td>
<td>+2.0 ± 1.1</td>
<td>0.013</td>
</tr>
<tr>
<td>Resting DBP</td>
<td>-2.0 ± 1.4</td>
<td>+1.3 ± 1.5</td>
<td>0.112</td>
</tr>
<tr>
<td>Resting Pulse rate</td>
<td>-2.8 ± 1.8</td>
<td>+2.9 ± 2.8</td>
<td>0.086</td>
</tr>
<tr>
<td>Stress SBP</td>
<td>-3.7 ± 1.4</td>
<td>-1.6 ± 1.2</td>
<td>0.257</td>
</tr>
<tr>
<td>Stress DBP</td>
<td>-3.6 ± 1.6</td>
<td>-0.4 ± 1.2</td>
<td>0.102</td>
</tr>
<tr>
<td>Stress Pulse rate</td>
<td>-5.7 ± 1.8</td>
<td>-0.001 ± 3.2</td>
<td>0.117</td>
</tr>
<tr>
<td>Reactivity SBP</td>
<td>-23.7 ± 14.1</td>
<td>-37.4 ± 16.2</td>
<td>0.525</td>
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<tr>
<td>Reactivity DBP</td>
<td>45.8 ± 58.3</td>
<td>-55.5 ± 23.1</td>
<td>0.112</td>
</tr>
<tr>
<td>Reactivity Pulse rate</td>
<td>-44.7 ± 25.9</td>
<td>-139.8 ± 168.0</td>
<td>0.572</td>
</tr>
<tr>
<td>Recovery 1 SBP</td>
<td>-3.1 ± 1.2</td>
<td>+0.2 ± 1.2</td>
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<tr>
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<td>-2.02 ± 1.3</td>
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<td>Recovery 1 Pulse rate</td>
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<td>+2.2 ± 2.6</td>
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<td>Recovery 2 SBP</td>
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<td>+0.4 ± 1.1</td>
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<td>Recovery 2 Pulse rate</td>
<td>-5.6 ± 1.6</td>
<td>+1.3 ± 2.2</td>
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<tr>
<td>Recovery 3 SBP</td>
<td>-2.4 ± 1.2</td>
<td>+0.6 ± 1.3</td>
<td>0.097</td>
</tr>
<tr>
<td>Recovery 3 DBP</td>
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<td>0.505</td>
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<tr>
<td>Recovery 3 Pulse rate</td>
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<td>+1.65 ± 1.5</td>
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<td>DBP</td>
<td>Pulse rate</td>
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<td>+2.9 ± 2.4</td>
<td>0.006</td>
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</tbody>
</table>

¹All data are mean ± SEM percentage changes, post-treatment minus baseline.

²P-value comparing intervention and control groups (independent samples t-test).

³SBP (Systolic blood pressure).

⁴DBP (Diastolic blood pressure).

⁵Reactivity in SBP, DBP and pulse rate calculated as stress minus resting values.

⁶Minute 22 to minute 30 of the stress test.

⁷Minute 33 to minute 39 of the stress test.

⁸Minute 42 to minute 48 of the stress test.

⁹Minute 51 to minute 57 of the stress test.
Figure 1

Baseline

A

D

Post-treatment

B

E

C

F

SBP (mmHg)

resting     stress                         recovery

Time (min)     0           14          21            30          39           48           57

115

120

125

130

135

140

145

150

75

80

85

90

95

100

75

80

85

90

95

100

58

62

66

70

74

78

82

resting     stress                          recovery

Time (min) 0           14          21            30          39           48           57

Pulse rate (bpm)
Figure 2

- **% Change in SBP**
  - Resting: 0
  - Stress: 14
  - Recovery: 21

- **% Change in DBP**
  - Resting: 0
  - Stress: 14
  - Recovery: 21

- **% Change in pulse rate**
  - Resting: 0
  - Stress: 14
  - Recovery: 21