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# Blood Pressure Biofeedback Exerts Intermediate-Term Effects on Blood Pressure and Pressure Reactivity in Individuals with Mild Hypertension: A Randomized Controlled Study

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## ABSTRACT

**Objective:** This randomized controlled study examined whether a 4-week blood pressure (BP) biofeedback program can reduce BP and BP reactivity to stress in participants with mild hypertension.

**Methods:** Participants in the active biofeedback group ($n = 20$) were trained in 4 weekly laboratory sessions to self-regulate their BP with continuous BP feedback signals, whereas participants in the sham biofeedback group ($n = 18$) were told to manipulate their BP without feedback signals. BP, skin temperature, skin conductance, BP reactivity to stress, body weight, and state anxiety were assessed before training and repeated at the eighth week after the training.

**Results:** The decreases in systolic ($12.6 \pm 8.8$ versus $4.1 \pm 5.7$) and mean BP ($8.2 \pm 6.9$ versus $3.3 \pm 4.9$) from baseline at week 12 follow-up were significantly greater in the active biofeedback group compared with the sham biofeedback group ($p = 0.001$ and 0.017, respectively). Results from analysis of covariance with the follow-up systolic blood pressure (SBP) (or mean arterial pressure [MAP]) as the dependent variable, baseline SBP (or MAP) as the covariate, and group as the independent variable showed that biofeedback training effectively lowered SBP and MAP ($p = 0.013$ and 0.026, respectively). The pre-to-post differences in skin conductance and SBP reactivity were statistically significant for the biofeedback group ($p = 0.005$ and 0.01, respectively), but not for the control group. For the sample as a whole and for the biofeedback group, the state anxiety score and body weight remained unchanged.

**Conclusions:** BP biofeedback exerts a specific treatment effect in reducing BP in individuals with mild hypertension, possibly through reducing pressor reactivity to stress.

Benefits of lowering blood pressure (BP) in hypertensive patients are well documented;[1,2] however, BP control remains a major public health problem. In the United States, approximately 27% of adults age 25 and older have hypertension.[3] In 77% of these cases, the condition is either untreated or uncontrolled. According to the National Health and Nutrition Examination Survey in the United States, BP control (systolic blood pressure [SBP] $<140$ and diastolic blood pressure [DBP] $<90$ mm Hg) rates in adults ages 18–74 between 1999 and 2000, though improved as

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compared to previous data, are still far below the Healthy People 2010 goal of 50%; 30% are still unaware they have hypertension and only 34% have their BP under control.[4] Likewise, the prevalence of hypertension in Taiwan is 26% in males and 19% in females, age 19 and older, according to a survey conducted between 1993 and 1996.[5] Of those Taiwanese adults with hypertension, 78% of males and 61% of females did not know they had hypertension, 13% of males and 28% of female took medications for it, and only 2% of males and 5% of females had their BP under control. Clearly, the rates of BP control in Taiwan are far from optimal.

Poor control of hypertension can be attributed, in part, to poor adherence to antihypertensive therapy. Depending on the medications prescribed, approximately 29% to 56% of patients who are initially prescribed antihypertensive medications discontinue therapy by the end of the first year.[6] Although the latest Joint National Committee on Blood Pressure Prevention, Detection, and Treatment of High Blood Pressure guideline did not include complementary and alternative medicine (CAM) in the treatment recommendation for all stages of hypertension,[4] it has been reported that about 64% of hypertensive patients used CAM in a study of 521 consecutive patients visiting a hypertension clinic in India.[7] In that study, fear of adverse effects of conventional pharmacotherapy was the most commonly cited reason for CAM use in patients with hypertension. There is thus a need to study the value and feasibility of CAM therapies, including biofeedback, so that patients—and their providers—can make informed decisions. To date, there has been a paucity of studies that examine the efficacy of these therapies.

Various biobehavioral interventions have been used to lower BP. Previous studies that determined the efficacy of behavioral interventions have included patients who had been treated pharmacologically. Consequently, it is difficult to separate the contribution of pharmacotherapy from the BP-lowering effects of behavioral treatments. Although it has been suggested that multiple-component behavioral interventions are more effective than any single modality,[8] it remains of interest to assess whether such a single modality as direct BP biofeedback can exert specific BP-lowering effects. Moreover, there are few studies that examine intermediate- and/or long-term treatment effects of direct BP biofeedback, and there are limited data available regarding mechanisms involved in BP self-regulation. The purpose of this study was to examine whether a 4-week BP biofeedback program can reduce BP and BP reactivity to stress in untreated individuals with stage I hypertension. Specifically, we tested whether BP biofeedback exerts a specific intermediate-term treatment effect other than a generalized relaxation effect that may be induced by regularly eliciting the relaxation response or simply regularly visiting a therapist. To that end, we used an active control condition and a randomized design.

## METHODS

### *Participants*

This study was approved by the Institutional Review Board of Taipei Medical University. Participants were volunteers recruited through BP screening, flyers, and referral from physicians. Participants were eligible if they were between 19 and 56 years old, able to read and write, and diagnosed with stage I hypertension (SBP from 140 to 159 mm Hg or DBP from 90 to 99 mm Hg). The average of four BP readings taken on two separate occasions, at least a week apart, was used to make the diagnosis of hypertension. Participants were excluded from the study if they had received any cardiovascular medication or had received medications for hypertension within the previous 2 months. Other exclusion criteria included chronic kidney or liver disease, neurologic disorders, psychiatric disorders, or diabetes. Each potential participant was seen by a cardiologist to verify the eligibility to participate in the study. All participants gave written informed consent.

This study utilized a randomized controlled design. All participants were informed about appropriate lifestyle modifications to control hypertension, such as weight loss, regular exercise, low alcohol consumption, sodium restriction, smoking cessation, and stress management. However, lifestyle modifications were not part of the BP biofeedback program. With a permuted block randomization design, participants were then randomly assigned to the active biofeedback (treatment condition) group ($n = 21$) or the sham biofeedback (control condition) group ($n = 21$). The active biofeedback group received 4-week direct BP biofeedback training with beat-to-beat BP signals, whereas the sham biofeedback group received 4-week BP self-regulation training without BP feedback signals. The data collectors were blind to the group assignment. The informed consent forms were written in a way that concealed the study treatment and the control condition. An independent research assistant generated the random allocation sequence using computerized software, and this was concealed until the follow-up data collection was completed. Figure 1 shows the flow of the participants in the study.

### *Instruments*

*Noninvasive beat-to-beat finger arterial pressure monitor.* Beat-to-beat BP signals were obtained with the use of finger arterial pressure measurement device (Finometer, TNO Biomedical Instrumentation, Amsterdam, Netherlands). The Finometer device permits the reconstruction of brachial pressure from noninvasive finger arterial pressure measurements by applying a generalized waveform filter. This instrument operates through an inflatable finger pressure cuff equipped with an infrared photoplethsymograph devised to measure finger artery blood volume under the cuff.

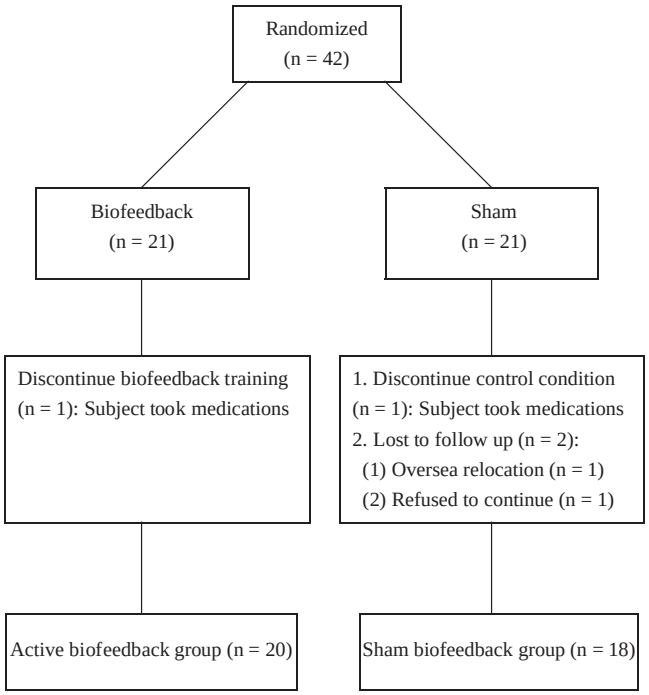


**FIG. 1.** Flow of participants in the study.

*Automatic blood pressure recorder.* BP and heart rate (HR) were recorded by an autonomic noninvasive cuff-oscillometric recorder (Model 90207, SpaceLabs™ Inc., Redmond, WA). Multiple resting measurements 2 minutes apart were taken 5 minutes after participants were seated. The mean of the two readings that agreed within 5 mm Hg was defined as the clinic BP.

*Finger temperature and skin conductance measurements.* Finger skin temperature was measured using a thermistor taped to the ventral surface of the distal phalanx of the right index finger. Skin conductance was measured by attaching the two electrodes to the palmar surfaces of the middle phalanxes of the middle and fourth fingers. Skin temperature and conductance signals were recorded using a Procomp$^+$ encoder coupled with BioGraph 2.1 software (Thought Technology Ltd., West Chazy, NY).

*State-Trait Anxiety Inventory.* The Spielberger State-Trait Anxiety Inventory (STAI) consists of two separate 20-item self-report scales for measuring state and trait anxiety.[9] The state measure of the STAI, which examines the level of anxiety that the person experiences at a given moment in time, was administered to all subjects at baseline and follow-up to evaluate the nonspecific effects produced by anxiety reduction.

## *Laboratory stress testing*

A laboratory stress session was used to induce BP reactivity. The session involved participants completing a stress task—the Stroop Color and Word Test (SCWT) while beat-to-beat BP was measured.[10,11] The SCWT is composed of a color-name reading task, a color-naming task, and an interference task. Briefly, the stress task was preceded by a 2-minute baseline period and followed by a 2-minute recovery period. BP and HR were continuously recorded before, during, and after the SCWT using a noninvasive beat-to-beat finger arterial pressure monitor (Finometer). BP reactivity was defined as the difference in mean pressures between the task and the baseline period.

## *Interventions*

After completion of the baseline data collection, each participant received a 10-minute individual training of a slow and deep diaphragmatic breathing technique. The biofeedback training program was composed of four weekly BP treatment sessions. Each treatment session lasted for approximately 40 to 45 minutes (a 5-minute briefing period, a 30-minute BP self-regulation training period, and a 5- to 10-minute session review). Both BP biofeedback and sham biofeedback training were administered by a trained nurse under the supervision of a certified biofeedback therapist.

*Blood pressure self-regulation with biofeedback (active biofeedback).* Self-regulation with biofeedback is based on the concept of operant conditioning described by the behavioral psychologist, Burrhus Frederic Skinner.[12] During each of the four treatment sessions, participants in the active biofeedback group performed three trials of BP self-regulation while observing the BP display. The continuous, beat-to-beat BP signals were displayed using the Finometer. Each trial consisted of four periods during which participants practiced manipulating their SBP, including (1) lowering BP (3 minutes), (2) resting (1 minute), (3) raising BP (1 minute), and (4) lowering BP (3 minutes). During the lowering or raising BP periods, the therapist instructed the participants to watch the SBP display and maintain their SBP below or above a predetermined target SBP level. The participant attempted, through taking slow and deep breaths and adopting a passive attitude, to bring the SBP level below the target level during the lowering BP periods. During the raising BP period, the participant was instructed to engage in mental actions to bring the SBP level above the target level. The average SBP level during a quiet resting period immediately before training was used as the initial target SBP. If a participant successfully maintained an SBP below the target level throughout 75% of the second lowering BP period, the target SBP for the following trial was lowered by 5 mm Hg.

*Blood pressure self-regulation without biofeedback (sham biofeedback).* Similar to the active biofeedback group, the sham biofeedback group was told to manipulate their BP in 4 laboratory sessions. However, SBP feedback signals were not provided to the control group. For each session during the treatment period, the sham biofeedback group

performed three trials of BP self-regulation. Each trial consisted of four periods similar to those for the experimental group. Similar to the active biofeedback group, participants in the control group were told to lower their BP by taking slow and deep breaths while adopting a passive attitude and to raise BP by engaging in some sort of mental actions.

After the 4-week training program, all participants were encouraged to continue practicing slow and deep breathing at least twice daily, although biofeedback were not available to either group. To maintain the treatment effects, each participant was advised to self-monitor BP at home for the 8 weeks of the follow-up period.

### *Data collection and management*

After the eligibility screening and prior to the trial, all participants underwent a 2-week run-in period, during which BP was measured weekly. The run-in period was used to ensure that all participants were in a stable condition. After the run-in period, clinic BP, skin temperature, and conductance, height, body weight (BW), and STAI were measured and recorded as baseline measurements. All baseline measurements were recorded prior to randomization. After the 4-week treatment period, there was an 8-week follow-up period. Research assistants who collected the physiologic measurements at baseline and during the follow-up period were blind to the group assignments. Clinic BP, skin temperature, skin conductance, BW, and STAI were measured in the resting state for all participants at 8 weeks after the treatment period.

To test the effect of direct BP biofeedback training on BP reactivity, the laboratory stress session was used at baseline and 8 weeks after the treatment period.

### *Study outcomes*

The primary outcomes of this study were the decreases in clinic SBP and mean arterial pressure (MAP) at the week-12 follow-up from the baseline levels. The secondary outcome was the decrease in SBP reactivity to stress at the week-12 follow-up from the baseline level.

### *Statistical procedures*

The group differences in biodemographic, lifestyle, anxiety, and physiologic data at baseline were compared using the Student's *t* test for continuous variables and the chi-square test or Fisher's Exact test for nominal variables. The differences between baseline BP and follow-up BP means were calculated for each group. A group comparison of the change score between groups was performed with the use of *t* test and 95% confidence interval. The efficacy of direct BP biofeedback on SBP and MAP reduction was examined using analysis of covariance (ANCOVA) with the baseline measures as covariate and follow-up measures as the dependent variable. The pre-to-post test differences of skin temperature, conductance, SBP reactivity, and anxiety were examined with the paired *t* test.

## RESULTS

The participants were recruited from October 1, 2003 through February 28, 2004, with data collection between January 2004 and June 2005. A total of 42 subjects consented to participate in this study and were randomized to the active biofeedback group ($n = 21$) and the sham biofeedback group ($n = 21$). One (1) participant in the active biofeedback and one in the sham biofeedback group were excluded from the data analysis because they took antihypertensive medications during the study period. Two (2) participants (1 refused to continue and 1 relocated to an overseas area) in the sham biofeedback group withdrew from the study during the follow-up period.

Included in the analysis were 38 individuals (men and women), ages 20 to 55 years (mean 43.1 ± 10.9), with stage I hypertension. The two study groups were comparable in biodemographic, anxiety, and lifestyle data (Table 1). The age and the percentage of women were lower in the sham biofeedback group than in the active biofeedback group, but the differences did not reach statistical significance. For both

Table 1. Biodemographic Data, Anxiety Level, and Lifestyle Habits of the Two Study Groups at Baseline

| | *Group* | | |
|---|---|---|---|
| | *Biofeedback (n = 20)* | *Sham (n = 18)* | p |
| BW (kg)[a,b] | 68.5 ± 7.7 | 74.4 ± 11.9 | 0.073 |
| BMI (kg/m$^2$)[a,b] | 26.1 ± 3.1 | 26.4 ± 3.7 | 0.754 |
| Age (years)[a,b] | 46.5 ± 10.3 | 39.9 ± 10.8 | 0.083 |
| Education (%)[c] | | | 0.684 |
| ≤High school | 35 | 22 | |
| College | 55 | 66 | |
| ≥Graduate school | 10 | 12 | |
| Sex (%)[d] | | | 0.101 |
| Male | 50 | 78 | |
| Female | 50 | 22 | |
| Marital status (%)[d] | | | 0.709 |
| Yes | 80 | 72 | |
| No | 20 | 28 | |
| S-Anxiety[a,b] | 38.5 ± 10.3 | 39.8 ± 11.1 | 0.704 |
| Smoking (%)[d] | | | 0.344 |
| Yes | 20 | 5 | |
| No | 80 | 95 | |
| Exercise (%)[c] | | | 0.54 |
| Yes | 68 | 53 | |
| No | 32 | 47 | |
| Family history of hypertension (%)[c] | | | 0.56 |
| Yes | 65 | 78 | |
| No | 35 | 22 | |

[a]Values are expressed as means ± standard deviation.
[b]Group comparison by *t* test.
[c]Group comparison by chi-square test.
[d]Group comparison by Fisher's Exact test.
BMI, body-mass index; S-Anxiety, state measure of the Spielberger State Trait Anxiety Inventory; BW, body weight.

groups, the lifestyle variables including smoking status and exercise habit did not change significantly from pretest to post-test (data not shown). For the sample as a whole and for the biofeedback group, baseline and follow-up BW were not significantly different ($p = 0.237$ and 0.667, respectively). Similarly, anxiety remained unchanged for the sample as a whole and for the biofeedback group ($p = 0.098$ and $p = 0.721$, respectively).

Baseline, follow-up, and change score of SBP and MAP data were calculated for both groups (Table 2). Baseline SBP was significantly higher ($p = 0.014$) in the active biofeedback than in the sham biofeedback group. The decreases in SBP and MAP levels from baseline at week 12 follow-up were significantly greater in the active biofeedback group as compared with the sham biofeedback group ($p = 0.001$ and $p = 0.017$, respectively). The sham biofeedback group had more males than the active biofeedback group. However, the differences in baseline BP, follow-up BP, and change score between sexes were not statistically significant (data not shown).

Results from the ANCOVA with the follow-up SBP (or MAP) as the dependent variable, baseline SBP (or MAP) as the covariate, and group as the independent variable showed that biofeedback training significantly lowered SBP and MAP ($p = 0.013$ and 0.026, respectively, Table 3).

A comparison of the baseline and follow-up measurements of skin temperature and conductance between the active biofeedback and the sham biofeedback groups did not reveal statistically significant differences (Table 4). The pre-to-post paired differences in skin conductance were statistically significant for the biofeedback group ($p = 0.005$), but not for the control group ($p = 0.59$). The pre-to-post paired differences in skin temperature were not statistically significant for either group (Table 4).

SBP reactivity to SCWT was not significantly different between groups at baseline and follow-up (Table 4). The pre-to-post paired SBP reactivity differences were statistically significant for the biofeedback group ($p = 0.01$), but not for the control group ($p = 0.056$).

Table 2. Results of Blood Pressure Biofeedback for Systolic and Mean Arterial Pressure Reduction

| | *SBP (mm Hg)* | | *Differences between means* | |
|---|---|---|---|---|
| | *Biofeedback* | *Sham* | *(95% CI)* | p |
| Baseline | 148.4 ± 8.6 | 142.1 ± 5.9 | 1.3–11.2 | 0.014 |
| Follow-up[a] | 135.7 ± 11.2 | 138.1 ± 5.5 | −8.2–3.6 | 0.432 |
| Change scores | 12.6 ± 8.8 | 4.1 ± 5.7 | 3.6–13.5 | 0.001 |
| | *MAP (mm Hg)* | | *Differences between means* | |
| | *Biofeedback* | *Sham* | *(95% CI)* | p |
| Baseline | 112.6 ± 7.1 | 110.1 ± 6.2 | −1.8–6.9 | 0.246 |
| Follow-up[a] | 104.4 ± 10.2 | 106.8 ± 6.3 | −7.9–3.1 | 0.384 |
| Change scores | 8.2 ± 6.9 | 3.3 ± 4.9 | 0.9–8.9 | 0.017 |

Values are expressed as mean ± standard deviation. Group comparison by *t* test.

[a]Eight weeks after blood pressure biofeedback or control condition.

CI = confidence interval; SBP = systolic blood pressure; MAP = mean arterial pressure.

Table 3. Efficacy of Direct Blood Pressure Biofeedback for Hypertension by Analysis of Covariance

| | *Systolic blood pressure* | | | |
|---|---|---|---|---|
| | *β* | *SE* | p | *95% CI for β* |
| Constant | 36.758 | 23.117 | 0.121 | −2.234–94.610 |
| Baseline | 0.713 | 0.162 | <0.001 | 0.383–1.042 |
| Group[a] | −6.771 | 2.682 | 0.013 | −12.00–−1.541 |
| | *Mean arterial pressure* | | | |
| | *β* | *SE* | p | *95% CI for β* |
| Constant | 6.518 | 16.892 | 0.702 | −27.775–40.810 |
| Baseline | 0.911 | 0.153 | <0.001 | 0.601–1.222 |
| Group[a] | −4.729 | 2.027 | 0.026 | −8.844–−0.613 |

[a]1 = active biofeedback group, O = sham biofeedback group. SE, standard error; CI, confidence interval.

*Note 1:* Follow-up systolic blood pressure for the active biofeedback group = 36.8 + 0.7 × (baseline systolic pressure) − 6.8 × (group).

*Note 2:* Follow-up mean arterial pressure for the active biofeedback group = 6.6 + 0.9 × (baseline mean arterial pressure) − 4.7 × (group).

## DISCUSSION

This study examined the efficacy of BP self-regulation with biofeedback against an active control condition in a randomized controlled design in mild hypertensive individuals. The nurse-guided BP self-regulation techniques used in this study were well received by the participants, and the overall withdrawal rate was less than 10%. Our findings demonstrated that BP self-regulation with continuous SBP signals (i.e., BP biofeedback) is capable of reducing SBP and MAP by 12.6 mm Hg and 8.2 mm Hg, respectively, at 8 weeks after training. In addition, BP biofeedback exerts a specific treatment effect other than a generalized relaxation response because the pre-to-post decrease in SBP and MAP were significantly greater in the biofeedback group. This notion was further supported by the finding that anxiety level was not significantly different between pretest and post-test. As such, lowering anxiety level as a result of adapting to the research environment could not account for the BP-lowering effect observed for the BP biofeedback. One might argue that the group difference could have been a result of

Table 4. Group Comparison of Baseline and Follow-up Differences in Skin Temperature, Skin Conductance and Blood Pressure Reactivity

| | *Skin temperature (°F)* | | |
|---|---|---|---|
| | *Biofeedback* | *Sham* | p |
| Baseline[a] | 90.3 ± 5.0 | 90.9 ± 3.3 | 0.921 |
| Follow-up[a,b] | 89.8 ± 6.3 | 92.1 ± 2.1 | 0.139 |
| Paired differences[c] | −0.5 ± 7.1 ($p = 0.74$) | 1.3 ± 3.9 ($p = 0.19$) | |
| | *Skin conductance (μS)* | | |
| | *Biofeedback* | *Sham* | p |
| Baseline[a] | 5.4 ± 3.7 | 5.5 ± 4.2 | 0.641 |
| Follow-up[a,b] | 2.8 ± 1.6 | 4.5 ± 5.5 | 0.239 |
| Paired differences[c] | −2.5 ± 3.4 ($p = 0.005$) | −0.7 ± 5.5 ($p = 0.59$) | |
| | *Systolic blood pressure reactivity (mm Hg)* | | |
| | *Biofeedback* | *Sham* | p |
| Baseline[a] | 12 ± 8.5 | 10.1 ± 8 | 0.485 |
| Follow-up[a,b] | 5.1 ± 5.7 | 4.1 ± 8.4 | 0.694 |
| Paired differences[c] | −7.2 ± 10.7 ($p = 0.01$) | −5.9 ± 12.2 ($p = 0.056$) | |

Values are expressed as mean ± standard deviation.
[a]Group comparison of baseline and follow-up scores by *t* test.
[b]Eight (8) weeks after blood pressure biofeedback or control condition.
[c]Within-group paired differences examined by paired *t* test.

"regression to the mean" because the large baseline differences in BP between groups. However, the ANCOVA procedure used presumably corrects for the baseline differences in BP between groups. Moreover, we used a 2-week run-in period during which BP was measured weekly to ensure that all participants were in a stable condition prior to the trial. Therefore, it is unlikely that the observed group differences were simply a result of an inflation of treatment effects because of an unstable baseline.

The BP-lowering effects of the BP biofeedback observed in the present study are comparable to those demonstrated by previous studies of short-term (i.e., 2 weeks) BP biofeedback effects.[13,14] The belief that BP biofeedback exerts specific treatment effects for controlling of BP has been challenged by a previous study in that acute BP-lowering capability was not superior on active BP biofeedback compared with placebo biofeedback.[15] Our study differed from that study in that we examined intermediate-term instead of acute BP-lowering effect of BP biofeedback and the outcome was assessed 8 weeks after the treatment period.

To elucidate possible mechanisms underlying the beneficial effects of BP biofeedback for BP control, we measured indices of autonomic nervous activity (i.e., skin temperature and skin conductance) as well as used a stress protocol to examine stress reactivity before and 8 weeks after the treatment period. As can be seen in Table 4, both groups had high skin temperature at baseline and the changes were not significant after training. We found that biofeedback training significantly lowered the level of skin conductance. Skin conductance, also known as electrodermal activity, represents changes in electrical conductance of the skin caused by an increase in activity of sweat glands that result from sympathetic activity.[16] In addition, our findings revealed that the BP-lowering effect of BP biofeedback is at least in part attributed to an attenuation of SBP reactivity to mental stress (i.e., SCWT). These findings were consistent with findings from a previous study that BP biofeedback with autoshaping significantly attenuated BP reactivity to mental stress (i.e., mental arithmetic) testing.[13]

Hyperreactivity to stress has long been linked to the development of hypertension.[17,18] In the present study, BP biofeedback exerted intermediate-term effects on BP and pressor reactivity. A previous study demonstrated that the routine practice of a slow and regular breathing exercise for 8 consecutive weeks significantly reduced BP in individuals with hypertension.[19] Although long-term effects have yet to be determined, slow breathing has been reported to acutely improve baroreflex sensitivity in individuals with essential hypertension.[20] In the present study, both groups of subjects were trained to perform slow and deep breathing as a means to lower BP. With the addition of BP feedback signals for BP self-regulation training along with routine practice of a slow and deep breathing exercise, the active biofeedback group achieved better BP reduction outcomes than the sham biofeedback group. Thus, we speculate that repeated practice of BP self-regulation techniques using con-

tinuous BP feedback signals may very likely increase baroreceptor sensitivity so that the effect of daily stressors on BP is prevented or minimized. Enhanced baroreceptor sensitivity may also explain the apparent lack of group difference observed in a previous study in which one group was trained with continuous SBP signals whereas the other group was trained with partially disguised SBP signals.[15] The control group in our study, on the other hand, was told to self-regulate their BP but was prevented from viewing the actual BP signals. Because this study did not assess baroreceptor sensitivity, whether BP biofeedback exerts its BP lowering effect via enhancing baroreceptor sensitivity cannot be ascertained.

Other indices of autonomic nervous system such as measurements of heart rate variability and baroreceptor sensitivity can be used in future studies to explore the mechanism of BP-lowering effects of direct BP biofeedback. The hypothalamus–pituitary–adrenal (HPA) axis is the other stress system that may also be involved in the stress and the disease pathway.[21,22] The question of whether or not BP biofeedback has an effect on the HPA axis remained to be studied.

Several methodological issues may affect the validity of the study. First, repeated BP measurements may produce a BP-lowering effect. In this study, both groups of participants were advised to perform daily self-monitoring of BP. However, we did not monitor the adherence to self-measurements of BP. Second, the adherence to the practice of deep breathing during the 8 weeks of the follow-up period was not monitored. One may argue that the observed greater BP reduction in the active biofeedback group was, at least partially, accounted for by better compliance with the practice of deep breathing and self-monitoring of BP. Finally, the BP-lowering effects demonstrated by this study were small. Had there been more treatment sessions, for example, using the standard biofeedback training protocol—two training sessions a week for 8 weeks—the BP-lowering effect might have been more pronounced.

## CONCLUSIONS

In conclusion, direct BP biofeedback is efficacious in attenuating BP and pressor reactivity to stress in individuals with mild hypertension. The results of the present study provide evidence of efficacy for a future trial that examines the effect and mechanisms of direct BP biofeedback in patients with prehypertension to early hypertension.

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