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Device-Guided Paced Respiration as an Adjunctive Therapy for Hypertension in Obstructive Sleep Apnea: A Pilot Feasibility Study

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Abstract

Background—Data suggest that device-guided paced respiration (<10 breaths/minute) may reduce blood pressure in hypertensive patients. We hypothesized that daily device-guided slow breathing may lower blood pressure in patients with hypertension and obstructive sleep apnea (OSA).

Methods—In this one-arm pilot study, we enrolled 25 subjects with hypertension and OSA. Subjects were asked to perform device-guided paced respiration 30 minutes a day for 8 weeks. Our primary outcome was change in office systolic and diastolic blood pressures from baseline to 8 weeks.

Results—Twenty-four subjects completed the study. Mean baseline blood pressure was 140.0 ± 10.2 mmHg systolic and 82.7 ± 8.9 mmHg diastolic. Complete device data were available for 17 subjects. Mean device adherence was $81\% \pm 24\%$ and 51% achieved a mean breath rate 10 breaths/minute over 8 weeks. Three subjects had changes in their anti-hypertensive medications during the study. Among the remaining 21 subjects, mean difference in office blood pressure from baseline to 8 weeks was -9.6 ± 11.8 mmHg systolic ($p = < 0.01$) and -2.52 ± 8.9 mmHg diastolic ($p = 0.21$).

Conclusion—Device-guided paced respiration may lower systolic blood pressure in patients with hypertension and OSA; however, our findings need to be confirmed with larger randomized controlled trials.

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Keywords

paced respiration; breathing exercise; high blood pressure; non-pharmacological therapy; sleep apnea; lung

Introduction

Patients with obstructive sleep apnea (OSA) are frequently afflicted with other co-morbid conditions, such as hypertension,[1–4] diabetes,[5, 6] and obesity.[7, 8] Treatment of OSA improves some associated abnormalities, but the magnitude of these effects has been variable. For example, two meta-analyses have suggested that treatment of OSA with continuous positive airway pressure therapy (CPAP) yields an average improvement in daytime blood pressure of only about 2 mmHg.[9, 10] As a result, some have suggested adjunctive therapies to treat hypertension among those with sleep apnea.

Deep breathing exercises are some of the more commonly used alternative therapies, with nearly 13% of adults in the United States practicing annually.[11] While alternative mind-body therapies are most frequently used to treat psychiatric and pain conditions, one respiratory-biofeedback device, which uses slow deep breathing, is currently FDA-approved for the treatment for hypertension.[12] Vagally-mediated sympathoinhibition is often cited as the primary mechanism for the effects of deep inspiration upon blood pressure.[13] As such, positive airway pressure (PAP) devices, which are used to treat OSA and increase tidal volume, may also subsequently lower sympathetic activity. However, the enduring effects of such an intervention on systemic blood pressure are unknown.

Based on this logic, we sought to elucidate the feasibility and influence of a novel daytime PAP device upon clinical blood pressure measurements in patients with concomitant OSA and hypertension. Patients with OSA were studied due to their known sympathoexcitation as well as their ability and motivation to tolerate positive airway pressure therapy. Furthermore, a positive-airway pressure device provides a potent stimulus for reduction in breathing rates with minimal training, increases tidal volume, thereby enhancing possible sympatholytic effects, and could ultimately be incorporated into standard continuous positive airway pressure devices for use prior to sleep initiation in patients with sleep apnea. We hypothesized that device-guided paced respiration would yield important improvements in clinical daytime systemic blood pressure.

Methods

Study Sample

We recruited 25 patients previously diagnosed with hypertension and obstructive sleep apnea from the sleep disorders clinic at Brigham and Women's Hospital in Boston, MA, and the community using brochures, newspaper advertisements, and internet postings, for this one-arm pilot study. Participants with a history of hypertension were included if they were 20–75 years old, had an elevated mean office blood pressure ($>120/80$ mmHg) at screening, were either on a stable anti-hypertensive drug regimen or taking no anti-hypertensive medications for the past 3 months, and were using nocturnal continuous airway pressure (CPAP) at a stable setting for the past 3 months. We excluded participants with a mean systolic blood pressure >159 mmHg or diastolic blood pressure >99 mmHg at the screening visit, any known form of secondary hypertension (e.g., excessive alcohol intake; medication induced hypertension), moderate to severe chronic obstructive pulmonary disease, respiratory failure, cardiac disease (i.e., congestive heart failure, ischemic heart disease, or cardiac arrhythmia), neoplastic disorders other than non-melanoma skin cancers, high

caffeine intake (>5 cups of caffeinated beverages per day), an active or former (>10 pack-year) smoking history, and a health condition that would interfere with intervention (e.g., dementia; claustrophobia). All participants gave written informed consent before beginning this IRB-approved protocol.

Study design

After baseline assessment, participants were provided with the breathing device and instructed to use the device twice daily, during a period of wakefulness, for a total of 30 minutes per day for eight consecutive weeks. Participants were instructed to continue their nocturnal CPAP and anti-hypertensive medications as previously prescribed. Additionally, we asked participants not to modify their current diet or exercise regimen or initiate a practice inclusive of breathing exercises (e.g., yoga, meditation) during the study. Participants returned to our clinic for a second visit at four weeks and for a final visit at eight weeks.

Equipment/Intervention

During the baseline visit, participants were given detailed instructions on device use and underwent a coached practice session. Since we used a bi-level positive pressure device (BiPAP^R Pro 2, Philips Respironics, Murrysville, PA), we were able to titrate differentially the inspiratory and expiratory pressures high enough to allow the subject to differentiate inspiration from expiration, while maximizing comfort. After one minute of use, the device algorithm guided participants to reduce their respiratory rate to <10 breaths/minute by adjusting selected respiratory parameters. This respiratory rate was based upon prior clinical and physiological studies examining the effects of slow breathing on blood pressure and autonomic tone.[14–18] After 15 minutes of use, the device emitted an audible tone to alert the participants that the session had ended and reverted to spontaneous mode. Participants could then reset the machine to perform another paced breathing session, or choose to complete a second session at a later time. At this initial visit, participants were coached to ensure proper device use and attainment of a respiratory rate lower than their baseline rate. Participants were contacted within the first week of the initial study visit and at one-week intervals to ensure proper functioning of the device and to answer additional questions. Additional in-person coaching was offered as needed. The device recorded data for each session, including time and duration of each session, continuous respiratory rate, average tidal volume, and cumulative time of respiratory rate <10 breaths/minute. Device data were downloaded at subsequent study visits. Given that one of the primary objectives of this study was to assess feasibility (and to determine effect sizes) of the intervention, this was a one-arm open-label trial, without a control group.

Primary outcome measures

Blood Pressure Measurements—Clinical blood pressure measurements were based on a common protocol adapted from procedures recommended by the American Heart Association.[19] Blood pressure was measured with the participant in the sitting position after at least 5 minutes of rest. A sphygmomanometer was used, and one of two cuff sizes (regular adult or large) was chosen on the basis of the circumference of the participant's arm. Two separate blood pressure measurements were taken about 10 minutes apart and the average was calculated. Participants were asked to avoid alcohol and caffeine for six hours prior to their blood pressure measurement, and to avoid exercise for at least 30 minutes prior to their blood pressure measurement. Clinical blood pressure measurements were performed by the same study physician for each participant and at the same time of day, unless a scheduling conflict existed.

Additional data collection—Anthropometric data including height (baseline only) and weight at baseline, 4 weeks, and 8 weeks were collected. At all three study visits, patients underwent a history and physical by a study physician. Data on adverse events, side effects, changes in medications, and other changes in health status were also collected.

Statistical analysis—Data on blood pressure before and after device use were analyzed using two-tailed paired Student's t-tests. To explore further the relationship between paced respiration and change in clinical blood pressure measurements, we performed pre-specified analyses using Spearman's rank correlation coefficient. All analyses were performed using SAS statistical software, version 9.2 (Cary, North Carolina); P-values <0.05 considered significant.

Results

Table 1 lists the baseline patient characteristics of all 25 subjects enrolled in the study. In general, subjects were middle aged, taking anti-hypertensive medications, and had severe sleep apnea and obesity. One participant dropped out of the study due to time constraints. Three participants had changes in their antihypertensive regimen (one self-discontinued his medication when he perceived a hemodynamic benefit to paced breathing; two participants made medication errors and resumed therapy when medications were reconciled with their primary care physicians), and were thus excluded from the primary analysis (n=21).

Exposure Assessment: Feasibility

Seventy-one percent of participants (17/24) had complete device data (Table 2). Two subjects had only partial device data (i.e., only 4 weeks of data) and five subjects had no device data. We were not able to discern if this was due to technical issues or nonadherence. Overall adherence rates were satisfactory, with 76% of participants for whom we had complete device data (n=17) using the device for at least 70% of expected cumulative time. Mean breath rate during device use was higher than anticipated (11.3 ± 4.9 breaths/min), though was lower than spontaneous respiratory rates (16.3 ± 4.6 breaths/min, p-value <0.001). Fifty-three percent of participants achieved an average respiratory rate during paced respiration of <10 breaths/minute.

Outcomes: Clinical Blood Pressure

For our primary outcomes (n=21), systolic and diastolic blood pressures declined between baseline and eight weeks, with differences in systolic and mean arterial blood pressures achieving statistical significance (difference in systolic blood pressure, -9.6 ± 11.8 mmHg, p-value < 0.01; diastolic blood pressure -2.52 ± 8.9 mmHg, p-value 0.21; mean pressure -4.90 ± 8.4 , p-value 0.02). Individual participant changes in blood pressure are depicted in Figure 1. Overall, 71% of subjects achieved at least a 5 mmHg reduction in blood pressure, which is clinically significant.[20]

No adverse events related to the device were reported by participants enrolled in this study.

Exploratory Analyses

In additional analyses, we did not find correlations between changes in either systolic or diastolic pressure and mean breath rate during paced respiration over eight weeks (systolic blood pressure $r = -0.20$, $p = 0.42$; diastolic blood pressure $r = 0.16$, $p = 0.49$). Small sample sizes precluded analyses of the relationship of specific breath rate targets (e.g., <10 breaths/minute) achieved. Our data demonstrated a significant association between adherence to the device (i.e., use for 70% of expected cumulative time) and beneficial changes in mean arterial ($r = -0.58$, $p\text{-value} = < 0.01$) and diastolic pressure (-0.56 , $p\text{-value} = 0.01$), but not

systolic pressure. Lastly, we found no correlation between weight loss and changes in either systolic or diastolic blood pressure (p -values = 0.41 and 0.35, respectively).

Discussion

We found that patients with obstructive sleep apnea and hypertension utilizing a novel BiPAP^R device had a decrease in systolic blood pressure over eight weeks. Interestingly, declines in systolic, diastolic, or mean blood pressure were not associated with either mean breath rate or tidal volume during device use. However, improvements in mean arterial and diastolic pressures were correlated with greater adherence to the device.

Paced Breathing and Hypertension

Our results are consistent with several clinical studies evaluating the role of paced respiration (respiratory rate under 10 breaths per minute) for the long-term treatment of hypertension. Six trials published in English evaluating an audio-feedback device-guided paced respiration reported beneficial changes in the mean systolic blood pressure ranging from -6.4 mmHg to -15.0 mmHg, with smaller effects on diastolic pressure.[16, 17, 21–23]. Our results are also consistent with a recently published study examining the anti-hypertensive effects of slow breathing training via heart rate variability biofeedback. In this study, Nolan et al. demonstrated that hypertensive patients randomized to regular practice of slow breathing (i.e., 0.1Hz) experienced reductions in daytime and 24-hour systolic pressure (-2.4 ± 0.90 mmHg, $p < 0.01$; -2.06 ± 0.90 mmHg, $p < 0.05$), but not diastolic pressure when measured after 8 weeks of training.[24] However, our results contrast with the results of three recent randomized clinical trials that failed to demonstrate an improvement in systolic or diastolic blood pressure in patients performing device-guided paced respiration compared with a control activity (i.e., listening to music).[18, 25, 26] This discrepancy may reflect differences in study methodologies, including sample characteristics (e.g., people with diabetes) and lack of power to detect differences between groups. To our knowledge, no study has evaluated device-guided paced respiration for hypertension exclusively in patients with concomitant obstructive sleep apnea in whom sympathoexcitation is well known.[27] Given that positive airway pressure devices raise intrathoracic pressure whereas passive paced breathing devices lower pleural pressure, we would anticipate differential hemodynamic effects of these two techniques. As a result, the existing literature on paced breathing (using patient controlled active breathing with diaphragmatic contraction) may not be directly comparable to the present study.

Device-guided paced breathing researchers hypothesize that slow, deep breathing induces changes in blood pressure in hypertensive patients by reducing sympathetic activity. Physiological research has demonstrated acute respiratory modulation of direct measures of sympathetic activity (i.e., muscle sympathetic nerve activity (MSNA)). Seals et al. described profound respiratory modulation during both passive and mechanically-ventilated slow breathing in healthy participants and lung transplant patients, though the primary driving factor (i.e., respiratory rate or tidal volume) and resultant physiological correlates (e.g., lung inflation induced sympathoinhibition; baroreflex activation) were not experimentally uncoupled.[28, 29] Furthermore, while they observed strong breath-by-breath modulation of MSNA, overall MSNA did not differ among experimental respiratory patterns. In contrast, recent data have demonstrated acute reduction in muscle sympathetic nerve activity during slow passive respiration (i.e., around 6 breaths/min). Raupauch et al. demonstrated reductions in sympathetic nerve activity during 0.1Hz respiration with 0.25 Hz (61.3 ± 4.6 vs. 53.0 ± 4.3 bursts per incidence) in patients with chronic obstructive pulmonary disease.[30] Similarly, Oneda *et al.* reported a reduction in sympathetic nerve activity in patients when hypertension of -8 bursts/minute during slow breathing compared to no change in control listening to calm music.[31]

Additional evidence supporting the sympatholytic effects of slow breathing patterns comes from studies utilizing indirect measures, such as arterial baroreflex. For example, Joseph et al. demonstrated enhanced baroreceptor sensitivity during 0.10 Hz breathing compared with spontaneous breathing in hypertensive subjects. Furthermore, both systolic and diastolic blood pressure decreased during slow breathing ($149.7\text{mmHg} \pm 3.7$ to $141.1 \pm 4\text{mmHg}$ and 82.7 ± 3 to $77.8 \pm 3.7\text{mmHg}$).[32] Similarly, Reyes del Paso et al. reported 0.10 Hz respiration enhanced baroreceptor sensitivity in mildly hypertensive subjects.[33] However, the method of baroreflex sensitivity measures used in these studies has been criticized for its inability to distinguish the feedback effects of blood pressure on heart period from the feed-forward relations of the system.[34] The current literature, though limited in design and breadth, suggests that slow deep breathing may reduce sympathetic activity, however, whether these changes are sustained over time remains unknown.

Limitations

Though this study had many strengths, we address the following limitations in interpretation of our data. To begin, we had a relatively small sample size, particularly given the variability of clinical blood pressure measurements. We performed this pilot study to provide sufficient preliminary data to determine the feasibility and logistics of a more definitive study. Second, we studied only modest degrees of blood pressure elevation because we believe it is unethical to leave severe hypertension untreated in this high-risk population. Third, this was an unblinded one-arm pilot study, and therefore we could not distinguish whether clinical blood pressure improvements were due to device use, or rather to additional factors, such as placebo effects, regression to the mean, habituation to the blood pressure measurement, and/or the Hawthorne effect. We also could not assess whether positive-airway pressure device-guided breathing leads to differential effects compared with spontaneous slow breathing. Thus, blinded controlled studies are required, although we believe that pilot studies are critical before the expense and risk of large scale randomized controlled studies can be justified. Fourth, although individuals in this trial were given rigorous instructions regarding participation, it is possible that minor changes in diet, exercise, medication adherence, or sleep duration/CPAP use occurred during the course of our study. Because our goal was to study the long term effects of mechanical slow deep breathing, we did not feel it would be realistic to perform these studies in-laboratory which would likely be required to control all such variables and activities. Lastly, we did not achieve perfect adherence with the device. We see this as a result rather than a limitation. In clinical practice, adherence to therapy is often less than that achieved in clinical trials. In fact, we did attempt a similar study in non-OSA participants as a control arm, but abandoned this research when >50% of participants were non-adherent with therapy. Thus, adherence to the device may ultimately be an issue in achieving optimal results. Conversely, the sub-optimal use of the device still yielded important blood pressure improvements, a result, which should be biased towards the null hypothesis, i.e. blood pressure reductions may have been greater if better adherence had been achieved. Despite these limitations, we find our results to be encouraging and do plan further mechanistic and clinical research in this area.

Conclusion

In this pilot study, we demonstrated that device-guided paced respiration may produce clinically meaningful changes in systolic blood pressure in patients with obstructive sleep apnea and hypertension. Future research is needed to further evaluate positive airway pressure-guided paced respiration as an adjunctive therapy for hypertension in patients with sleep apnea and to elucidate underlying physiological mechanisms.

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Figure 1.
Change in Daytime Clinical Blood Pressure by Subject

Table 1

Participant Characteristics at Baseline (n=25)

Age, yrs, mean ($\pm$ SD)	55.0 (6.7)
Male, percent	52.0
SBP, mmHg, mean ($\pm$ SD)	140.0 (10.2)
DBP,mmHg, mean ($\pm$ SD)	82.7 (8.9)
On antihypertensive drug, percent	84%
Number of antihypertensive drugs, mean ($\pm$ SD)	1.5 (1.0)
Body mass index, kg/m ² , mean ($\pm$ SD)	38.9 (8.6)
AHI, events/hour,mean ($\pm$ SD)	41.3 (14.6)

SD:standard deviation

SBP:systolic blood pressure

DBP:diastolic blood pressure

AHI:apnea-hypopnea index

Table 2Daytime BiPAP^R Use and Respiratory Patterns (n=17)

Daytime BiPAP ^R Use Characteristic	Mean $\pm$ Standard Deviation
Adherence, % of expected use, over 8 weeks	80.9 $\pm$ 24.1
Session time/day, minutes	28.5 $\pm$ 2.7
Respiratory rate during device use, over 8 weeks, bpm	11.3 $\pm$ 4.9
Respiratory rate <10 /min, % total use	50.6 $\pm$ 37.7
Average tidal volume, Liters	699.1 $\pm$ 378.5

bpm= breaths per minute