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Short-term expiratory muscle strength training attenuates sleep apnea and improves sleep quality in patients with obstructive sleep apnea

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ABSTRACT

This study investigated the effects of 5 weeks of expiratory muscle strength training (EMST) on sleep apnea, sleep quality, and respiratory muscle strength in patients with different levels of obstructive sleep apnea (OSA). Twenty-five outpatients who received a diagnosis of OSA participated in the study and were assigned to either the EMST group (n = 13) or control group (CTRL, n = 12). The training intensity for the EMST group was 75% of the maximum static expiratory (PE_{max}) score (5 days/week). The PE_{max}, apnea–hypoapnea index (AHI), Epworth Sleepiness Scale (ESS), and Pittsburgh Sleep Quality Index (PSQI) scores were evaluated before and after the treatment. EMST improved the scores for AHI (−40%), PE_{max} (+68%), and PSQI (−28%) and reduced the PSQI scores of the moderate OSA subgroup but not the mild OSA subgroup. The percent changes (Δ%) in the AHI and PE_{max} scores of participants with OSA were negatively correlated. We demonstrated that EMST effectively improved sleep apnea, sleep quality, and expiratory muscle strength in participants with OSA. Participants with moderate OSA exhibited greater improvement than did those with mild OSA, and the improvement in PE_{max} scores was correlated with a decrease in sleep apnea.

1. Introduction

Obstructive sleep apnea (OSA), a highly prevalent sleep disorder in middle-aged adults, is characterized by sleep-related upper airway dysfunction and repetitive airway obstruction during sleep. This pathological impairment generally results in various levels of snoring and upper airway resistance syndrome (Redline et al., 2010; Young et al., 1993). By definition, OSA occurs when the muscles of the upper airway and intercostal regions relax during sleep, impeding breathing for at least 10 s. This leads to reduced nocturnal blood oxygenation, excessive daytime sleepiness (EDS), fatigue, and headaches (Yaman et al., 2007). Individuals who experience OSA and EDS simultaneously could therefore be at a substantial risk of work disability (Omachi et al., 2009; Sjosten et al., 2009). Previous studies have reported a strong association between OSA and the rapid development of degenerative diseases, such as coronary artery disease, arrhythmia, left ventricular dysfunction, diabetes, and hypertension (Chami et al., 2011; Frija-Orvoen, 2016; Somers et al., 2008). Moreover, patients with OSA do not exhibit identical conditions, and the severity of conditions differs among patients. Therefore, the apnea–hypopnea index (AHI) is a commonly used and salient clinical evaluation tool for quantitating the severity of OSA.

Therapeutic options for OSA are limited in clinical practice. Continuous positive airway pressure (CPAP) and upper airway surgery are two of the most widely prescribed options (Franklin et al., 2009; Kreivi et al., 2010; Lim et al., 2004). However, both are invasive and may cause discomfort in patients. These treatments can also be extremely costly; financial burdens are at least partially responsible for the limited success of long-term CPAP treatment, owing to low adherence (Kreivi et al., 2010). In summary, the positive benefits of these treatments remain inconclusive in the literature (Franklin et al., 2009; Lim et al., 2004).

Previous studies have revealed the ability of respiratory muscle strength training to attenuate OSA symptoms and enhance respiratory muscle strength (Beaumont et al., 2015; Vranish and Bailey, 2016; Wang et al., 2002). For example, a previous study reported that respiratory training using the didgeridoo (a traditional Australian wind instrument) reduced sleep apnea in participants with moderate OSA (Puhan et al., 2006). Because playing the didgeridoo primarily requires the use of expiratory muscles, expiratory muscle strength training (EMST) can be justifiably considered for the treatment of OSA. However, unlike using an EMST device, playing this musical instrument does not enable patients to precisely control the intensity of training by

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expiring against a resistive force. EMST has also been reported to improve respiratory capacity and muscle strength in patients with chronic obstructive pulmonary disease or Parkinson disease (Chiara et al., 2006; Mota et al., 2007; Pitts et al., 2009; Sapienza et al., 2011). Moreover, previous studies have reported that individuals who underwent respiratory muscle training involving both inspiratory and expiratory phases complained about difficulty and discomfort associated with the inspiratory phase, which may have affected their training outcomes. Thus, inspiratory muscle training may not be ideal for individuals with preexisting difficulty in breathing. To the best of our knowledge, no investigations to date have evaluated the effects of EMST using an intensity-controlled device on sleep quality in patients with OSA.

Thus, we hypothesized that EMST reduces OSA symptoms and improves sleep quality in patients with mild to moderate OSA. This study investigated the effects of a 5-week EMST program on sleep apnea, respiratory muscle strength, EDS, and sleep quality in patients with OSA.

2. Methods

2.1. Participants

Twenty-nine outpatients from a sleep disorder diagnostic center volunteered to participate in this study. The patients were aged between 20 and 60 years and were currently experiencing OSA (AHI > 5/h). The exclusion criteria were as follows: (1) body mass index (BMI) > 30 kg/m^2; (2) ongoing CPAP treatment or use of an antisnoring device; (3) having undergone uvulopalatopharyngoplasty or maxillomandibular advancement surgery; (4) experiencing periodic leg movements during sleep; (5) experiencing insomnia; (6) presence of any diseases related to the brain, lungs, or cardiovascular system, and (7) ongoing dialysis treatment. The present study was approved by the institutional review board (IRB) of Taipei Medical University, and all the tests and the training program were conducted only after the eligible participants provided written informed consent.

2.2. Experimental design and procedure

All participants were matched on the basis of sex, baseline AHI, and BMI. Next, they were randomly assigned to either the EMST or control (CTRL) group. Before the intervention (pretest), all participants underwent tests to determine their PE_{max}, AHI, Epworth Sleepiness Scale (ESS), and Pittsburgh Sleep Quality Index (PSQI) scores. After baseline data was collected, all participants received either sham control training (CTRL group) or EMST training (EMST group) during the 5-week intervention period, in accordance with the experimental design. After the intervention (posttest), all participants received identical evaluations as described in the pretest period.

2.3. Respiratory muscle strength training

EMST using an EMST trainer (EMST150; Aspire products LLC., USA) was selected as the treatment modality for the training program because of its proven effectiveness for improving muscle strength (Troche et al., 2010). With regard to training intensity, the resistance pressure for the EMST group was set at 75% of each participant's PE_{max} (frequency: 5 days/week; 25 breaths/day, 5 breaths/cycle for a total of 5 cycles/day) (Pitts et al., 2009; Sapienza et al., 2011; Troche et al., 2010). For the CTRL group, the resistance pressure was set at 0% of each participant's PE_{max} score. The remaining training conditions of the CTRL were identical to those of the EMST group. During training, each participant used a nose clip to ensure that inhalation and exhalation were performed orally. Participants were asked to inhale and then to exhale with as much force as possible. An interval of 30–60 s was allowed for rest after each breathing maneuver and a 2-min break was provided after each breathing cycle was completed. The participants used their hands to press both sides of their lips to prevent air leakage and muscle fatigue as well as to ensure that the exhaled air passed steadily through the EMST and achieved the target threshold pressure.

Throughout the 5-week intervention period, the PE_{max} scores of the participants in the EMST group were measured weekly to adjust the training intensities and ensure that the resistance pressure was maintained at 75% of their PE_{max} score (Baker et al., 2005; Sapienza et al., 2011). By contrast, the training intensity for the CTRL group was maintained constant at 0% of their PE_{max} score. The participants were required to maintain a training diary and weekly phone interviews were conducted to ensure the quality of training (Troche et al., 2010). Only those participants who completed at least 80% of the training regimen were included in further data analysis.

2.4. Evaluation of respiratory muscle strength

A respiratory muscle pressure meter (MicroRPM, UK) was used to determine the PE_{max} scores of the participants as an indicator of their respiratory muscle function. The PE_{max} score of each participant was measured 10 times, as suggested in the 2002 guidelines of the American Thoracic Society and European Respiratory Society. Disturbances such as coughing, leakage, and instrument blockage were avoided while the measurements were conducted, and three measurements with less than 5% variance were included in the calculation. The PE_{max} score was considered the maximum value (Society, 2002).

2.5. Apnea–hypopnea index

A polysomnography sleep diagnosis system (Embletta X100, USA) was used to assess the participants' AHI scores. The system was operated according to the manufacturers' instructions. An electroencephalogram, electrooculogram, and electromyogram were used to measure nasal airflow, chest and abdominal breathing movements, and blood oxygen saturation, respectively. The recorded physiological signals were interpreted by two licensed sleep technologists according to the standards of the American Academy of Sleep Medicine to ensure assessment quality. The AHI score of each participant was determined by dividing the total frequencies of apnea and hypopneas by the duration (in hours) of sleep (Iber et al., 2007).

2.6. Epworth sleepiness scale, Chinese version

The Epworth Sleepiness Scale, Chinese version (ESS-C) is based on a questionnaire developed by Murray Johns (Johns, 1992). The reliability and validity of the ESS-C were previously verified by Chen et al. (2002). Specifically, the Cronbach's alpha (internal consistency) of the scale was 0.81. Furthermore, the test–retest reliability of the ESS-C was examined before and 2 weeks after the treatment to assess the consistency of the scale. The correlation coefficient was 0.74 (Chen et al., 2002).

2.7. Pittsburgh sleep quality index, Chinese version

The Pittsburgh Sleep Quality Index, Chinese version (PSQI-C) is based on a questionnaire developed by Buysse et al. (Buysse et al., 1989). The reliability and validity of the PSQI-C were previously verified by Tsai et al. (2005). The measured and remeasured Cronbach's alpha values (internal consistency) were 0.83 and 0.82, respectively, and the correlation coefficient of test–retest reliability was 0.85. For total scores higher than 5, the sensitivity and specificity of the scale were 98% and 55%, respectively (Tsai et al., 2005).

2.8. Sample size calculation

On the basis of a previous study published by Vranish and Bailey (2016), we estimated that an approximate 4-point change (PSQI from 9.1 to 5.1, n = 12 per group, S.E.M = 0.9) in the measured statistics

would indicate a significant effect. The values used for determining the minimum number of participants required per group to determine a statistical difference at $\alpha = 0.05$ were ($Z\alpha = Z(0.05/2) = 1.96$), $\beta = 0.10(Z\beta = Z0.1 = 1.28)$, $\sigma = 3.12$, and $\Delta = 4$ based on the sample size formula $n = [2*\sigma^2*(Z\alpha + Z\beta)^2]/\Delta^2$. The calculated value of n was 12.75. Therefore, the number of participants needed per group was between 12 and 13. For the calculation, n is the sample size, Zα is the value for the two-tailed α, Zβ is the value for the one-tailed β, σ is the sample standard deviation, and Δ is the minimum difference between the study groups that can be considered important.

2.9. Statistical analyses

SPSS Version 19 for Windows was used for data analyses. The Mann–Whitney *U* test and Pearson chi-square test were used to compare differences in the data on basic characteristics and outcome measurements of the EMST and CTRL groups. The Mann–Whitney *U* test was also applied to evaluate the percent changes in the PE_{max}, AHI, ESS, and PSQI scores of the participants before and after training. Pearson correlation analysis was used to determine the correlations among the PE_{max}, AHI, ESS, and PSQI of the participants. The α value for statistical significance was set at 0.05. All results are presented as mean ± standard error of the mean.

3. Results

3.1. Participant characteristics

Initially, 29 OSA outpatients matched by AHI, sex, and BMI participated in this study and were assigned to either the EMST ($n = 15$) or CTRL ($n = 14$) group. However, during the intervention, two participants from each group could not complete the posttest assessment because of illness or other personal reasons. Therefore, 25 participants completed the intervention and data collection (21 men and 4 women; 13 and 12 in the EMST and CTRL groups, respectively). Among these participants, 14 participants were classified in the mild OSA subgroup ($5 \leq$ AHI < 15; EMST: $n = 7$ and CTRL: $n = 7$) and 11 participants were classified in the moderate OSA subgroup ($15 \leq$ AHI < 30; EMST: $n = 6$ and CTRL: $n = 5$). Age, height, weight, and BMI at baseline did not differ significantly between the two groups (pretest; Table 1). The basic sleep-quality-related characteristics of all participants are shown in Table 2. BMI, respiratory muscle strength, and sleep-quality-related parameters were not significantly different between the two groups before the treatment. Moreover, to ensure that the confounding effects from baseline AHI scores were nullified, we performed a correlational analysis between the change in AHI score and the baseline AHI score, and we observed no correlations between the two scores ($r = 0.091$; $P = 0.332$; $r^2 = 0.0083$).

Table 1
Basic Characteristics of Participants (n = 25).

Variable	Number (%)	EMST group	CTRL group	*P* value
	N(%)	N(%)	N(%)	
Total Number	25 (100)	13 (48.0)	12(52.0)	n.a
Height (cm)[a]		170.6 ± 2.0	170.5 ± 2.0	0.87
Weight (kg)[a]		72.6 ± 2.1	72.3 ± 3.2	0.94
BMI (kg/m^2)[a]		24.9 ± 0.5	24.7 ± 0.8	0.89
Gender[b]				
Male	21 (84.0)	11 (84.6)	10(83.3)	n.a
Female	4 (16.0)	2 (15.4)	2(16.7)	n.a
Age[a]		44.3 ± 2.9	48.0 ± 3.1	0.48
BMI (kg/m^2)[a]		24.9 ± 0.5	24.7 ± 0.8	0.89
AHI (times/h)[a]		16.5 ± 2.2	14.6 ± 1.5	0.68
ESS Score[a]		9.9 ± 1.1	9.8 ± 0.9	0.98
PSQI Sleep Quality[a]		7.4 ± 1.1	6.7 ± 0.8	0.85
PE_{max} (cmH2O)[a]		134.8 ± 10.3	108.6 ± 11.6	0.08

Data were expressed as Mean ± Standard Error ± (Mean S.E.).
[a] Mann-Whitney *U* test ($p < 0.05$).
[b] Pearson Chi-Square test ($p < 0.05$).

Table 2
Basic characteristics of participants at baseline (n = 25).

		EMST	CTRL	*P* value
Overall	BMI	24.9 ± 0.5	24.7 ± 0.8	0.89
(n = 25)	AHI	16.5 ± 2.2	14.6 ± 1.5	0.68
EMST n = 13 CTRL	PEmax	134.8 ± 10.4	108.6 ± 11.6	0.08
n = 12	PSQI	7.4 ± 1.1	6.7 ± 0.8	0.85
	ESS	9.8 ± 1.1	9.8 ± 0.9	0.98
Mild	BMI	24.9 ± 0.7	24.7 ± 0.9	0.88
(n = 14)	AHI	10.3 ± 0.8	10.7 ± 0.9	0.84
EMST n = 7 CTRL	PEmax	142.4 ± 14.7	119.9 ± 17.0	0.34
n = 7	PSQI	6.6 ± 1.7	5.6 ± 0.9	0.60
	ESS	8.1 ± 1.2	10.6 ± 1.3	0.20
Moderate	BMI	24.9 ± 0.9	24.8 ± 1.49	0.97
(n = 11)	AHI	23.6 ± 2.2	20.0 ± 1.1	0.20
EMST n = 6 CTRL	PEmax	125.8 ± 14.9	92.8 ± 13.1	0.14
n = 5	PSQI	8.3 ± 1.4	8.2 ± 1.4	0.95
	ESS	11.8 ± 1.8	8.8 ± 1.1	0.20

Mean ± SE; EMST: expiratory muscle strength training group; CTRL: control group; Mild: participants with mild OSA symptom ($5 \leq$ AHI < 15); Moderate: participants with moderate OSA symptom ($15 \leq$ AHI < 30); BMI: body mass index (kg/m^2); AHI: apnea-hypopnea index; PEmax: maximum static expiratory mouth pressure; PSQI: Pittsburgh sleep quality index; ESS: Epworth sleepiness scale.

3.2. Effect of EMST on AHI and PE_{max}

Fig. 1A illustrates the percent changes in AHI scores after treatment in both groups. After 5 weeks of intervention, the reduction in the AHI scores of the EMST group (−40% ± 6%) was significantly greater than that in the CTRL group (4% ± 6%; $P < 0.05$). Thereafter, the participants were sorted into either the mild OSA ($5 \leq$ AHI < 15) or moderate OSA ($15 \leq$ AHI < 30) subgroup depending on the severity of their OSA symptoms. We further analyzed the percent changes in the AHI scores of the participants with differences in the severity of OSA between the different EMST groups. In the moderate OSA subgroup, the percent change in the AHI score of the EMST group (−26% ± 9%) was significantly greater than that in the CTRL group (4% ± 4%; $P < 0.05$). In addition, in the mild OSA subgroup, the percent change in the AHI score of the EMST group (−51% ± 7%) was also significantly higher than that in the CTRL group (4% ± 9%; $P < 0.05$).

Fig. 1B displays the percent changes in the PE_{max} scores of the participants after the treatment. After 5 weeks of intervention, the improvement in PE_{max} was significantly higher in the EMST group (68% ± 12%) than that in the CTRL group (9% ± 5%; $P < 0.05$). Moreover, the PE_{max} percent change in the EMST group was significantly higher than that in the CTRL group among the participants with moderate OSA (EMST: 66% ± 17% vs. CTRL: 7% ± 9%; $P < 0.05$). Identical percent change trends in PE_{max} score were also observed in participants with mild OSA symptoms (EMST: 70% ± 18% vs. CTRL: 10% ± 7%; $P < 0.05$).

3.3. Effect of EMST on the parameters of sleep quality

Fig. 1C displays the percent changes in the sleep quality scores of the EMST and CTRL groups after the treatment. After 5 weeks of intervention, the sleep quality scores of the EMST group (−28% ± 5%) improved significantly more than did those of the CTRL group (10% ± 14%; $P < 0.05$). We then further analyzed the improvement in parameters of sleep quality between the two groups. EMST significantly decreased the PSQI scores for the moderate OSA subgroup ($P < 0.05$) but not for the mild OSA subgroup.

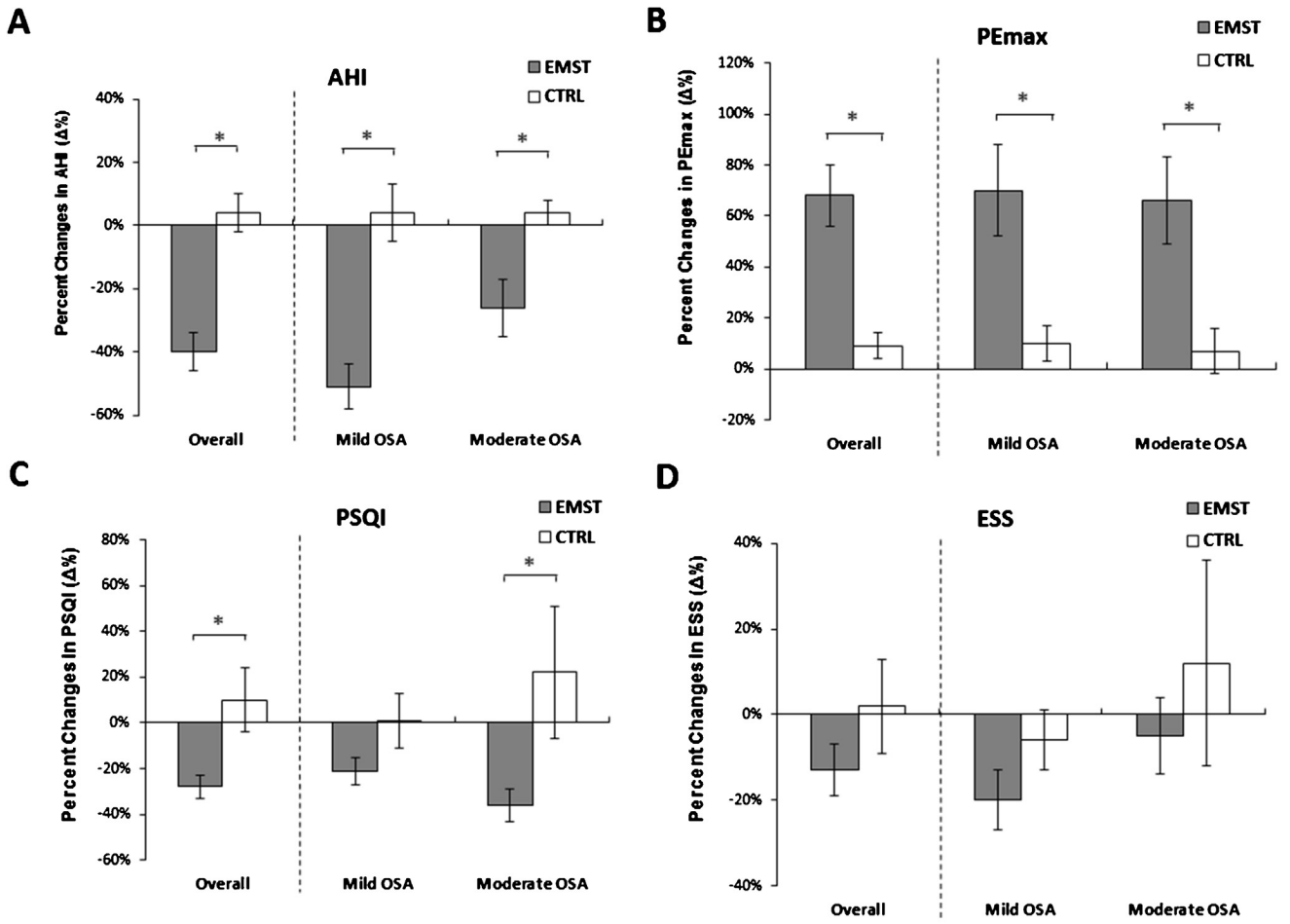

Fig. 1. Effect of respiratory muscle training on sleep apnea status (AHI score), expiratory muscle maximal strength (PE_{max} score), sleep quality (PSQI score), and subjective excessive daytime sleepiness (EDS). Mild OSA: $5 \leq$ AHI < 15; EMST: $n = 7$ and CTRL: $n = 7$; Moderate OSA: $15 \leq$ AHI < 30; EMST: $n = 6$ and CTRL: $n = 5$; EMST: respiratory muscle strength training group; CTRL: sham control treatment group. * denotes a significant difference between the EMST and CTRL groups.

Fig. 1D illustrates the subjective EDS of the participants in terms of ESS. Following the intervention, the percent changes in the ESS scores did not differ between the EMST and CTRL groups. Similarly, in the mild and moderate OSA subgroups, no differences in ESS scores were observed between the EMST and CTRL groups after the 5-week intervention.

3.4. Correlation between respiratory muscle strength and OSA severity

Fig. 2 illustrates the correlations between the percent changes of OSA severity index (AHI) and PE_{max} scores. The percent changes in the AHI and PE_{max} scores after the treatment exhibited a significant negative correlation ($r = -0.443$; $P = 0.013$). However, no significant correlations were observed between percent changes in AHI scores and those in ESS ($r = 0.143$; $P = 0.247$) and PSQI ($r = 0.290$; $P = 0.080$) scores.

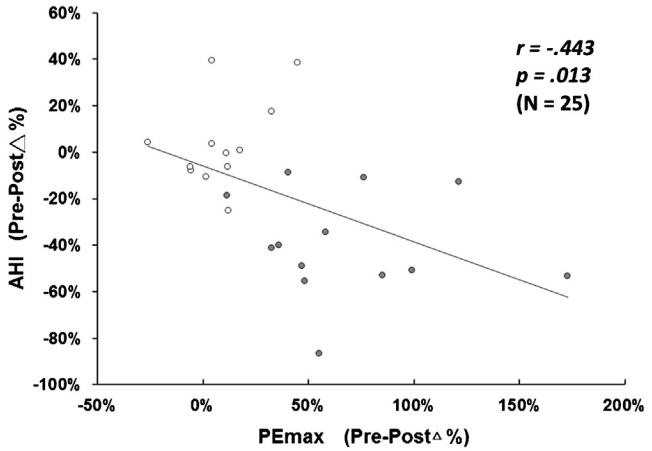

Fig. 2. Correlations between the percent changes in OSA severity index (AHI) and PE_{max} scores. Open circles denote the participants in the CTRL group ($n = 12$), and filled black circles denote the participants in the EMST group ($n = 13$).

4. Discussion

The primary findings of this study were that 5 weeks of respiratory muscle strength training improved sleep apnea, respiratory muscle strength, and sleep quality in participants with mild to moderate OSA symptoms. However, when we further analyzed the participants with different levels of OSA symptoms, we observed that the severity of participants' initial symptoms did not affect the outcomes of respiratory muscle strength training. In this study, we showed that the improvement in sleep quality after EMST was more evident in patients with moderate OSA than in those with mild OSA. Furthermore, the improvement in expiratory muscle strength and sleep apnea status exhibited a significant negative correlation after the 5-week training period, thus suggesting that respiratory muscle strength increased as the apnea index decreased.

After 5 weeks of respiratory muscle training, the AHI scores significantly decreased in the EMST group (EMST group: −40%; CTRL

group: +4%), indicating that the intervention facilitated a reduction in the severity of OSA from moderate to mild. Moreover, the intervention reduced the AHI scores of the participants with mild OSA by 51% and reduced their OSA severity to within the normal range (AHI < 5). Similarly, a previous study reported that a 16-week respiratory muscle training program using the didgeridoo significantly reduced the EDS condition by 37% and 48%, respectively, according to the ESS and AHI scores of participants with moderate OSA (Puhan et al., 2006). Notably, although the effects of EMST on reducing sleep apnea in our study are comparable to those reported by Puhan et al. (2006), the training period in our study was only 5 weeks compared with the previously reported 16-week period. The difference between our study and that by Puhan et al. is most likely due to the adjustable resistance mechanism of our EMST device, which enabled us to periodically manipulate the training load according to the participants' capacity throughout the 5-week intervention period. Taken together, these findings suggest that respiratory muscle strength training exerts a clear and rapid therapeutic benefit, attenuating sleep apnea symptoms in patients with mild or moderate OSA.

Because OSA is characterized by sleep-related upper airway dysfunction and repetitive airway obstruction during sleep, increasing the strength of the surrounding upper airway muscle groups can be expected to prevent airway collapse during sleep. Improvement in respiratory muscle strength can attenuate sleep apnea symptoms (i.e., decline in AHI score) (Kuehn and Moon, 1994; Wheeler et al., 2007). Wheeler et al. (2007) reported that the patterns of neuromuscular activation in the submental muscle were longer in the group trained using an expiratory device. These results imply that EMST training might ameliorate upper airway dysfunction, particularly in the submental muscle group. Furthermore, several previous studies using various training models, such as an EMST trainer, tongue exercise training with electrical nerve stimulation, or oropharyngeal exercise training, have demonstrated the benefits of upper airway muscle strength training for improving expiratory muscle strength (Anand et al., 2012) and attenuating sleep apnea symptoms (Guimaraes et al., 2009; Lequeux et al., 2005). Thus, the ameliorative effects of playing a wind instrument or using an EMST device on sleep apnea symptoms could be due to the gain of sufficient air pressure for supporting the nasopharynx, thereby increasing oropharyngeal or upper airway muscle tension (Burkhead et al., 2007). Together with our findings, the benefits of EMST on neuromuscular activation of the submental muscle groups may help explain the attenuation of OSA symptoms observed in our study.

The EMST significantly improved the PE_{max}, AHI, and sleep quality scores of all subcategory groups. Notably, the improvement in sleep quality scores after EMST was more evident in participants with moderate OSA than in those with mild OSA (Fig. 1C). The variation in improvement between participants with mild and moderate OSA was possibly because the participants with more severe OSA (moderate group) had poorer sleep quality scores; thus, they had higher potential for improvement than did participants with mild OSA. Improvement after treatment may have been absent in those with mild OSA because of the floor effect. Hence, sleep quality improvement is less likely in patients with minor sleep difficulties. The data presented in this study suggests that the severity of a patient's initial OSA symptoms may affect sleep quality outcomes following respiratory muscle strength training. These findings are consistent with those of a previous study by Puhan et al., which reported that respiratory training using a wind instrument significantly improved respiratory muscle strength and sleep apnea, but not sleep quality, in patients with OSA (Puhan et al., 2006). One possibility for the absence of improvement in sleep quality might be that the PSQI assessment was based on the evaluation of the participants' sleep status within a month. Because the EMST duration in this study was only 5 weeks, possible improvements in sleep quality might have been masked in patients with mild OSA symptoms. In other words, the absence of improved sleep quality could be attributed to the short duration of training period as well as participant selection.

In addition to sleep apnea and poor sleep quality, EDS has been recognized as a primary clinical impairment for patients with OSA (Chen et al., 2011; Sun et al., 2012). Previous studies have reported that oropharyngeal exercise training (Guimaraes et al., 2009) and didgeridoo practice (Puhan et al., 2006) effectively alleviate EDS after 12–16 weeks of intervention. Furthermore, several studies have demonstrated the benefits of CPAP for improving both objective and subjective sleepiness (Bednarek et al., 1999; White et al., 2002). In this study, we report that a 5-week EMST intervention treatment was inadequate for producing considerable EDS reduction in patients with mild to moderate OSA. Compared with respiratory muscle training, CPAP, an antisnoring device, and upper airway muscle training appear to be more effective in alleviating EDS. However, in light of the positive effect of our treatment on sleep apnea and sleep quality, future studies with longer intervention periods are warranted to identify the effects of EMST training on EDS.

Our data indicated that the improvement in PE_{max} scores were negatively correlated with AHI scores (i.e., the decrease in sleep apnea severity) in participants with mild to moderate OSA ($r = -0.443$; $P = 0.013$). Because EMST produced improved PE_{max} scores, our findings suggest that an increase in respiratory muscle strength because of EMST might account for the clear reduction in sleep apnea symptoms. To the best of our knowledge, this study is the first to report an inverse relationship between PE_{max} and AHI scores of participants with OSA. This finding is significant because it suggests that the EMST-enhanced expiratory muscle strength might contribute to the amelioration of OSA symptoms. Our findings also provide additional evidence that EMST effectively alleviates OSA symptoms and improves sleep quality simply by strengthening the upper airway muscles.

Our study had some limitations. For example, we did not measure inspiratory muscle strength in this study. Therefore, we cannot ignore the possibility that our EMST training potentially reduces OSA symptoms by increasing inspiratory muscle strength. In addition, subgrouping the participants based on OSA severity resulted in a smaller sample size (mild OSA: seven participants per treatment; moderate OSA: five or six participants per treatment). However, EMST clearly reduced sleep apnea symptoms in both subgroups. This effect indicates the potential therapeutic benefits of EMST in clinical settings.

5. Conclusions

In summary, this investigation demonstrated that 5 weeks of respiratory muscle training using an EMST trainer improved sleep apnea and the PE_{max} scores of individuals with mild to moderate OSA. Although EMST significantly improved the PE_{max} and AHI scores of all subgroups, patients with moderate OSA exhibited more improvement in sleep quality after EMST than did those with mild OSA. However, the 5-week EMST intervention had no alleviating effect on EDS. Furthermore, the improvements in PE_{max} scores were inversely correlated with the AHI scores of participants with mild to moderate OSA, suggesting that enhancing respiratory muscle strength using EMST is, at least partially, an effective modality to reduce sleep apnea.

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Conflicts of interest

This work was not an industry-supported study. The authors report no conflicts of interest.

Ethical approval and informed consent

The present study was approved by the IRB of Taipei Medical University, and the training program and all tests followed the guidelines provided by the IRB. This study was conducted in accordance with the 1964 Helsinki Declaration and its amendments. All the experimental procedures were performed only after written informed consent was provided by the eligible participants.

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