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RESISTIVE RESPIRATORY TRAINING IMPROVES BLOOD PRESSURE REGULATION IN INDIVIDUALS WITH CHRONIC SPINAL CORD INJURY

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Abstract

Objective—To investigate the effects of resistive Respiratory Motor Training (RMT) on pulmonary function and orthostatic stress-mediated cardiovascular and autonomic responses in individuals with chronic Spinal Cord Injury (SCI).

Design—Before-after intervention case-controlled clinical study.

Participants—Individuals with chronic C₃–T₂ SCI diagnosed with orthostatic hypotension (OH) (n=11) and healthy, non-injured (NI) controls (n=10).

Intervention—21 ± 2 (mean ± SD) sessions of resistive inspiratory-expiratory RMT performed 5 days a week during a one-month period.

Main Outcome Measures—Standard Pulmonary Function Test (PFT): Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV₁), Maximal Inspiratory Pressure (PI_{max}), and Maximal Expiratory Pressure (PE_{max}) and beat-to-beat arterial blood pressure (BP), heart rate (HR), and respiratory rate during orthostatic sit-up stress test acquired before and after RMT program.

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Results—Completion of RMT intervention abolished OH in 7 out of 11 individuals. FVC, low-frequency component of power spectral density (LF PSD) of BP and HR oscillations, baroreflex effectiveness and cross correlations between BP, HR, and respiratory rate during orthostatic challenge were significantly improved, approaching levels observed in NI individuals. These findings indicate increased sympathetic activation and baroreflex effectiveness in association with improved respiratory-cardiovascular interactions in response to the sudden decrease in BP.

Conclusion—Resistive respiratory training increases respiratory capacity and improves orthostatically mediated respiratory, cardiovascular, and autonomic responses suggesting that this intervention can be an efficacious therapy for managing OH after SCI.

Keywords

Spinal cord injury; Respiratory training; Respiratory function; Autonomic regulation; Blood pressure; Orthostatic hypotension

INTRODUCTION

Approximately 60% of the people with chronic spinal cord injury (SCI) exhibit symptoms of orthostatic hypotension (OH) ^{1,2} which is associated with an inability to participate in activities of daily life ^{3,4} and rehabilitation ^{5,6} with increased risk of stroke, ⁷ cognitive dysfunction, and mood disturbances. ^{8,9}

Respiration affects hemodynamics by supporting cardiac output, ^{10–12} and by participating in autonomic regulation of heart rate (HR) and blood pressure (BP) via baroreflex modulations. ^{13–16} These respiratory-cardiovascular interactions ^{17,18} are compromised after SCI. ^{1,11,19–22} Currently, there is no standardized management strategy for BP dysregulation after SCI. ^{5,23,24} Respiratory training is a widely used technique in clinical practice, but the consistent benefits of this intervention are somewhat contentious due to poor understanding of the underlying therapeutic mechanisms. ^{25–27} The objective of this study was to investigate the effects of resistive inspiratory-expiratory training, termed here as a Respiratory Motor Training (RMT), on pulmonary function and orthostatic stress-mediated cardiovascular and autonomic responses. It was hypothesized that RMT improves baroreflex responses and respiratory-cardiovascular interactions in patients with SCI-induced OH. This technique has never been evaluated for its ability to improve BP regulation in individuals with chronic SCI.

METHODS

Material

Informed consent was obtained as approved by the Institutional Review Board for Human Research according to the inclusion criteria: at least 18 years of age; a minimum of 6 months since SCI; no ventilatory dependence; and orthostatic hypotension defined as a decrease in arterial systolic blood pressure of at least 20 mm Hg or a reduction in diastolic blood pressure at least 10 mm Hg or more upon changing from a supine position to upright posture. ² Individuals with painful musculoskeletal dysfunction; unhealed fracture; contractures; clinically significant depression or ongoing drug abuse; cardiovascular,

respiratory, bladder, or renal diseases unrelated to SCI; HIV/AIDS related illness; anemia, hypovolemia, pregnancy, endocrine and neurological diseases were excluded from this study.

Clinical assessment

Eleven participants with SCI and ten physically (age, sex, height, and weight) matched non-injured (NI) controls participated in this study. The neurological level and completeness of the spinal cord lesion were determined using the American Spinal Cord Injury Association Impairment Scale (AIS).²⁸ Seven participants were classified as having motor-complete SCI and four participants were diagnosed with motor-incomplete SCI ranging from C₃ to T₂ (Table 1).

Pulmonary function test (PFT)

Standard spirometrical measurements [forced vital capacity (FVC), forced expiratory volume in one second (FEV₁), maximum inspiratory airway pressure (PI_{max}), maximum expiratory airway pressure (PE_{max})]^{13,29,30} were assessed as described previously.³¹

Orthostatic stress test

A sit-up test with continuous recordings of BP, HR, and respiratory rate was used to diagnose OH and for beat-to-beat data acquisition at 1000 Hz and analyzed using Matlab software (The MathWorks, Natick, MA).^{32,33} Systolic and diastolic BP (SBP and DBP) were acquired from a finger cuff using Portapres-2 (Finapres Medical System B.A., Netherlands) and ML880 PowerLab 16/30 (ADInstruments, Colorado Springs, CO) systems. Brachial BP measurements using a Dinamap V100 (GE Medical Systems, Milwaukee, WI) were acquired at the beginning and end of each position phase to calibrate the beat-to-beat BP values. The ML132 three-lead II electrodes (ADInstruments) and Inductotrace System bands (Ambulatory Monitoring Inc., Ardsley, NY) were used to record electrocardiogram and to assess the respiratory rate.³²

Hemodynamic variables (mean $\pm$ SD) were calculated for 5 minutes (min) intervals of 15-min supine position, 1 min intervals within first 3 min of sitting, and 3 min intervals from 3 to 15 min of sitting position. Spectral power of HR, BP, and respiratory rate were calculated for 5 min intervals in both positions. Each interval was linearly detrended, and spectral power was estimated using Welch's averaged periodogram method (500-point windows with 50% overlapping segments). Mean spectral power was calculated for low-frequency (LF, 0.04 – 0.15 Hz) and high-frequency (HF, 0.15 – 0.4 Hz) regions using trapezoidal integration over the specified frequency range.^{34–36} Baroreflex effectiveness index (BEI, %) and baroreflex sensitivity (BS, ms/mm Hg) were determined using standard approach.³⁵ The cross correlation coefficients between SBP and HR oscillations in LF / HF regions and between HR and respiratory rate oscillations in HF region were calculated as described previously.³²

Respiratory Motor Training (RMT)

Research participants were seated in their personal wheelchair during each training session with an approximately 45° head-up tilt. A threshold positive expiratory pressure device and inspiratory muscle trainer (Respironics Inc., Cedar Grove, NJ) were assembled using a

three-way valve system (Airlife 001504, Allegiance Healthcare Corp., McGaw Park, IL) with flanged mouthpiece. The participants were performing 6 work sets, 5 minutes in duration, separated by rest intervals lasting 3 minutes. Participants were trained 5 days/week, for 45 min/day during 1 month. The training was initiated at intensity equal to 20% of their individual PI_{max} and PE_{max} with progressive increases as tolerated up to 40% of these values at the end of the training program.^{37–39} No adverse events related to significant changes in BP or symptoms of intolerance were observed during training sessions.

Statistical analysis

The study sample size was estimated using “G Power” tool⁴⁰ with the level of significance (α) and statistical power ($1-\beta$) set at 0.05 and 0.8, respectively. The effect size was estimated with Cohen’s d calculations for paired samples. Comparisons between PFT unidentified data sets obtained before and after the RMT program were made using the paired two-tailed t-test. Unidentified hemodynamic data were fit with a linear mixed effects model and reported as mean $\pm$ SD. Research group, position (supine or seated), and an interaction term were the fixed factors in the model. A random intercept was included to account for the added covariation of repeated measurements. Comparisons between groups and positions were derived from the mixed model fit through standard F-tests of linear contrasts of the model coefficients. All hypothesis tests were conducted at the $p < 0.05$ with the level of significance (α) being set at 0.05. All analyses were conducted using the open-source R software 3.0.2 package (R Development Core Team, 2013).

RESULTS

Pulmonary function test (PFT)

The PFT outcomes were increased post RMT as compared to the pre-training levels, reaching significance in FVC values ($p < 0.05$) (Table 2).

Orthostatic stress test

Before training, OH was diagnosed in all 11 SCI individuals: in 7 of them the “orthostatic” drop in BP was detected within 3 min and in the other 4, within 10 min of assuming the sitting position. After RMT, this OH drop was seen in only 4 participants (in 2 individuals within 3 min and in the other 2 within 10 min of onset of the sitting phase). One individual (A43), before RMT, became presyncopal after only 5 minutes of the orthostatic challenge which required protocol cessation; after RMT, however, he was able to complete the test. Figure 1 shows BP responses in one individual (A38) before and after RMT while Table 3 summarizes the effects of RMT on BP across all subjects.

In the SCI group, before RMT, both SBP and DBP variables decreased continuously within the first 6 min of the seated position and stabilized at lowered levels from 6 to 15 min. In contrast, upon completion of the RMT, both pressures decreased for first 3 min, and then were maintained stable from 3 to 15 min in the seated position (Fig. 2A, 2B). Compared to the pre-training baseline, these supine-to-seated decreases in BP were significantly smaller after RMT (Fig. 3A, 3B). The HR responses among the groups in both positions were similar: stable during the supine position and significantly increased in seated position from

60 ± 10 BPM to 67 ± 8 ($p = .0006$) in NI, from 58 ± 6 to 73 ± 16 ($p = .0002$) in SCI before RMT and 58 ± 8 to 77 ± 15 ($p = .006$) in SCI after RMT.

Prior to RMT, baroreflex effectiveness remained significantly decreased compared to the supine baseline throughout the entire 15-min seating period. Conversely, this significant decrease was only seen during initial (0–5 minute) period when assessed after the RMT (Fig. 4A). Baroreflex sensitivity in the seated position was significantly lower than baseline in all participants from NI and SCI groups before and after RMT (Fig. 4B).

Group averages of the LF component of HR power spectral density (PSD) during supine baseline were not different across the groups (Fig. 5A). Compared to baseline, this index significantly increased during 10–15 min of seated position in the NI group and significantly decreased during 5–15 min in SCI individuals when assessed before RMT. This significant drop in the upright position was not seen after RMT (Fig. 5A). Group mean of LF PSD of SBP (Fig. 5B) and DBP (Fig. 5C) at rest tended to be lower in the SCI group compared to the NI both before and after RMT, though neither difference reached significance. Both indexes significantly increased in response to the orthostatic stress in NI individuals. In individuals with SCI, compared to the baseline, LF PSD of SBP significantly increased during 0–5 min of seated position both as recorded before and after the RMT. This increase, however, was elevated vs. the nadir during the remainder of the seated period only after the RMT (Fig. 5B). LF PSD of DBP in SCI group did not show any significant change in seated phase when recorded both before and after the RMT (Fig. 5C).

In supine position, the high-frequency component of HR (HF PSD) was not significantly different in NI (282 ± 194) and SCI (202 ± 119) groups. These values were slightly decreased to 263 ± 204 in NI individuals. In contrast, there was significant decrease to 49 ± 45 in the SCI group during the last 5 minute of sitting position. After the RMT, this pattern was not changed showing a significant decrease from 183 ± 99 in supine to 60 ± 49 during the last 5 minutes in seated position.

Both the LF and HF components of the cross correlation between BP and HR were examined. There were no significant differences in the supine condition between any group in the mean magnitude of the negative component (e.g., a decrease in BP associated with an increase in HR and vice versa) of the LF cross correlation. This measure was increased significantly at 10–15 min of seated position in the NI group. In the SCI group, this index decreased significantly within 5 min of sitting position before and after RMT. These values remained significantly lower than baseline in participants with SCI before RMT, whereas after RMT it increased back toward its baseline during the remainder of the sitting duration. Figure 6, where a red coloration indicates a strong positive cross correlation and blue a strong negative, provides a visual summary of this dynamic process within the low frequency range. Cross correlation coefficients between HR and SBP in HF region were similar in all groups during last 5 min of supine and sitting positions as well as before and after RMT in SCI group (NI: -0.70 ± 0.1 vs. -0.76 ± 0.1 ; SCI: -0.72 ± 0.1 vs. -0.67 ± 0.2 ; and -0.71 ± 0.1 vs. -0.68 ± 0.2).

Group mean respiratory rate oscillations (Hz) were similar among NI and SCI individuals: in NI group: 0.23 ± 0.08 and 0.23 ± 0.05 ; in SCI group: 0.22 ± 0.06 and 0.25 ± 0.05 before training and 0.25 ± 0.07 and 0.26 ± 0.08 after training (in supine and in sitting positions, respectively). The NI group showed positive cross correlation with almost zero time delay between respiratory oscillations and HR fluctuations throughout the test with increased intensity from 5 to 15 min in seated position. In the SCI group, this correlation at rest was negative with no post-RMT change; but, after RMT, in the seated position it was positive with significant improvement toward the normative values observed in NI individuals (Fig. 7 A and B).

DISCUSSION

In the present study, it is reported that resistive RMT reduces the severity of OH in individuals with chronic SCI. The findings indicate that this effect is associated with significantly increased respiratory capacity, improved baroreflex-mediated autonomic activation, and better synchronized cardiorespiratory coupling interactions in response to the assuming an upright posture.

The most clinically relevant finding is that RMT resulted in a remarkable improvement in the ability to maintain physiological levels of the BP in response to the orthostatic stress. The ability to assume an upright posture without an orthostatic drop in BP was acquired in 64% participants with SCI. These finding seems particularly remarkable given that these individuals had not established an effective orthostatic response in the average of 53 ± 72 months after SCI, indicating that the RMT can be used to shorten the time between injury and the initial steps leading to eventual resumption of a more stable lifestyle.

The results of this study have indicated increase in post-RMT respiratory performance associated with improved respiratory motor function (Table 2). This finding is consistent with reports of others suggesting that respiratory exercise is effective for improving respiratory function in individuals with SCI during acute⁴¹ and chronic SCI,^{42–47} as well as in patients with other disorders.^{48–50} However, the approach of reciprocal inspiratory-expiratory training with adjustable load has never been previously used in SCI population. A term “Respiratory Motor Training” was chosen in lieu of the more commonly utilized “Respiratory Muscle Training” in order to reflect the intent to affect not only the respiratory muscles, but to intervene the respiratory neuro-muscular motor control and to challenge the respiratory-cardiovascular coupling interactions during cycles of intrathoracic pressure fluctuations that is accompanied by BP oscillations. These oscillations are thought to potentially “train” the baroreflex and autonomic networks to respond to fluctuations in BP in a more normal physiological manner. Furthermore, it has been shown that stiffening of the central arteries is responsible for the majority of cardiovagal baroreflex decline after high level SCI.⁵¹ By imposing healthy sheer stress on arteries with embedded baroreceptors, the oscillations in BP during the RMT session may improve the arterial stiffening and increase baroreceptors’ elasticity.

The reported data indicate that the diagnosis of OH using sit-up orthostatic stress test^{33,52} in individuals with SCI requires up to a 15-min long observation period in the upright seated

position (Table 3). Documented in 54% of non-SCI population, this delayed response was associated with milder abnormalities of sympathetic adrenergic function that progressed over time.^{53–55} After SCI, this phenomenon may be aggravated by functional deconditioning and neurodegeneration. Interestingly, the results of this study indicate that all post-RMT improvements related to the cardiovascular and autonomic function were attributable to the responses that occurred predominately during second half of the seated phase of the orthostatic stress test (Table 3 and Figs. 1–7).

A number of observations jointly point towards the conclusion that RMT improved baroreflex responses via augmented autonomic responses. First, BEI and BE were improved after RMT indicating better coordination between beat-to-beat BP and HR changes mediated by the cardiovagal reflex (Fig. 4A and B). Since the cardiovagal control is intact after SCI,^{56–58} this improvement can be associated with decreased orthostatically mediated vagal withdrawal and better cardiopulmonary coupling that heavily depends on parasympathetic control.⁵⁹ Second, LF HR spectral power, primarily indicating the cardiac sympathetic activity, as well as LF arterial BP power, a marker of efficacious sympathetic vasomotor outflow, significantly decreased after assuming the upright posture in the participants before RMT, but were maintained after training (Fig. 5). The most conservative interpretation of the increase in the negative LF cross correlation following RMT (Fig. 6) is that the training somehow helped re-enable that component of the baroreflex dependent upon the sympathetic innervation. The RMT may well have exerted its influence via functional improvement in both branches of the autonomic nervous system. We speculate that such enhancement may be via the improved balance between intact vagal control and injured sympathetic pathways at the fringe of the lesion that are not functioning for a time post-injury, but which may ultimately regain at least minimal function.⁶⁰

Inspiration and expiration have mechanical sequelae with significant hydraulic effects.^{12,61–64} Even during a transition to an upright posture an increased intensity of respiratory movements secondary to RMT might be expected to defend, or even increase, the pressure gradient for venous return and, thereby, increasing the cardiac output.¹¹ It has been shown that patients with OH⁶⁵ and individuals with tetraplegia subjects can prevent the orthostatic events by voluntarily increased breathing effort during transferring from supine to the seated position.⁶⁶ In the present study, RMT significantly improved synchronization between HR and respiration (Fig. 7) which may indicate more effective respiratory pumping to fight the orthostatic decrease in BP.

It has been demonstrated that respiratory training protocols can modulate baroreflex responses through the increase in vagal activity and reduction in sympathetic activity in hypertensive individuals^{67,68} and rats.⁶⁹ In the present study, it was demonstrated that the RMT is associated with significantly increased sympathetic activation in response to sudden decrease in BP. Obviously, these findings are attributable to the fundamental differences in pathophysiological nature of the autonomic and BP abnormalities observed in SCI, hypertension, and heart failure. However, the fact that similar therapeutic approach to manage these very different diseases can produce the specifically desirable effects is intriguing and requires further investigation.

Limitations of the study

We did not assess the severity of injury to the spinal autonomic pathways that could affect the outcomes of RMT effectiveness.^{33,70} The study was also limited by group heterogeneity and relatively small sample size. It is always challenging to form demographically and clinically homogeneous groups from a highly diverse but limited SCI population. In addition, it was not feasible to elucidate the impact of combinatory effects of neurological, pulmonary and cardiovascular factors.

CONCLUSION

The results of this study indicate that RMT improves pulmonary function and orthostatically mediated respiratory, cardiovascular, and autonomic responses and suggest that this intervention can be an efficacious therapy for managing OH after SCI. These findings provide some insight into the mechanisms of these benefits, which can be associated with awakened autonomic networks, harnessing respiratory pump, and improved cardiovascular baroreflex function. These results highlight the need for future investigations into the links between pulmonary and cardiovascular interactions.

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List of abbreviations

AIS	Association impairment scale
BEI	Baroreflex effectiveness index
BP	Blood pressure
BS	Baroreflex sensitivity
FEV₁	Forced expiratory volume in one second
FVC	Forced vital capacity
HF	High-frequency
HR	Heart rate
LF	Low-frequency
OH	Orthostatic hypotension
PE_{max}	Maximal expiratory pressure
PFT	Pulmonary function test
PI_{max}	Maximal inspiratory pressure

PSD	Power spectral density
RMT	Respiratory motor training
SCI	Spinal cord injury

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Figure 1.

Dynamics (mean $\pm$ SD/min) of systolic and diastolic blood pressure (SBP and DBP, black) and heart rate (HR, gray) during sit-up orthostatic stress test in individual with C₄ AIS-A SCI (A38) before (A) and after (B) Respiratory Motor Training (RMT). Note that considerable drop in SBP (–20 mm Hg) and DBP (–11 mm Hg) characterizing orthostatic hypotension before RMT is not seen after RMT. Note also stability of the blood pressure and HR response achieved after RMT.

Figure 2.

Dynamics (mean $\pm$ SD) of (A) systolic and (B) diastolic blood pressure (SBP and DBP) during orthostatic stress test in non-injured (NI) and SCI group before and after respiratory motor training (RMT). Note significant ($p < .05$) increase in both SBP and DBP in NI individuals (*) and significantly decreased SBP and DBP in SCI individuals in upright position compared to the values obtained in supine position before training ($\dagger$) and SBP after RMT ($\bullet$). Note also positive dynamics in both SBP and DBP in participants with SCI after RMT and no significant difference compared to supine baseline in DBP after training.

Figure 3.

Change (mean $\pm$ SD) in (A) systolic and (B) diastolic blood pressure (SBP and DBP) during orthostatic stress test in seated position from supine baseline in SCI group before and after respiratory motor training (RMT). Note that RMT significantly ($p < .05$) mitigated the fall in both SBP and DBP observed before the RMT (•).

Figure 4.

Baroreflex effectiveness index (BEI) (**A**) and baroreflex sensitivity (BS) (**B**) during orthostatic stress test in non-injured (NI) and SCI group before and after respiratory motor training (RMT) (mean $\pm$ SD/5 min). Note a significant ($p < .05$) supine-to-seated decrease in BEI in SCI individuals before training ($\dagger$) and that this decrease was not significant during the late seated phase after RMT ($\bullet$). Note also the positive trend toward NI control curve that is observed after RMT in both BEI and BS in seated position.

Figure 5.

Low-frequency component of power spectral density (LF PSD) of heart rate (HR) (A); systolic (B) and diastolic blood pressure (SBP and DBP) oscillations (C) during orthostatic stress test in non-injured group (NI) and in SCI group before and after respiratory motor training (RMT) (mean $\pm$ SD/5 min). Note that compared to the supine baseline, in contrast to SCI individuals, all outcomes in NI individuals were significantly increased ($p < .05$) in seated position (*). Note also significantly decreased LF PSD of HR in SCI individuals in seated position before training (†) but no such significant difference in this outcome after training. In contrast to pre-training levels, LF PSD of SBP was significantly higher throughout seated period after RMT (•).

Figure 6.

Distribution (A) and magnitude (B) of the low-frequency (0.04–0.15 Hz) negative cross correlation between heart rate (HR) and systolic blood pressure (SBP) during orthostatic stress test in non-injured group (NI) and in SCI group before and after respiratory motor training (RMT) (mean $\pm$ SD/5 min). In the panel A, the lag is given on the vertical axes where negative lag refer to changes in SBP leading HR and positive lags refer to SBP lagging HR. The magnitude of the correlation is indicated by the color density according the scale presented in vertical bars on right. The strong blue band around 2.5 s in NI represents responses characteristic of baroreflex function. Note that compared to supine baseline, in contrast to SCI individuals, the negative magnitude were significantly increased ($p < .05$) in seated position in NI individuals (*). Note also its significant decrease in SCI individuals in seated position before training (†) and no significant difference in this outcome after training (•) associated with positive change in distribution.

Figure 7.

Distribution (A) and magnitude (B) of the high-frequency (0.15–0.4 Hz) negative cross correlation between heart rate (HR) and respiratory chest kinematics during orthostatic stress test in non-injured group (NI) and in SCI group before and after respiratory motor training (RMT) (mean $\pm$ SD/5 min). The lag is given on the vertical axes where negative lag refer to respiratory rate leading HR and positive lag refers to respiratory rate lagging the HR. The magnitude of the correlation is indicated by the color density according the scale presented in vertical bars on right. The red or blue bands around 0 sec represent positive (red) or negative (blue) correlation between HR and chest movements (positive correlation represents increase in HR during inspiration and decrease in HR during expiration). Note significantly ($p < .05$) improved the post-RMT correlation during orthostatic stress (seated position) compared to the pre-RMT levels (*).

Table 1

Characteristics of spinal cord injured (SCI) and non-injured (NI) participants.

Subjects		Age (years)	Sex	Height (cm)	Weight (kg)	Level of SCI	AIS category	Time after SCI (mo)
SCI (n = 11)	B06	41	F	170	56	C4	B	72
	A38	37	F	175	51	C4	A	252
	A43	30	M	188	95	T2	A	11
	B11	23	M	173	84	C5	B	96
	B13	31	M	180	97	C7	C	36
	B17	42	M	191	99	C5	B	15
	C30	18	F	168	42	C4	C	12
	B19	39	M	188	82	C6	B	7
	C26	33	M	183	75	C7	C	6
	A58	40	M	178	104	C3	A	22
	C34	20	M	193	64	C4	C	55
	Mean ± SD		32 ± 9	3 F & 8 M	181 ± 8	77 ± 21	N/A	N/A
NI (n = 10)		33 ± 10	2 F & 8 M	173 ± 10	75 ± 12	N/A	N/A	N/A

Table 2

Summary of Pulmonary Function Testing (PFT) values obtained before and after the Respiratory Motor Training (RMT).

Subjects n = 11	PFT							
	FVC (% predicted)		FEV ₁ (% predicted)		Pimax (cm H ₂ O)		PE _{max} (cm H ₂ O)	
	Before RMT	After RMT	Before RMT	After RMT	Before RMT	After RMT	Before RMT	After RMT
B06 (C4B)	46	57	38	40	-32	-41	25	26
A38 (C4A)	51	44	47	49	-20	-25	23	28
A43 (T2A)	52	72	55	67	-110	-127	56	56
B11 (C5B)	47	56	34	38	-93	-114	64	69
B13 (C7C)	77	67	74	66	-103	-101	64	50
B17 (C5B)	70	74	58	68	-61	-60	30	32
C30 (C4C)	41	69	45	46	-46	-46	27	26
B19 (C6B)	70	81	66	77	-54	-52	35	47
C26 (C7C)	60	76	67	76	-101	-103	70	77
A58 (C4A)	65	60	53	55	-20	-79	21	29
C34 (C7C)	37	40	29	28	-40	-42	34	41
Mean ± SD	56 ± 13	63 ± 13 *	51 ± 14	55 ± 16	-62 ± 34	-72 ± 35	41 ± 19	44 ± 18

Note that FVC was significantly increased after the RMT (*p < .05).

Table 3

Supine-to-seated change in systolic and diastolic blood pressure (SBP / DBP) during orthostatic stress test. Note that after the RMT, orthostatic hypotension (dark gray) was not seen (light gray) in seven out of eleven participants, all of whom were initially diagnosed with this condition.

Subjects	Seated position from 0 to 3 min (mm Hg)		Seated position from 3 to 10 min (mm Hg)	
	Before RMT	After RMT	Before RMT	After RMT
B06 (C4B)	-24 / -9	-36 / -26	-39 / -17	-40 / -28
A38 (C4A)	+10 / +7	+5 / +1	-20 / -11	-5 / -5
A43 (T2A)	-20 / -4	-17 / -1	-20 / -4 *	-14 / +4
B11 (C5B)	-26 / -9	-8 / +14	-31 / -4	-11 / +12
B13 (C7C)	-16 / -5	-1 / +12	-34 / -11	-15 / +5
B17 (C5B)	-31 / -17	-3 / 0	-46 / -25	-17 / -9
C30 (C4C)	-29 / -11	-11 / +12	-25 / -8	-19 / -1
B19 (C6B)	-21 / +4	-3 / +1	-29 / +1	-12 / -6
C26 (C7C)	-8 / -5	-10 / +6	-36 / -19	-34 / -13
A58 (C3A)	-2 / +5	-8 / -3	-27 / -4	-34 / -13
C34 (C4C)	-23 / -19	-24 / -24	-36 / -25	-36 / -27

* indicate that the participant was not able to complete the seated phase.