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Research Article

Impact of respiratory muscle training on muscle strength, pulmonary function, symptoms, and quality of life in COPD

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Abstract

Introduction

Chronic obstructive pulmonary disease (COPD) is characterized by respiratory muscle weakness, hyperinflation, and systemic inflammation, leading to impaired pulmonary function and quality of life. Respiratory muscle training (RMT) may strengthen the inspiratory and expiratory muscles, improve pulmonary function, reduce dyspnea, and enhance functional outcomes. This study assessed the impact of concurrent RMT on respiratory muscle strength, spirometry, dyspnea, and quality of life in patients with COPD.

Materials and Methods

This was a single-cohort pre/post-intervention study initially recruiting 43 patients with COPD to participate in an 8-week RMT program using a threshold pressure device. Both inspiratory and expiratory training were performed using a PowerLung device with adjustable resistance. Training consisted of three sets of ten breaths twice daily for each mode, and participants were instructed to increase resistance incrementally when the load became easy. Assessments included spirometry, maximum inspiratory pressure (MIP), maximum expiratory pressure (MEP), COPD Assessment Test (CAT), Medical Research Council (MRC) Breathlessness Scale, and Airways Questionnaire 20 (AQ20). Data were collected at baseline and post-intervention and analyzed using paired *t*-tests and Wilcoxon signed-rank tests, stratified by GOLD category.

Results

Twenty-seven participants completed the study. Statistically significant improvements were observed in MIP (mean increase 14.1 cm H_2O, $p < .001$), MEP (mean increase 20.1 cm H_2O, $p < .001$), CAT (mean decrease 2.92, $p = .020$), and AQ20 (mean decrease 1.67, $p = .005$). FEV_1 improved modestly but did not reach statistical significance (mean increase 0.0367 L, $p = .064$). The GOLD distribution included eight participants in GOLD 2, 12 in GOLD 3, and seven in GOLD 4. Improvements in MIP and MEP were statistically significant within all GOLD categories. A clinically meaningful increase in FEV_1 ($\geqslant$ 60 mL) was observed in participants in GOLD 3 and 4 stages, though not statistically significant. Correlations between muscle strength improvements and symptom scores were moderate to strong.

Discussion

Concurrent RMT improves respiratory muscle strength and quality of life in patients with COPD, with the greatest benefits observed in advanced disease stages. Enhanced respiratory muscle efficiency may reduce dyspnea and promote exercise tolerance.

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Conclusion

RMT is a promising intervention for COPD management that offers improved respiratory muscle strength and quality of life. Future studies should explore the long-term effects and optimize protocols for broader implementation.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) causes diverse anatomical changes in the lungs. Specifically, it results in airway and alveolar defects obstructing airflow, hyperinflation that mechanically disadvantages the diaphragm, and skeletal muscle weakness.[1] Combining this with circulating inflammatory mediators, patients with chronic lung disease often experience skeletal muscle wasting and cachexia.[1] The detrimental effects of hyperinflation, specifically diaphragmatic shortening with resulting mechanical disadvantage, result in increased respiratory muscle work, such as elevated work of breathing observed in patients with rib abnormalities and scoliosis.[2] This leads to dyspnea, which can hinder exercise and daily activities.

Patients with advanced COPD exhibit myopathic changes, characterized by altered muscle fibre composition in both the diaphragm and other skeletal muscle groups.[3] Additionally, non-diaphragmatic muscles in these patients undergo a transition from oxidative Type I fibres to anaerobic glycolytic Type IIB fibres.[3] Furthermore, there is a decrease in muscle capillary density measured as capillaries/mm^2 in the skeletal muscle of patients with COPD.[3] Disuse has been postulated to be the mechanism for fiber changes, but evidence also links these changes to the inflammation and oxidative stress associated with COPD.[3,4] Conversely, in the diaphragm of COPD patients, the percentage of Type I fibres is increased, likely as an adaptation to prevent muscle fatigue.[3] Skinned muscle fibres from the diaphragm of COPD patients show a reduction in myosin content compared to diaphragm muscle fibres from non-COPD subjects.[5] This reduction is likely to contribute to a decrease in the maximum force generated by the diaphragm in COPD patients. Therefore, respiratory muscle weakness generally worsens with COPD progression. COPD is primarily an airway disease; however, patients with advanced stages may exhibit clinical manifestations like those observed in neuromuscular diseases. The neuromuscular component is often underappreciated clinically but presents a potential avenue for testing and treatment.[6]

Respiratory muscle (RM) testing and training provide an avenue for quantifying the level of dysfunction and strengthening respiratory muscles. Low maximum expiratory pressure (MEP) is associated with diminished cough strength. Low maximum inspiratory pressure (MIP) is a predictor of respiratory mortality in patients with COPD.[2] Testing of respiratory muscle strength is measured using either a basic aneroid manometer or more sophisticated digital devices. The American Thoracic Society developed a position statement on respiratory muscle testing in which specifications for the basic and advanced apparatus were provided.[7] Prediction equations were available for MIP and MEP; however, these equations have not been validated in the COPD population.[8]

Respiratory muscle training is accomplished using two types of resistive training devices: threshold pressure resistance using a spring-loaded valve, or flow resistance using an adjustable orifice. Resistance can be provided during inspiration, expiration, or concurrently during inspiration and expiration. The available literature implies that RMT can offer benefits to the COPD population by improving strength, endurance, and perceived dyspnea.[8-11] We hypothesize that 8 weeks of concurrent inspiratory and expiratory muscle training will lead to significant improvements in respiratory muscle strength (MIP and MEP), spirometric values, perceived dyspnea, and quality of life in patients with COPD, with the most pronounced benefits occurring in those with more severe disease.

MATERIALS AND METHODS

Researchers employed a single-cohort pre/post-intervention study protocol to investigate the effects of 8 weeks of concurrent respiratory muscle training in patients with COPD at various stages. The initial study cohort included 43 participants. Inclusion criteria required participants to have a diagnosis of COPD according to the GOLD guidelines and to be between 40 and 90 years of age in their usual state of health. Patients with acute changes in sputum colour, fever, recent antibiotic use, or pregnancy at any gestational age were excluded. Before providing informed consent, all participants were given detailed study procedures and associated risks.

This study was approved by the university's Institutional Review Board (IRB number 2015R4181). Data were collected on two separate days, eight weeks apart. During the initial session, participants underwent assessments, including height and weight measurements, spirometry, maximum inspiratory and expiratory pressure evaluations, and completion of the COPD Assessment Test (CAT), Medical Research Council (MRC) Breathlessness Scale, and Airways Questionnaire 20 (AQ20). Following baseline assessments, participants received instructions on the respiratory muscle training device through demonstrations and return demonstrations. They were instructed to use the device twice daily (morning and evening) over the subsequent eight weeks. All baseline assessments were repeated after 8 weeks of RMT. We chose an 8-week duration based on prior research that demonstrated significant improvements in respiratory muscle strength and quality of life over similar timeframes.[11] This duration balances sufficient time for physiological adaptations with feasibility and participant compliance.

Spirometric testing adhered to ATS guidelines[12] using the EasyOne Spirometer (nDD Medical Technologies). Maximum inspiratory and expiratory pressures were measured with a pressure gauge manometer (Instrumentation Industries) and MicroRPM (Micro Medical/Vyaire), respectively,

Table 1. Demographic data of study participants

	N	Mean	Median	Std. Dev.	Min	Max
Age (years)	27	71.56	73.00	7.149	56	82
Height (in)	27	66.5	66.0	3.46	60.5	72.0
Weight (lbs)	27	166.40	159.0	42.25	98.6	239
BMI (kg/m^2)	27	26.42	25.70	6.03	17.7	40.7

following the ATS criteria for respiratory muscle testing.[7] Dyspnea and breathlessness were assessed using the MRC Breathlessness Scale, a validated tool for COPD patients.[13] Quality of life was evaluated using the AQ20 and CAT, which have both been validated in assessing quality of life and health status in COPD patients.[14-17]

RESPIRATORY MUSCLE TRAINING

The study participants received a PowerLung Muscle Training Device (PowerLung, Inc.) to provide inspiratory and expiratory muscle training. The PowerLung threshold device uses a spring-loaded valve to generate resistance to breathing, with separate control of inspiratory and expiratory resistances. The exercise protocol required the participants to complete three sets of 10 inspiratory breaths and three sets of 10 expiratory breaths twice daily. For each breath within the set, the participant was instructed to perform a 3-second inspiration, a 2-second pause, and a 3-second expiration. When participants completed 10 breaths (1 set) with easy effort, they were instructed to increase the resistance by ⅛ of a turn on the inspiratory or expiratory valve. They recorded changes in a training log provided by the researchers. While the PowerLung device uses a spring-loaded valve to adjust resistance, the absolute pressure load (e.g., in cmH_2O) associated with a ⅛-turn adjustment is not provided by the manufacturer and is not directly quantifiable without bench testing. However, the device is designed to offer progressive resistance training, and increased turns produce visibly greater loading effort. Additionally, study personnel contacted each participant at 4 weeks to reinforce adherence, answer questions, and review progress.

STATISTICAL ANALYSIS

The data analysis was performed using SPSS, v27 (IBM, Inc.). Statistical significance was set at $p < .05$. For an effect size of 0.6, power of 0.8, and 2-sided with 5% significance, an estimated 24 participants would provide the optimal sample size. Power analysis was conducted using the SPSS program. The sample size calculation was conducted for the paired *t*-test comparison of pre- and post-intervention outcomes in the full cohort. The sample size was estimated based on a medium effect size of 0.6, and consistent with prior respiratory muscle training studies showing moderate to large improvements in MIP and MEP outcomes.[10,14] This effect size was selected to ensure adequate power to detect physiologically meaningful changes in respiratory muscle strength. The paired *t*-test was used to analyze the differences in pre- and post-data values for all participants and the respiratory muscle, spirometry, and questionnaire data. The Wilcoxon signed-rank test was used to determine the pre-post differences in respiratory muscle, spirometry, and questionnaire data based on the COPD GOLD categories. Subgroup analyses by GOLD stage (GOLD 2, 3, 4) were exploratory. The study was powered a priori for paired pre–post comparisons in the full cohort (primary endpoints: MIP and MEP) and was not powered specifically for within-stage subgroup tests given the small cell sizes (n = 8, 12, and 7, respectively).

RESULTS

Of the 43 participants initially enrolled, 16 were not included in the post-data analysis. Thirteen did not complete the study. Reasons for non-completion included personal schedule conflicts, withdrawal of consent, development of unrelated health issues, and lost to follow-up despite multiple contact attempts. Three participants were removed from the study because they failed to meet spirometry criteria for obstructive lung disease ($FEV_1/FVC > 0.70$). While dropout was substantial, the final cohort of 27 participants represented a broad distribution of GOLD classifications and completed all intervention and data collection protocols. The dropout rate limited subgroup comparisons; however, the consistency of statistically significant improvements across multiple outcome variables suggests the observed effects are likely attributable to the intervention rather than sampling bias.

For the 27 participants completing the study, there was no missing data. Demographic data are presented in **Table 1**. Pulmonary function test values, questionnaire scores, and respiratory muscle trainer settings are presented in **Table 2**. A total of 27 participants completed all study requirements, which included pre-test collection, eight weeks of respiratory muscle training, and post-test collection. **Table 3** shows the GOLD COPD classifications of the participants at the start and end of the study. Of the 27 participants completing the study, 14 identified as female and 13 identified as male, and the mean age was 71.6 years.

For the entire cohort, pre-vs-post data analysis demonstrated that an 8-week respiratory muscle training program resulted in a statistically significant difference in MIP (mean increase 14.1 cm H_2O, $p < 0.001$), MEP (mean increase 20.1 cm H_2O, $p < 0.001$), CAT scores (mean decrease 2.92, $p < 0.02$), and AQ20 scores (mean decrease 1.67, $p < 0.005$). We also analyzed the difference between the pre- vs post-settings for PowerLung. Both the inspiratory and expi-

Table 2. Pulmonary function test values, questionnaire scores, and respiratory muscle trainer settings of study participants

	Period	*N*	Minimum	Maximum	Mean	*SD*
MIP (cm H_2O)	Pre	27	-28	-107	-63.04	20.78
MIP (cm H_2O)	Post	27	-33	-126	-77.15	23.69
MEP (cm H_2O)	Pre	27	28	134	77.85	26.78
MEP (cm H_2O)	Post	27	40	157	97.96	31.97
FVC (L)	Pre	27	0.78	3.98	2.21	0.73
FVC (L)	Post	27	0.84	3.73	2.24	0.67
FEV1 (L)	Pre	27	0.27	2.05	1.04	0.44
FEV1 (L)	Post	27	0.35	1.90	1.07	0.43
FEV1/FVC Ratio (%)	Pre	27	0.27	0.66	0.47	0.10
FEV1/FVC Ratio (%)	Post	27	0.23	0.69	0.475	0.10
MRC QUEST (score)	Pre	27	1.0	4.5	2.50	1.08
MRC QUEST (score)	Post	27	1.0	4.0	2.54	1.09
CAT QUEST (score)	Pre	27	4	30	17.07	6.57
CAT QUEST (score)	Post	27	5	22	14.15	5.07
AQ20 (score)	Pre	27	1	16	8.30	4.21
AQ20 (score)	Post	27	0	13	6.63	3.61
PowerLung Inspiratory Setting (#)	Pre	27	1	3	1.69	0.61
PowerLung Inspiratory Setting (#)	Post	27	1	6	3.35	1.24
PowerLung Expiratory Setting (#)	Pre	27	1	1.5	1.15	0.23
PowerLung Expiratory Setting (#)	Post	27	1	3	1.94	0.62

Table 3. GOLD category distribution of study participants

		Start of the study (# of participants)	End of the study (# of participants)
Valid	GOLD 1	0	0
	GOLD 2	8	9
	GOLD 3	12	12
	GOLD 4	7	6
	Total	27	27

ratory settings pre-vs post-intervention reached statistical significance at $p < 0.001$. **Table 4** shows the paired *t*-test results for items demonstrating a statistically significant difference at the 0.05 level. The other paired *t*-test items did not reach statistical significance for the entire cohort. However, FEV_1 approached significance, with a *p*-value of 0.064. Effect sizes reported in Table 4 ranged from medium (i.e. 0.478) to large (i.e. > 0.8).

Among the 27 participants, eight were initially classified as GOLD 2 (FEV1 50-79% predicted), 12 as GOLD 3 (FEV1 30-49% predicted), and seven as GOLD 4 (FEV1 < 30% predicted).[1] Following 8 weeks of concurrent RMT, nine were classified as GOLD 2 (FEV1 50-79% predicted), 12 as GOLD 3 (FEV1 30-49% predicted), and six as GOLD 4 (FEV1 < 30% predicted). We had two GOLD 3 participants convert to GOLD 2, one GOLD 4 participant convert to GOLD 3, and one GOLD 2 participant convert to GOLD 3. The nonparametric Wilcoxon signed-rank test was conducted to examine the differences in pre- and post-test values based on the GOLD category. In this analysis, the post-MIP values for GOLD categories 2 ($p < 0.008$), 3 ($p < 0.033$), and 4 ($p < 0.027$) were significantly different from the pre-values at the 0.05 level. The post-MEP values for GOLD categories 2 ($p < 0.013$), 3 ($p < 0.023$), and 4 ($p < 0.046$) were significantly different from the pre-values). The Wilcoxon signed-rank test also revealed a statistically significant difference between the pre- and post-CAT scores for the GOLD 4 participants ($p < 0.027$). Wilcoxon signed-rank analysis did not find a significant difference in FEV_1 based on GOLD category, e.g. GOLD 2 ($p = .812$), GOLD 3 ($p = .09$), and GOLD 4 ($p = .074$). See **Table 5** for the items demonstrating a significant difference from the Wilcoxon signed-rank analysis. Because subgroup analyses were not powered a priori and per-group samples were small, these findings are exploratory and intended to inform future studies.

Table 4. Paired *t*-test results for respiratory muscle, spirometry, questionnaires, and PowerLung device settings

	Mean	Std. Deviation	95% Confidence Interval of the Difference Lower	Upper	*t*	df	Sig. (2-tailed)	Effect size
MIP Post vs Pre (cm H_2O)	14.111	12.333	18.990	9.232	5.945	26	.001*	1.144
MEP Post vs Pre (cm H_2O)	20.111	20.112	12.155	28.067	5.196	26	.001*	1.000
FVC Post – Pre (L)	0.0252	.25165	-0.07436	0.12473	0.520	26	.607	0.477
FEV1 Post – Pre (L)	0.0367	0.0985	-0.00231	0.07564	1.934	26	.064	.759
FEV1/FVC Ratio (%) Post – Pre	0.0087	.04145	-.007731	0.0251	1.086	26	.287	.589
CAT (score) Post vs Pre	2.926	6.120	0.505	5.347	2.484	26	.020*	.478
AQ20 (score) Post vs Pre	1.667	2.828	0.548	2.786	3.062	26	.005*	.589
PowerLung Insp Setting (#) Post vs Pre	1.667	1.323	1.143	2.190	6.547	26	.001*	1.260
PowerLung Exp Setting (#) Post vs Pre	0.7963	0.6203	0.5509	1.04169	6.670	26	.001*	1.284

* Indicates a statistically significant value at the 0.05 level.

Table 5. Wilcoxon signed-rank test for pre versus post comparisons based on GOLD category

	GOLD 2	
	z score	*p*-value
MIP	-2.67	.008*
MEP	-2.49	.013*
FEV1	-.237	.812
CAT	-.238	.812
MRC	-.272	.785
AQ20	-1.83	.068
	GOLD 3	
	z score	*p*-value
MIP	-2.14	.033*
MEP	--2.28	.023*
FEV1	-1.69	.090
CAT	-1.56	.119
MRC	-1.61	.107
AQ20	-1.44	.151
	GOLD 4	
	z score	*p*-value
MIP	-2.21	.027*
MEP	-1.99	.046*
FEV1	-1.79	.074
CAT	-2.21	.027*
MRC	-.680	.496
AQ20	-1.38	.167

* Indicates a statistically significant value at the 0.05 level.

Subgroup analysis based on BMI was constrained by small sample sizes. Specifically, two participants had a BMI < 19, while seven had a BMI > 30. Among those with a BMI < 19, improvements were observed in MEP, but not in MIP. Most participants with a BMI between 20-30 or 30 demonstrated improvements in MIP and MEP as well as in AQ20 and CAT scores.

ADHERENCE WITH TRAINING PROTOCOL

Adherence to the training protocol was supported through structured follow-up. Participants recorded any missed training sessions and resistance setting adjustments in a provided logbook. At the midpoint (week 4), study personnel contacted each participant to verify usage, reinforce proper technique, and encourage continued engagement. Based on participant logbooks, we had a 3% missed session rate for all participants. The missed sessions ranged from a high of 19 to a low of 0. A complete 8-week session of RMT for this study would equal 112 sessions. Progression was monitored using device settings, and participants were instructed to increase the resistance by ⅛ of a turn when 10 breaths could be performed without significant effort. Table 4 demonstrates statistically significant increases in both inspiratory and expiratory resistance settings ($p < .001$), suggesting that participants not only adhered to the protocol but also progressively increased training load in accordance with the instructions.

DISCUSSION

This study aimed to determine the impact of concurrent respiratory muscle training in participants with COPD. After eight weeks of RMT, we observed statistically significant improvements in MIP, MEP, and quality of life scores across the entire cohort. Most participants demonstrated improvements or stabilization in the MIP, MEP, FEV1, MRC, CAT, and AQ20 scores. The results of the study showed that participants experienced an average increase of 14.11 cm H_2O in MIP and an average increase of 20.11 cm H_2O in MEP. The improvements were statistically significant. Furthermore, the increases in MIP surpassed the increases reported in a recent systematic review conducted by Figueiredo.[11] In the review, the mean rise in isolated MIP

(reported as PiMax) was 10.64 cm H_2O. Unfortunately, no data were provided in the MEP review. Of the 27 participants in our study, 26 were able to increase their MIP value, and 24 were able to increase their MEP value after 8 weeks of concurrent RMT. Concurrent RMT may lead to improved neuromuscular efficiency, which can enhance respiratory muscle strength. For instance, the phrenic nerve, which is responsible for transmitting electrical signals to the diaphragm, shows increased activation following respiratory resistance training. This heightened phrenic motor neuron activation, along with the recruitment of additional phrenic nerve axons, could contribute to the observed gains in respiratory muscle strength.[18,19]

Similar to skeletal muscle training, respiratory muscles adapt to increased workloads. Patients with COPD have altered chest wall mechanics as well as myopathic changes that support a neuromuscular component of the disease.[20] RMT has the capacity to improve some of these alterations. This is evidence on the capability of inspiratory muscle training to improve MIP in patients with COPD; however, determining which COPD patients derive the greatest benefit from RMT has not been well established.[21]

Our post-study MIP changes were consistent with the systematic review and meta-analysis by Huang et al.[22] However, the authors of that systematic review did not report a statistically significant change in MEP. Our patients used concurrent muscle training, which likely produced greater stress on the respiratory muscles than isolated inspiratory or expiratory muscle training. We also attributed our improvements in MIP and MEP to the study protocol and follow-up communication during the 8-week period. The participants were instructed to increase the inspiratory and expiratory training settings when the load was perceived to be too easy. Our participants demonstrated a significant difference in the starting and ending PowerLung settings. This demonstrates that they were actively engaged in the training exercise, which increased when breathing was easy. Additionally, a researcher contacted participants after 4 weeks to inquire about their progress, answer any questions, and encourage adherence to the protocol.

When assessing spirometry changes, FEV_1 improved in most GOLD 3 and 4 participants. On average, GOLD 3 and 4 participants experienced a 76 mL increase in FEV_1. The GOLD 3 participants demonstrated an FEV_1 increase of 83 mL, and GOLD 4 participants a 63 mL increase. It's important to note that the clinically important difference for FEV_1 improvement in COPD is typically considered to be around 100 mL or a change of 5-10% from baseline.[23,24] The available evidence demonstrates that improvements in FEV_1 within this range are often associated with improvements in symptoms, exercise tolerance, and health-related quality of life.[24] Our GOLD 3 and 4 participants demonstrated an average change from baseline for FEV_1 of 8.8%. We are encouraged by the FEV_1 changes in these participants, who were categorized as having severe and very severe COPD. The typical annual decline in FEV_1 for patients is approximately 50-60 mL/year.[23] Forestalling further declines in lung function in this population could impact the sensation of dyspnea, exercise capacity, and impairment in quality of life, the most reported symptoms of COPD.[25]

Ramirez-Sarmieto[26] demonstrated that inspiratory muscle training can mitigate some of the myopathic changes, in part by reversing the altered ratio of Type I and II muscle fibres in respiratory muscle biopsies taken before and after training. The increases observed in our study may be attributed to the concurrent training protocol, as well as personalized adjustments to resistance settings based on participant feedback. This suggests that patients at varying stages of disease progression can derive distinct benefits from RMT, with those in more advanced stages potentially benefiting more from expiratory muscle strengthening due to its role in cough effectiveness and airway clearance.[6] Our GOLD 2 participants did not demonstrate the same level of improvement in FEV_1 as the other participants. It is possible that the benefits of RMT were less impactful on GOLD 2 participants since they started with stronger respiratory muscles.

Prior COPD trials have predominantly evaluated inspiratory muscle training (IMT) alone, showing consistent gains in MIP and patient-reported outcomes, but variable effects on expiratory strength and spirometry.[10,11,22,25] In contrast, our protocol employed concurrent inspiratory and expiratory training (IMT+EMT) with a threshold device and demonstrated significant improvements in both MIP and MEP across GOLD stages, along with CAT and AQ20 benefits. These findings align with work directly comparing isolated IMT, isolated EMT, and combined training, where the combined approach produced broader strength adaptations (including MEP) than IMT alone and is mechanistically attractive for COPD by addressing cough effectiveness and airway clearance in addition to inspiratory function.[27] Taken together with IMT-only systematic reviews,[10,11,22] the present data suggest that adding EMT may extend benefit beyond inspiratory strength, potentially improving expiratory pressure generation relevant to secretion management and symptom relief.

Muscle-specific adaptations to IMT in COPD are complex. Some evidence indicates that with inspiratory loading, patients, particularly in advanced disease with hyperinflation, may recruit accessory (extra-diaphragmatic) muscles to a greater extent due to altered mechanics and diaphragmatic shortening, potentially limiting diaphragm-focused training effects.[20,28,29] At the same time, classic mechanistic and histologic studies demonstrate diaphragmatic neuromuscular adaptations and structural changes after training (e.g., fibre-type shifts and myosin content/force characteristics), supporting a diaphragm contribution to observed strength gains.[26] Additionally, resistive loading studies show increased phrenic motoneuron activation with inspiratory resistance, consistent with training-related drive to the diaphragm.[18,19] These seemingly divergent observations likely reflect disease severity, loading strategy, and compensatory patterns. Our combined IMT+EMT protocol may distribute load more evenly across the respiratory pump, enhancing both inspiratory and expiratory muscle performance, and improving thoracoabdominal co-

ordination and cough effectiveness, while still permitting diaphragmatic adaptation.

Our subgroup analyses also demonstrated statistically significant improvements in MIP and MEP across all GOLD stages. The fact that patients with GOLD 2, 3, and 4 disease all achieved measurable gains in both inspiratory and expiratory pressures underscores the robustness of the training effect. These findings suggest that respiratory muscle weakness in COPD remains amenable to targeted intervention across the disease spectrum, even in advanced stages where hyperinflation and neuromuscular inefficiency are most pronounced. Importantly, the consistent improvements in MIP and MEP may explain the observed benefits in patient-reported outcomes, since stronger inspiratory pressures are known to be an independent predictor of mortality in COPD, and higher expiratory pressures are closely associated with more effective cough and secretion clearance.[2]

We conducted GOLD-stratified analyses to explore whether disease severity modifies response to RMT, a biologically plausible question given greater neuromuscular dysfunction and hyperinflation with advancing COPD. These analyses were not powered and therefore serve to guide future, prospectively powered trials.

Our quality-of-life findings following eight weeks of RMT support this assumption. The CAT is a patient-reported outcome measure that assesses the impact of COPD on daily life. It included questions related to breathlessness, fatigue, chest tightness, and emotional impact. The AQ20 provides a quality-of-life assessment associated with breathlessness, activities of daily living, sleep, and coughing. There is supporting evidence for respiratory muscle training effects on quality of life.[30-33] RMT strengthens the diaphragm and other respiratory muscles. Stronger muscles improve breathing efficiency, making breathing easier, especially during activities of daily living. Stronger respiratory muscles allow increased exercise capacity. With improved exercise tolerance, individuals with COPD can engage in more physical activity. As exercise tolerance improves, individuals can sustain physical activity for a longer duration. This increased endurance translates to a better overall physical function and a greater sense of well-being. Our findings demonstrated direct improvements in the quality-of-life variables following training. Although no significant changes were detected in the Medical Research Council (MRC) Breathlessness Scale, this may be explained by its relatively coarse scoring system, which may lack sensitivity to detect subtle changes in breathlessness following RMT.[13] Future studies could benefit from incorporating more granular tools, such as the Borg Dyspnea Scale, to capture nuanced changes in this domain.

RMT focuses on strengthening muscles involved in breathing through controlled and coordinated breathing exercises. RMT has the potential to improve lung function by enhancing muscle strength and coordination. This was anticipated through better breathing mechanics, reduced air trapping, and improved respiratory efficiency. Gradual increases in resistance force muscles to work harder, leading to hypertrophy (muscle growth) and improved force generation. Using resistance-based training devices or techniques, such as respiratory muscle trainers, creates a demand for respiratory muscles, particularly the diaphragm and intercostal muscles.

LIMITATIONS

While this study demonstrated the efficacy of RMT, several limitations warrant consideration. First, the absence of a control group limits the ability to isolate the effects of RMT from other potential confounders such as spontaneous disease variation or placebo effects. Second, the small sample size, particularly within the GOLD stage subgroups, may have reduced the generalizability of the findings. The GOLD-stratified analyses were underpowered and exploratory; effect estimates within GOLD categories should be confirmed in larger, prospectively powered studies. Finally, reliance on patient self-reports for adherence to the training protocol introduces potential biases that could influence outcomes. Future research with larger, randomized controlled trials is needed to validate these findings and to establish optimal RMT protocols for specific patient subgroups.

CONCLUSIONS

In conclusion, concurrent inspiratory and expiratory muscle training is a suitable intervention to improve respiratory muscle strength and quality of life in patients with COPD. RMT is a low-cost, non-pharmacologic intervention that can be integrated into pulmonary rehabilitation programs, particularly for patients in GOLD stages 3 and 4 who are most likely to benefit. We also recommend that respiratory therapists and rehabilitation providers consider RMT as an adjunct to standard care. These findings provide a basis for integrating RMT into comprehensive management strategies for COPD, particularly for patients with advanced disease, who may derive the greatest benefits. Further research is needed to explore the long-term effects of RMT and its integration with other rehabilitation modalities. By targeting both inspiratory and expiratory muscle groups, the RMT addresses the multifaceted respiratory dysfunction observed in COPD, including hyperinflation-induced diaphragmatic shortening and diminished expiratory pressure. Additionally, improvements in respiratory muscle strength may reduce dyspnea and fatigue, thereby enabling greater participation in exercise-based rehabilitation.

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COMPETING INTERESTS

All authors have completed the ICMJE uniform disclosure form and declare no conflict of interest.

ETHICAL APPROVAL

This study was approved by the university's Institutional Review Board (IRB number 2015R4181).

AI STATEMENT

The authors confirm that no generative AI or AI-assisted technology was used to generate content.

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