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Inspiratory Muscle Training in Chronic Obstructive Pulmonary Disease Patients: A Scoping Review

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ABSTRACT

Introduction: COPD is a leading cause of chronic disease and death globally, with high mortality rates for those hospitalized or requiring mechanical ventilation. Delaying disease progression and improving patient outcomes can reduce healthcare costs and societal impact. Respiratory muscles play a crucial role in COPD, and specific endurance respiratory exercises have been shown to improve muscle fibers and mitochondrial activity, reducing oxidative stress and fatigue. Inspiratory Muscle Training (IMT) is a recommended pulmonary rehabilitation technique that enhances lung capacity and improves exercise performance by altering muscle fiber types and distribution. However, the impact of IMT implemented in COPD remains unclear. This scoping review summarizes the existing studies relating to the potential application of IMT in COPD patients.

Content: A scoping review was performed in PubMed, Academic Search Complete/EBCSCO, and Grey Literature sources, including Google Scholar. IMT studies evaluate interventions for COPD patients that were published in 2018-2023 were included.

Results: We reviewed 644 studies, nine IMT interventions met inclusion criteria and were included in the current study. In the majority of studies, improvements were noted through the implementation of IMT interventions. Several facilitators and barriers were noted during implementation and contributed to the success or failure of the intervention.

Conclusion: Overall, IMT had conflicting effects on a variety of COPD patient outcomes. Numerous studies demonstrated, however, that IMT can improve COPD-related outcomes, such as respiratory muscle function, dyspnea symptoms, quality of life, autonomic function, exercise tolerance, diaphragmatic strength and reduced exertional dyspnea, and balance.

Keywords: Scoping review; inspiratory muscle training; chronic obstructive pulmonary disease

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Introduction

COPD is among the primary causes of chronic disease and death worldwide.[1] Annual death rates range from 11 to 50 percent for those who require hospitalization due to a COPD exacerbation, from 5 to 50 percent for those who require mechanical ventilation, and from 37 to 80 percent for those who are readmitted to the hospital due to a previous exacerbation.[2] Health care costs may be reduced and the disease's global and societal effect lessened if actions are taken to delay the progression of the disease and improve patients' prognoses.[3] Individuals with COPD have restricted exercise capacity due to several factors, including breathing, gas exchange, cardiovascular disease[4], and peripheral muscle abnormalities.[5]

Respiratory muscles are responsible for increasing airflow to the lungs by raising the ribs and widening the chest wall, as well as decreasing airway resistance and intrathoracic pressure.[6] It is possible that the activation of respiratory muscles during respiration differs substantially from that of other skeletal muscles. However, the capacity of these two muscle groups to adapt to a variety of situations and functional demands is comparable, culminating in a comparable response to a training stimulus.[6,7] Powers and Criswell (Year?) have discovered that specific endurance respiratory exercise improved the number of fibres and mitochondrial activity in respiratory muscles.[8] The research determined that exercise had a positive effect on oxidative stress and delayed respiratory muscle fatigue.[8] Type I, type IIA, and type IIB fibres are prevalent in respiratory muscles and are also found in peripheral skeletal muscles. Type I, Type IIA, and Type IIB muscle fibers are classifications based on contraction speed, fatigue resistance, energy production, and suitability for different activities. Type I fibers contract slowly, resist fatigue, rely on aerobic energy, and excel in endurance tasks. Type IIA fibers contract moderately, have intermediate fatigue resistance, use aerobic metabolism, and are suitable for mixed endurance-strength activities. Type IIB fibers contract rapidly, fatigue quickly, depend on anaerobic energy, and are well-suited for intense bursts of activity like sprints or weightlifting. The amount and placement of these fibres in the diaphragm muscle, however, differ from those in other peripheral muscles like the quadriceps. The diaphragm has 80% oxidative fibres (types I and IIA), which are fatigue-resistant fibres, while the quadriceps contains just 35-45%.[9]

COPD patients have inspiratory muscle dysfunction, which is followed by dyspnea and impaired exercise capacity.[10,11] As a consequence, pulmonary rehabilitation is recommended as an effective cardiorespiratory therapeutic technique, resulting in increased exercise performance and reduced dyspnea in individuals with varied degrees of disease severity.(12) Inspiratory Muscle Training (IMT) is a pulmonary rehabilitation technique that enhances physical fitness by maximizing lung capacity.[13]

IMT additionally comes with structural alterations in the types and distribution of muscle fibres in the inspiratory muscles. After five weeks of IMT, the number of type I fibres and the size of type II fibres in external intercostal muscle increased in COPD patients.(14) IMT improved inspiratory muscle work capacity by reducing relative work (percentage of maximal muscle work capacity). After IMT, the quantity of cardiac output consumed by the inspiratory muscles decreases; as a result, a larger portion of cardiac output can be redirected to peripheral muscles, thereby increasing exercise capacity.[15]

Gosselink et al. conducted a meta-analysis of 32 randomized controlled trials (RCTs) on the impacts of IMT in patients with COPD. The authors performed general and subgroup analyses of training mode (resistance strength and the inclusion of pulmonary rehabilitation) and subject characteristics. IMT increases inspiratory muscle strength, functional capacity, dyspnea, and quality of life, according to the authors. IMT utiliszed in pulmonary rehabilitation programs improved inspiratory muscle strength and exercise performance in participants with inspiratory muscle weakness.[16] However, the impact of IMT implemented in COPD remains unclear. This scoping review summarizes the existing studies relating to the potential application of IMT in COPD patients.

Objectives

The purpose of this scoping review is to identify and assess the literature on inspiratory muscle training in COPD. What improvements or effects have been reported for COPD patients who train their inspiratory muscles?

Search Strategy

Using the Joanna Briggs Institute (JBI) methodology for scoping reviews, the researchers conducted a scoping review. The databases searched for scholastic peer-reviewed articles included PubMed, Academic Search Complete/EBCSCO, and Google Scholar, in addition to Grey Literature sources.

Studies evaluating the effect of inspiratory muscle training on COPD patients that were published between January 2018 and January 2023 are included. One reviewer extracted the data, and the second reviewer verified thirty percent of the studies.

A preliminary search of PubMed, Academic Search Complete/EBCSCO, and Grey Literature sources, including Google Scholar, was conducted, followed by an analysis of the text words in the title and abstract, as well as the index terms used to describe the articles. The identified keywords and index terms were then utilized in a second search of relevant databases. Thirdly, the reference lists of chosen studies were combed for additional pertinent articles. All studies, regardless of publication date, were considered for inclusion in the review. There was also consideration for including translated studies in the review. The search criteria contained two components. The first construct pertained to the population and was restricted to COPD patient-focused studies. The second construct was limited to inspiratory muscle training and related to the intervention. The inquiry was conducted utilizing a combination of the two concepts.

Inclusion and Exclusion Criteria

Inclusion criteria were studies examining the short- and long-term effects of inspiratory muscle training on stable or acutely exacerbating COPD patients. The studies must include at least one of the following outcomes: dyspnea, quality of life, exercise capacity, or maximal inspiratory pressure (PImax). This evaluation was limited to randomized controlled trials (RCT).

Not included in the exclusion criteria were sample size or the absence of a control group. Two researchers (ZF and IS) evaluated the title and abstract to identify potentially relevant articles. Inconsistencies were resolved through consensus or consultation with a third investigator.

Quality Rating

Using standardized critical appraisal instruments for Randomized Controlled Trials from JBI to determine the extent to which studies addressed the likelihood of bias in design, conduct, and analysis served as the basis for determining methodological quality.

Result

Initial investigation yielded 644 studies. After removing duplicates (102) of the result of searching, it remained 542. Both evaluators screened all titles solely for relevance, resulting in the selection of 117 articles and the exclusion of 385. Then, both evaluators (IS and ZF) evaluated all titles and abstracts in Rayyan, resulting in the exclusion of 135 articles and the retention of 26. The full-text versions of the included articles were obtained. The reviewers excluded 17 studies due to ineligibility requirements. No authors were contacted for further details. In accordance with the PRISMA Diagram (Figure 1), nine articles remained for inclusion in the present investigation.

Synthesis of Results and Methods of Analysis

Included were nine journal articles with peer review. The characteristics of the study (author, year, study design, population results, and conclusion) are outlined in Table 1. There are provided narrative descriptions of the investigations. The search was designed to be exhaustive in order to cover the current literature, and the included studies were published from 2018 to 2023.

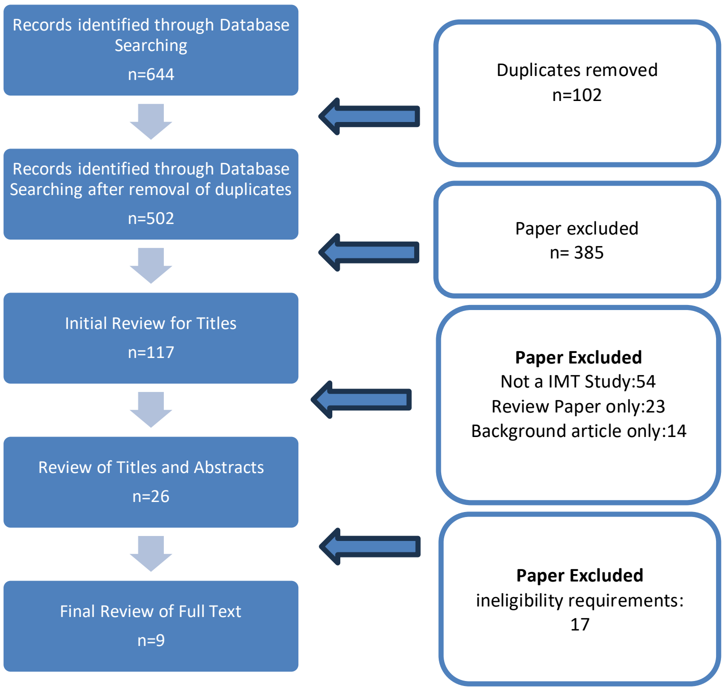

Figure 1 PRISMA diagram.[17]

Table 1 Study Characteristics (Design, Type, Population and Primary Outcomes)

Author	**Year**	**Study Design**	**Population**	**Outcomes Measures**	**Results**
Noppawan Charususin, et al. [18]	2018	Randomized Control Trial (RCT)	With inspiratory muscular weakness (Pimax: 5115 cm H2O) and cOPD, 219 patients were randomly assigned to the either intervention group (iMt+Pr; n=110) or the control group (Sham-iMt+Pr; n=109).	• 6MWD • Respiratory Muscle Function • Endurance Cycling Time	Adjunctive iMt-induced improvements in respiratory muscle function did not result in further gains in the main endpoint, the 6MWD. Additional increases in endurance time and decreases in dyspnea symptoms were seen during an endurance cycling test (secondary outcome).

Marc Beaumont, et al. [19]	2018	Single-Blind Randomized Controlled Trial	149 patients with severe or very severe COPD (FEV1 50% expected) were divided into two groups: PRP+IMT (n = 74) and PRP alone (n = 75).	• Dyspnoea (using the Multidimensional Dyspnoea Profile questionnaire at the end of a 6-min walk test (6MWT) at 4 weeks • The Borg (end of the 6MWT) • Modified Medical Research Council scales and in functional parameters (maximal inspiratory pressure (PImax), inspiratory capacity, 6MWT and quality of life)	Dyspnoea significantly decreased in both groups, although there was no statistically significant difference between the two groups in terms of the improvement. When comparing IMT+PRP versus PRP alone, the only statistically significant increase in PImax was seen.
Konrad Schultz, et al. [20]	2018	Randomized Controlled Trial used a parallel group design	Following high-intensity interval-based IMT (n=281) versus sham IMT (n=280) was assigned to 561 COPD patients (medical history of COPD, forced expiratory volume in one second (FEV1)/vital capacity 70%, and FEV1% pred 80% post-bronchodilation).	• Maximal Inspiratory Pressure (Pimax) • 6-Min Walk Distance, • Dyspnoea • Quality Of Life • Lung Function	PImax and FIV1 are improved when IMT is routinely included to a 3-week intense pulmonary rehabilitation programme. Functional ability, dyspnea, or quality of life were not further improved by IMT.
Yasemin Buran, et al .[21]	2022	Prospective single-blind randomized trial	Only IMT (n = 30) or MT above IMT at 40% of maximum inspiratory pressure (MIP) (n = 30)	• Functional Capacity • Respiratory Muscle Strength • Pulmonary Function • Dyspnea • Fatigue • Quality Of Life	The IMT + MT group outperformed the IMT alone in terms of functional capacity, respiratory muscle strength, pulmonary function, dyspnea, tiredness perception, and quality of life.

Ana Lídia, Carvalho Cutrim, et al.[22]	2019	Single-Center Controlled Study,with Balanced Randomization	22 COPD subjects joined the study	• Pulmonary Capacities And Inspiratory Pressure • Total Six-Minute Walk Test • Cardiac Autonomic Modulation.	With enhanced vagal modulation (total variability and HF [ms2; adjusted p 0.05]), higher expiratory and inspiratory capabilities, and increased 6-minute walk distance, the intervention group showed improvements in cardiac autonomic modulation.
Rodrigo Koch, et al. [23]	2020	Prospective Randomized and Cross-Over Study	Prior to and following 10 sessions of high intensity IMT (three times per week) over the course of 30 days nine COPD, subject performed a total of four identical constant work rate tests on a cycle ergometer at 75% of maximum work rate white using proportional assisted ventilation (PAV, individually adjusted) or control ventilation (SHAM, 4cm $H_2$0)	• Borg Scale • Maximal exercise tolerance • Respiratory pattern	IMT added before PAV-supported aerobic exercise improves exercise tolerance, relieves dyspnea, and causes positive changes in the ventilatory pattern during high-intensity training in patients with severe COPD.
Daniel Langer et al.[24]	2018	Prospective single-blind randomized trial	Twenty participants were randomly assigned to either IMT or a sham training control group (n=10), with FEV1 values of 47 and 19% predicted, Pimax values of 59 and 14 cmH2O, and cycle ergometer peak work rates of 47 and 21% anticipated.	• Lung Function, • Respiratory Muscle Strength, • Activity-Related Dyspnea, • Exercise Capacity	IMT improved mechanically weakened individuals with COPD and low Pimax's strength and endurance of the inspiratory muscle.

Bilel Tounsi et al.[25]	2021	Prospective single-blind randomized trial	32 male patients (age range, 62+ 6 years) with moderate to severe COPD. They were randomly allocated to either the control group (ET) with 16 members or the experimental group (IMT+ET) with 16 members, both of which had comparable features.	• Functional balance was assessed by the Berg Balance Scale (BBS), the Timed-up and Go (TUG), the Single Leg Stance test (SLS), and the Activities- specific Balance Confidence (ABC) scale • The strength of the inspiratory muscles (PI$_{max}$) was assessed by maximal inspiratory mouth pressure. • Functional exercise performance was assessed by the 6 minutes walking test (6MWT)	According to BBS and ABC, the results show that IMT in addition to ET enhances inspiratory muscle performance and functional balance in COPD patients when compared to ET alone.
Wenhui Xu et al.[26]	2018	Prospective single-blind randomized trial	In 92 individuals, inspiratory muscle training (IMT), combined inspiratory and expiratory muscle training in the same cycle (CTSC), or combined inspiratory and expiratory muscle training in separate cycles (CTDC) were randomly and evenly allocated.	• Respiratory Muscle Strength	While IMT alone just raised PImax, CTSC and CTDC improved the strength of the inspiratory and expiratory muscles.

Discussion

Improvements in the 6MWD (the primary outcome) did not differ statistically significantly across groups in the study of Noppawan (2018). Contrarily, patients in the intervention group significantly improved their ability for endurance exercise and respiratory muscle function (the secondary target that was predetermined). During the cycling test, it was also discovered that the intervention group had much less symptoms of dyspnea. The significance of external effort and power as training quality attributes of the training stimulus given to the inspiratory muscles is emphasized by this study. The magnitude of PImax increases was not significantly correlated with training volume or compliance (number of training sessions completed). Surprisingly, they found that the number of training sessions combined with the total amount of labour completed (representing pressure and volume responses throughout individual sessions) was the greatest predictor of PImax increases in a multiple regression model (see online supplementary table E5). This emphasizes how important it is to assess training quality (total work done) during IMT to ensure that training adaptations in response to the intervention are efficient.[18]

Dyspnea was improved in both groups in the second randomized controlled trial comparing the effect of IMT during a pulmonary rehabilitation program to a pulmonary rehabilitation program without IMT in patients with severe or very severe COPD, but the difference in dyspnea was not found to be different, even in the subgroup of patients with inspiratory muscle weakness (PImax 60 cm H2O). The two groups' improvements in quality of life and 6MWD were not substantial between the groups. Only in the IMT group paired with pulmonary rehabilitation did PImax improve significantly more than in the pulmonary rehabilitation group. [19] By boosting type II fibers, IMT has the ability to increase diaphragmatic velocity, [14], shorten the inspiratory duration [27] as well as prolong expiratory time, which might lessen hyperinflation (as shown by changes in inspiratory capacity during effort).(28,29) Therefore, IMT may lessen dynamic hyperinflation and, consequently, dyspnea in individuals with severe COPD by increasing PImax.

In comparison to sham IMT, patients with moderate to severe COPD showed a significant improvement in PImax and FIV1, according to the main findings of the third RCT. However, other clinically significant outcomes, such as dyspnea, QoL, functional capacity, and other lung function parameters, did not show a similar improvement. The intervention group's mean increase in PImax of 0.94 kPa (9.59 cmH2O) compared to the control group's corresponds to a PImax mean difference of 8.60 cmH2O, which was made possible by much longer outpatient programs. No additional improvements in clinical outcomes were found despite notable increases in PImax. [20] There might be numerous causes for this. First, the causal model could just be incorrect, and improvements in clinical PImax might not translate into clinical outcomes.[30] However, a number of studies contrasting IMT alone with controls discovered modifications in clinical outcomes. (16,31,32) Second, a 0.94 kPa influence could be negligible enough to result in clinically significant benefits in pulmonary rehabilitation. Longer-term measures are thus needed. By using a 6-month intervention, MAGADLE et al., for instance, showed larger results in PImax as well as effects in functional ability. [33] Third, changes in PImax may only alter clinical outcomes if no other drugs are concurrently affecting them. As a result, prospective IMT benefits may be hidden by the impact of pulmonary rehabilitation on clinical results. Fourth, various patient subgroups (such as female patients vs. male patients) may react to IMT differently.

In contrast to the meta-analysis of GOSSELINK et al., PImax at baseline did not lessen the severity of the influence on PImax.[16]Regardless of PImax changes, patients with high PImax levels at T0 benefit considerably from the intervention. Furthermore, none of the secondary outcomes were impacted by baseline PImax. As a result, the third trial does not support the idea that IMT is exclusively beneficial for people who have weak inspiratory muscles. The discrepancies in the results of the two studies may be explained by using different methods. While we looked at correlations between patient characteristics, GOSSELINK et al. examined associations between study components (such as mean baseline PImax and mean impact of IMT).[16] If patients are not chosen at random from the same population, which is difficult to verify, inferences from the research to the patient level may be misleading.[30] This reasoning supports the validity of those findings, as does the large sample size of our study. The mean 6MWD increased by more than 80 m in both research arms, both statistically and clinically. The AMD between groups in the ITT analysis was 1.9 m. The per-protocol analysis indicates that IMT may increase females' 6MWD by more than 13 m (Cohen's d=0.21). It is uncertain why the results of the per-protocol analysis and the ITT differ. As a result, the results of the per-protocol analysis should be regarded with caution. Based on the ITT analysis, we conclude that sex had no moderating effect on 6MWD. [20]

For Quality of Life (QoL), similar results were seen. With moderate to large effect sizes, both groups significantly improved, although IMT had no further effects. However, Hosp_Acute Exacerbation of Chronic Obstructive Pulmonary Disease (AECOPD) and sex both impacted quality of life.[20] Men and patients with AECOPD tended to have little benefit or even worsening effects, while extra IMT seemed to enhance females on various QoL subscales. For possible power reasons, no earlier investigation has produced comparable findings. The sex impact is explained by a number of reasonable explanations. For instance, female inspiratory muscles may deplete more gradually.(34,35) Females could experience IMT less stressfully as a result, and they might gain more benefits from the rise in PImax in terms of QoL. Furthermore, "relaxation breathing" (control group) may benefit men more than women. Breathing slowly and steadily for 21 days can be similar to relaxation training. [36] This may have an effect on psychological but not physiological characteristics.[37] However, all of the effects are minor, and the clinical significance is unknown.

Furthermore, it was discovered a considerable improvement in FIV1. Despite the fact that numerous studies showed that the alterations in FIV1 are important,[38] the clinical relevance of a 100 mL difference is unknown, particularly as other lung function metrics did not improve.

The Fourth Study found that a 12-week IMT with MT program increases functional capacity, respiratory muscle strength, pulmonary function, and overall Quality of Life in COPD patients while decreasing dyspnea and tiredness perception. The research found that when pulmonary rehabilitation included MT, FVC improved. [21] Changes in the elastic characteristics of the lungs and chest wall cause an increase in static hyperinflation under certain situations. Expiration towards relaxation volume gets progressively longer as airway resistance and flow constraint increase, and the following inspiration starts before relaxation volume is attained. This exerts an additional stress on the inspiratory muscles at the end of exhalation: they must overcome an additional "threshold" load associated to the respiratory system's elastic rebound before inspiratory flow begins. Furthermore, this happens despite the inspiratory muscles' decreasing mechanical advantage.[39] The addition of MT to IMT enhanced inspiratory muscle strength and endurance more than IMT alone, and it has the potential to reduce the oxygen cost of voluntary hyperpnea and ameliorate patients' reported dyspnea. [40]

The Fifth Study examined how breathing capacity, exercise tolerance, and autonomic function in COPD patients changed after 12 weeks of IMT at 30% of maximal inspiratory pressure (PImax). Our study's key conclusion demonstrates that a regular IMT programme was successful in increasing the maximum strength of the inspiratory and expiratory muscles, cardiac autonomic modulation, and functional capacity as assessed by the 6MWT in COPD individuals. The IMT procedure, however, did not demonstrate significant gains in respiratory function. [22] They discovered that the IMT protocol increases the linear indexes SD1, SD2, and vagal modulation in the frequency domain by increasing HF (ms2) and in the time domain by improving RMSSD, total power, and SDNN, indicating an increase in overall variability in the subjects who underwent the IMT protocol. [22] One of the main mechanisms causing the decreased HRV in COPD is hypoxia. It is plausible that the present intermittent hypoxia in COPD patients is the root cause of autonomic dysfunction. [41] The findings also suggest that sympathetic activity predominates in autonomic dysfunction, which has a significant impact on inflammatory responses.(42,43) Other studies found a substantial negative correlation between blood IL-6 levels in COPD patients and the parasympathetic modulation index (pNN50). [44] These findings may be explained by the fact that IMT increases pulmonary vagal afferents while inhibiting sympathetic activity. In addition, IMT may increase oxygen supply due to tidal volume augmentation while decreasing chemoreflex activity and sympathetic activity. [45,46]

In the sixth study, in severe COPD, adding IMT before a PAV-supported aerobic session improves exercise tolerance and dyspnea alleviation, as well as generates favourable alterations in the ventilatory pattern during high-intensity training. [23]

The main conclusions of the seventh study were that, compared to the control group, 8 weeks of home-based IMT was associated with improved diaphragmatic strength, decreased EMGdi/EMGdimax ratios, and ratings of exertional dyspnea intensity.[24] The results support the idea that perceived dyspnea during exercise in COPD is caused by a higher ratio of diaphragmatic activation to maximum, which may be treated with IMT. It is believed that the higher central motor command output from the cortex necessary to create a certain force or tension by a weaker muscle is what causes the impression of greater perceived muscular effort to occur when any skeletal muscle is impaired (either experimentally or as a result of illness). It has not been conclusively proven that afferent signals from weaker muscles contribute proportionately to perceived effort, and such research present technical challenges. A significant increase in EMGdimax following similar magnitude IC manoeuvres pre- and post-IMT accounted for the majority of the decrease in the EMGdi/EMGdimax ratio. Due to a combination of increased strength and potential neuronal adaptations that facilitated motor unit recruitment during the 8-week training period, the training-induced increase in EMGdimax may reflect a greater ability to recruit more motor units during maximal voluntary diaphragmatic activation.[47,48] The decrease in tidal inspiratory EMGdi is in line with a reduction in the number of motor units required to generate a given force as a result of muscle hypertrophy. [14,47,48]

Eight studies revealed that with the addition of IMT to ET, the berg balance scale (BBS) score improved by 2.7 points, and the activities-specific balance confidence (ABC) scale increased by 7.2%. In both groups, they discovered marginally significant correlations between inspiratory muscular strength and BBS and ABC, but only in the experimental group. According to BBS and ABC, the experimental group (IMT+ET) exhibits a significant improvement in functional balance. [25] According to Ferraro et al., it might be explained by a hypothetical physiological mechanism(s) through which IMT administered to ET improves balance. The first proposed mechanism is based on the stimulation of the diaphragm and intercostal muscles to respond to a different movement in varied activities and at a different frequency, apparently to maintain balance during rapid and unsteady upper body motions. According to Ferraro et al., it might be explained by a hypothetical physiological mechanism(s) through which IMT administered to ET improves balance. The first proposed mechanism is based on the stimulation of the diaphragm and intercostal muscles to respond to a different movement in varied activities and at a different frequency, apparently to maintain balance during rapid and unsteady upper body motions. [51,52]

The second possibility is based on intra-abdominal pressure. This pressure is known to rise when the vertical load on the body rises during walking or running.[53] This significant intra-abdominal pressure may aid to extend the lumbar spine and manage its direction.[53] In order to sustain the increase in intra-abdominal pressure during postural/trunk movement, the diaphragm and abdominal muscles co-activated. The diaphragm is therefore assumed to support trunk postural control by elevating intraabdominal pressure.[53] The idea that strengthening the inspiratory muscles improves functional balance and may help restore the equilibrium that seems to have been lost due to increases in trunk muscular activity is supported based on these probable physiological pathways.

In the ninth investigation, they found that, although IMT alone had no discernible effect on PEmax, two forms of combination training might enhance both inspiratory and expiratory muscle. We also showed that breathing pattern improvement in COPD patients may be possible with respiratory muscle training, with the effect being more obvious in individuals with weak inspiratory muscles.[26] More research into the physiological mechanisms behind the interactions between the various training components is necessary since the CTSC group's breathing frequency was much lower.

The mechanism that restricts airflow while engaging the expiratory muscles is still somewhat uncertain, caused nonspecific response to increased respiratory stimulation likely caused the expiratory muscle to contract during exhalation.[54] The abdominal muscle is activated during expiration, maintaining the diaphragm muscle's fibre length and force-generating capacity despite lung hyperinflation.[55] As a result, problems with the expiratory muscles might lead to exhaustion in the inspiratory muscles.

Conclusion

Multiple studies on inspiratory muscle training (IMT) in patients with COPD were reviewed. Overall, IMT had mixed effects on various outcomes in COPD patients. Nonetheless, numerous studies demonstrated that IMT can enhance COPD-related outcomes, including respiratory muscle function, dyspnea symptoms, quality of life, autonomic function, exercise tolerance, diaphragmatic strength and reduced exertional dyspnea, and balance.

Conflicts of Interest

There is no conflict of interest

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