Original Article

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Maximal Respiratory Mouth Pressures Assessment in Stable Chronic Obstructive Pulmonary Disease Patients in a Tertiary Hospital in Southwest Nigeria

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Abstract:

Objective: This study aimed to assess the respiratory muscle strength in stable chronic obstructive pulmonary disease (COPD) patients, via measuring maximal respiratory mouth pressures [maximal inspiratory pressure (PImax) and maximal expiratory pressure (PEmax)] to determine its association with disease severity and quality of life.

Material and Methods: The study was a cross-sectional comparative study. A hundred and forty subjects (70 COPD patients and 70 controls) were recruited. Measurements of PImax, PEmax and spirometry were then performed. The health-related quality of life, severity of obstruction and dyspnea in the COPD patients were assessed using the COPD Assessment Test (CAT), post-bronchodilator Forced Expiratory Volume in 1 second (FEV 1) and the modified Medical Research Council (mMRC) dyspnea scale, respectively. Data was analyzed using Statistical Package for the Social Science (SPSS) version 25.0 (SPSS IL USA.).

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Results: The mean (±S.D.) PImax and PEmax of the COPD patients (31.78±14.40 cmH_2O and 54.80±18.89 cmH_2O, respectively) were significantly lower ($p<0.001$) than the controls (80.40±7.50 cmH_2O and 95.44±12.52 cmH_2O, respectively). Both the PImax and PEmax correlated positively with the FEV_1 of the COPD patients ($r=0.658$ and 0.534, respectively, $p<0.001$). The PImax and PEmax decreased as the mMRC dyspnea grade worsened ($p<0.001$). There was a negative correlation between PImax; PEmax and the CAT score of the COPD patients ($r=-0.704$ and -0.583, respectively, $p<0.001$).
Conclusion: There was significant respiratory muscle weakness in the COPD patients compared with the controls. The respiratory muscle weakness worsened as the airflow obstruction and dyspnea worsened. Respiratory muscle weakness may also add to the negative impact COPD has on the health status of COPD patients.

Keywords: COPD, maximal respiratory mouth pressure, PEmax, PImax, respiratory muscle strength

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a major public health challenge, as it is now one of the top three causes of death worldwide[1]. Additionally, it ranks high among the leading causes of morbidity and mortality worldwide[2]. Even though COPD primarily affects the lungs, it is also associated with a lot of systemic effects; which add to its increased morbidity. Respiratory muscle weakness is one of the adverse systemic effects of COPD[3], and it has been associated with reduced exercise tolerance including reduced quality of life[4]. Furthermore, respiratory muscle weakness is also an independent determinant of survival in adults with COPD[4].

Respiratory muscle weakness, seen in COPD, is caused by hyperinflation that occurs during the course of the disease and other multiple factors; such as malnutrition, systemic inflammation and possibly treatment with corticosteroids, leading to steroid-induced myopathy[4].

For a considerable number of years, the assessment of COPD has been based solely on the severity of airflow limitation[5]; however, being a respiratory disease with multiple systemic pathological components, adding to its increased morbidity[6], the horizon of assessment of COPD patients should rise beyond just assessing airflow limitation. Many instruments have been used to measure the ventilatory function and abnormalities in COPD, and standardized values have been obtained that are measurable and comparable in various centers around the world. However, the measurement of the respiratory muscle strength, which forms an integral part of an assessment of COPD, has had limited attention worldwide. Consequently, there are few centers where these are measured routinely

Measurement of the maximal respiratory pressures [maximal inspiratory pressure (PImax) and maximal expiratory pressure (PEmax)] is a simple, convenient and non-invasive way of assessing respiratory muscle strength[4,7,8]. Maximal inspiratory pressure (PImax) is the maximum negative pressure that can be generated from one inspiratory effort, starting from functional residual capacity (FRC) or residual volume (RV)[9]. Maximal expiratory pressure (PEmax) measures the maximum positive pressure that can be generated from one expiratory effort, starting from total lung capacity (TLC)[10].

Measurement of PImax and PEmax have been validated in many studies as a way of assessing respiratory muscle strength in patients with COPD[7,8,11,12]. Routine assessment of PImax and PEmax will help detect respiratory muscle dysfunction early in patients with COPD. Subsequent, prompt intervention; such as inspiratory muscle training, could improve the quality of life and reduce the morbidity and mortality associated with COPD[13]. In addition, there are no published studies on

the measurement of respiratory muscle strength in COPD patients in Nigeria, which has the largest black population in the world. Hence, this study is provides valuable data on this subject for reference purposes for blacks as well as influence management protocols.

The objectives of this study were to:

Assess the PImax and PEmax in stable COPD patients in the Southwestern region of Nigeria and compare it with controls.

Assess the association between PImax and PEmax; the level of dyspnea; and the severity of airflow obstruction in patients with COPD.

Determine the correlation between PImax and PEmax; and Health Related Quality of Life (HRQoL) in patients with COPD.

Material and Methods

This study was a cross-sectional comparative study. It was conducted at the COPD Clinic, Obafemi Awolowo University Teaching Hospitals Complex (OAUTHC), Ile-Ife, Osun-State, Nigeria, from; November 2020 to March 2021.

A total number of 140 study subjects, comprising 70 stable COPD patients and 70 age- and sex-matched controls, were recruited for this study.

The inclusion criteria for this study allowed for the enrolment of patients with COPD in a stable state (defined by no acute worsening of respiratory symptoms resulting in a change in dosage or frequency of administration of the routine medications or additional therapy, and no hospital admission in the preceding twelve weeks), who were ≥40 years of age and had given written informed consent to participate in the study.

Patients with other diseases that can cause respiratory muscle weakness; such as neuromuscular disease and heart failure, were excluded from the study. Patients who declined consent were not included in the study.

The control group comprised age and sex-matched subjects that were apparently healthy, not suffering from any acute or chronic respiratory disease and with normal spirometry results were recruited from the hospital staff and patients' relatives.

A structured questionnaire was used to obtain the socio-demographic data and clinical information from the participants. In COPD patients, the severity of dyspnea was assessed using the modified Medical Research Council (mMRC) dyspnea scale, while health-related quality of life was assessed using the COPD Assessment Test (CAT).

All the subjects had spirometry administered to determine their Forced Expiratory Volume in 1 second (FEV_1), Forced Vital Capacity (FVC) and the ratio of FEV_1/FVC, using the Micro 1 Diagnostic Spirometer (Vyaire Medical, Germany); according to the ATS/ERS spirometry guidelines 2019[14]. The PImax and PEmax of all the study subjects were measured using the portable Respiratory Pressure Meter --MicroRPM (Vyaire Medical, Germany).

According to the ATS/ERS Statement on Respiratory Muscle Testing[9], the procedure was first described in clear terms and demonstrated to the subjects. The procedure was performed in the sitting position. To measure the PImax, the MicroRPM Pressure Meter switch was slid from the off position to the PImax position. The subjects were instructed to insert the mouthpiece into their mouth, ensuring the flange is placed over the gums and inside the lips, while the bite blocks were positioned between the teeth. The subjects were further instructed to exhale till their lungs were empty (that is: exhale to residual volume), then make a forced inhalation against the MicroRPM Pressure Meter, exerting the maximum effort possible, for a duration of at least 2 seconds to measure the PImax. This was repeated three times to establish the best value. The displayed result, the maximum average pressure sustained over a second period of the test, in centimetres of water (cmH_2O) was recorded.

To measure the PEmax, the MicroRPM Pressure Meter switch was slid from the off position to the PEmax position. While still in the seated position, the subjects were instructed to insert the mouthpiece into their mouth, ensuring the flange was placed over the gums and inside the lips, while the bite blocks were positioned between the teeth. The subjects were further instructed to inhale until their lungs were full (that is: inhale to total lung capacity), then make a forced exhalation against the MicroRPM Pressure Meter, exerting maximum effort possible for a duration of at least 2 seconds to measure the PEmax. This was repeated three times to establish the best value. The displayed result, the maximum average pressure sustained over a second period of the test, in centimetres of water (cmH_2O) was recorded.

Definition of term

COPD patients: patients with chronic cough, sputum production, dyspnea, exposure to risk factors for COPD, and a spirometry that demonstrates a post-bronchodilator $FEV_1/FVC<0.70$[1].

Ethical approval was obtained from the Ethics and Research Committee of the Institution (Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Osun-State, Nigeria). Protocol Number: ERC/2020/03/15. Written informed consent was obtained from all the study participants before the study was conducted, in accordance with the Declaration of Helsinki.

The data obtained were analyzed using the Statistical Package for the Social Sciences (SPSS) version 25.0 computer software (SPSS IL U.S.A). Descriptive statistics were used to summarize univariate data and presented using frequency tables. The statistical tests used for the continuous variables in this study were: student T-test and ANOVA. Chi-square (X^2) tests were employed for the comparison of qualitative variables. Spearman's correlation was used to check the relationship between PImax, PE max, FEV_1 and FVC, as the data analyzed were significantly different from normally distributed data. Multiple linear regression was used to determine the association of CAT scores with FEV_1, mMRC, BMI, PImax and PEmax. A p-value of less than 0.05 was considered statistically significant.

Results

A total of hundred and forty subjects participated in the study; 70 COPD patients and 70 age- and sex-matched controls. The COPD patients comprised 25 (35.7%) males and 45 (64.3%) females, while the controls were 24 (34.3%) males and 46 (65.7%) females (p-value=0.859).

The ages ranged from 45–83 years, with a mean (±S.D.) of 71.28±7.50 for the COPD patients and 42–83 years with a mean (±S.D.) of 70.92±7.94 for the controls. The difference in age between the COPD patients and the controls was not statistically significant (p-value=0.785) (Table 1).

The mean (±S.D.) FEV_1 of the COPD patients was 53.41±20.26 %predicted, FVC was 83.12±20.88 %predicted and FEV_1/FVC was 54.19±11.46 %predicted. In the controls, the mean(±S.D.) FEV_1 was 89.47±16.04% predicted, FVC was 87.60±15.39 %predicted, and FEV_1/FVC was 82.98±9.74. The differences in the FEV_1 and FEV_1/FVC between the COPD patients and the controls were statistically significant, (p-value<0.001) Table 1.

The PImax of the COPD patients ranged between 10.00–69.00 cmH_2O, with a mean (±S.D.) of 31.78±14.40 cmH_2O, while their PEmax ranged between 13.00-96.00 cmH_2O; with a mean (±S.D.) of 54.80±18.89 cmH_2O (Table 1).

The PImax of the controls ranged between 62.00-104.00 cmH_2O, with a mean (±S.D.) of 80.40±7.51 cmH_2O while their PEmax ranged between 76.00-160.00 cmH_2O, with a mean (±S.D.) of 95.44±12.52 cmH_2O (Table 1).

The mean PImax and mean PEmax of the COPD patients were significantly lower (p-value<0.001) than that of the controls (Table 1).

All the COPD patients had PImax of less than 80 cmH_2O, while 98.6% had PEmax of less than 90 cmH_2O (Table 1).

Table 2 shows that the mean(±S.D.) PImax and PEmax decreased as the severity of COPD worsens from mild to very severe. The difference between the mean PImax and PEmax at the different stages of COPD severity was statistically significant (p-value<0.001). Four (5.7%); patients had mild disease, 36 (51.4%) moderate disease, 17 (24.3%) severe disease and 13 (18.6%) had very severe disease (Table 2).

Both PImax and PEmax correlated positively with the FEV_1 of the COPD patients, with p-value<0.001 (Table 3). As shown in Table 3, there was a strong negative correlation (r=-0.704) between the PImax of the patients with COPD and their CAT score, while there is a moderate negative correlation (r=-0.583) between the PEmax and the CAT score of the patients with COPD.

Table 1 Sociodemographic characteristics, anthropometric characteristics, lung function profile and the maximal respiratory mouth pressures (PImax and PEmax) of the study participants

Variable	COPD patients (n=70)	Controls (n=70)	p-value
Age range (years)	45-83	42-83	
Mean age±S.D. (years)	71.28±7.50	70.92±7.94	0.785
Age group (years)			
40-49	1(1.4%)	1(1.4%)	
50-59	3(4.3%)	4(5.7%)	
60-69	19(27.1%)	30(42.9%)	
70-70	39(55.7%)	26(37.1%)	
80 and above	8(11.4%)	9(12.9%)	
Males	25(35.7%)	24(34.3%)	1.000
Females	45(64.3%)	46(65.7%)	1.000
Mean±S.D. weight (kg)	60.43±12.96	69.92±13.32	<0.001
Mean±S.D. height (m)	1.59±0.08	1.58±0.08	0.689
Mean±S.D. BMI (kg/m^2)	23.86±5.18	27.68±5.38	<0.001
FEV_1 (L)	0.99±0.51	1.71±0.48	<0.001
FEV_1 (% predicted)	53.41±20.26	89.47±16.04	<0.001
FVC (L)	1.86±0.83	2.10±0.57	0.042
FVC (% predicted)	83.12±20.88	87.60±15.39	0.152
FEV_1/FVC (%)	54.19±11.46	82.98±9.74	<0.001
PImax range (cmH_2O)	10.00-69.00	62.00-104.00	
Mean±S.D. PImax (cmH_2O)	31.78±14.40	80.40±7.51	<0.001
PImax <80 cmH_2O	70(100%)	26(37.1%)	
PImax >80 cmH_2O	0(0%)	44(62.9%)	
PEmax range (cmH_2O)	13.00-96.00	76.00-160.00	
Mean±S.D. PEmax (cmH_2O)	54.80±18.89	95.44±12.52	<0.001
PEmax <90 cmH_2O	69(98.6%)	20(28.6%)	
PEmax >90 cmH_2O	1(1.4%)	50(71.4%)	

COPD=chronic obstructive pulmonary disease, FEV_1=forced expiratory volume in 1 second, FVC=forced vital capacity, PImax=maximal inspiratory pressure, PEmax=maximal expiratory pressure, BMI=body mass index, S.D.=stanard deviation

Table 2 Relationship between maximal respiratory mouth pressures (PImax/PEmax) and the severity of airflow obstruction in patients with COPD

Variable	Mild FEV_1 ≥80% n=4(5.7%)	Moderate FEV_1 50–79% n=36(51.4%)	Severe FEV_1 30–49% n=17(24.3%)	Very severe FEV_1 <30% n=3(18.6%)	p-value
PImax (cmH_2O)	43.00±14.14	38.50±14.42	23.65±6.75	20.38±7.97	<0.001
PEmax (cmH_2O)	68.50±16.84	61.61±16.43	49.59±17.18	38.54±16.61	<0.001

COPD=chronic obstructive pulmonary disease, FEV_1=forced expiratory volume in 1 second, PImax=maximal inspiratory pressure, PEmax=maximal expiratory pressure

Table 3 Correlation between PImax, PEmax and FEV_1, FVC and CAT

Variable	PImax		PEmax	
	Spearman's correlation	Significant (2-tailed)	Spearman's correlation	Significant (2-tailed)
FEV_1 (% Predicted)	0.658	<0.001	0.534	<0.001
FVC (% Predicted)	0.405	0.001	0.281	0.018
CAT	−0.704	<0.001	−0.583	<0.001

CAT=COPD assessment test, FEV_1=forced expiratory volume in 1 second, FVC=forced vital capacity, PImax=maximal inspiratory pressure, PEmax=maximal expiratory pressure

Table 4 Association between maximal respiratory mouth pressures (PImax/PEmax) and the level of dyspnea in patients with COPD

Variable	mMRC Grade 0 n=6	mMRC Grade 1 n=19	mMRC Grade 2 n=16	mMRC Grade 3 n=25	mMRC Grade 4 n=4	p-value
PImax (cmH_2O)	56.00±8.20	40.53±8.83	28.38±11.87	23.12±11.10	21.75±4.72	<0.001
PImax (cmH_2O)	74.50±9.00	65.32±14.41	56.88±15.58	43.68±17.67	36.50±12.12	<0.001

COPD=chronic obstructive pulmonary disease, PImax=maximal inspiratory pressure, PEmax=maximal expiratory pressure, mMRC=modified medical research council

Table 5 Association of CAT with FEV_1, mMRC, BMI, PImax and PEmax using multiple linear regression

Variable	Beta	95% Confidence limits		p-value
		Lower bound	Upper bound	
FEV_1	−0.149	−0.142	0.023	0.157
mMRC	0.562	2.380	5.835	<0.001
BMI	0.145	0.027	0.426	0.027
PImax	−0.219	−0.227	−0.018	0.022
PEmax	−0.039	−0.087	0.054	0.645

CAT=COPD assessment test, FEV_1=forced expiratory volume in 1 second, PImax=maximal inspiratory pressure, PEmax=maximal expiratory pressure, mMRC=modified medical research council, BMI=body mass index

Table 4 shows that both mean(±S.D.) PImax and PEmax progressively decreased as the performance on the modified MRC dyspnea scale worsens, from Grade 0 to 4. The difference between the mean PImax and PEmax at the different levels of the modified MRC dyspnea scale was statistically significant, (p-value<0.001).

Table 5 shows the association of CAT with FEV_1, mMRC, BMI, PImax, PEmax using a multiple linear regression model. The fitted model produced an adjusted R-square of 0.735, which implies that 73.5% of the variability of CAT scores is predicted by FEV_1, mMRC, BMI, PImax and PEmax.

The result showed that mMRC, BMI and PImax were significantly associated with CAT scores. For every increase in mMRC grades, the CAT score increases by 0.562, at a p-value<0.001, after controlling for the effects of FEV_1, BMI, PImax and PEmax. Similarly, an increase in BMI increases the CAT score by 0.145, at a p-value of 0.027, after controlling for the effects of FEV_1, mMRC, PImax and PEmax. On the other hand, CAT score reduces by 0.219 for every unit rise in PImax, at a p-value of 0.022, after controlling for the effects of FEV_1, BMI, mMRC and PEmax. Although, with every unit increase in FEV_1 and PE max, the CAT reduces by 0.149 (p-value of 0.157) and 0.039 (p-value of 0.645), respectively, after controlling for the effects of BMI, mMRC and PImax; their association with CAT is not statistically significant.

Discussion

The most widely applied tests of global inspiratory and expiratory muscle strength are the maximal inspiratory and expiratory pressures (PImax and PEmax), which can be measured using a mouth pressure meter[7,8,11]. The use of a mouth pressure meter, as a means of assessing the respiratory mouth pressure, was first reported by Black and Hyatt in the late 1960s[8]. The mouth pressure meter has been compared with other standard laboratory tests used in the assessment of respiratory muscle strength, and it is a validated means of measuring the maximal respiratory mouth pressure, both accurately and reliably[7].

The mean PImax and mean PEmax of the COPD patients were significantly lower, compared with the mean PImax and mean PEmax of the controls. This has been reported by other researchers in the literature[3,7,14-19]. The mean PImax and PEmax of the COPD patients in this study was however much less compared to similar studies by Nambiar et al[21] in India, Nam-Sik et al.[22] in Korea and Terzano et al.[10] in Italy. This may be a result of the age difference in the recruited patients in these studies. Furthermore, all of the COPD patients in this study had significant Inspiratory muscle weakness, and 98.6% had significant Expiratory muscle weakness. A high PImax, greater than 80 cmH_2O, or high PEmax, greater than 90 cmH_2O, rules out clinically significant inspiratory or expiratory weakness[23].

Studies have shown that the causes of respiratory muscle weakness in many patients with COPD are multifactorial, and these include pulmonary hyperinflation with increased residual volume. This shortens the inspiratory muscles; thereby, reducing the efficacy of contraction. Other factors that has been implicated in respiratory muscle weakness in COPD patients include: malnutrition, muscular atrophy, steroid-induced myopathy and reduced blood flow to the respiratory muscles[23-29]. These processes contribute to the reduced efficiency of the respiratory muscles in patients with COPD, and translates to a measurable decrease in the maximal inspiratory pressure (PImax) as well as a decrease in the maximal expiratory pressure (PEmax)[10].

This study showed a decline in the mean PImax and mean PEmax of COPD patients, as the severity of airflow obstruction worsens using the GOLD staging of the severity of COPD. In addition, the difference in the mean PImax as well as the mean PEmax at the different stages of COPD severity were statistically significant: the PImax and PEmax correlated positively with the FEV_1. These findings

are similar to those reported by Nambiar et al.[21] and Vyas et al.[31]. The decrease in the PEmax, seen in advanced COPD is as a result of the generalized muscle weakness commonly associated with it[32]. The corresponding decrease seen in the PImax as the disease severity increases could be due to hyperinflation associated with advanced COPD[21]. Pulmonary hyperinflation worsens as the airflow limitation increases. Hyperinflation shortens the inspiratory muscles, with a consequent reduction in the efficacy of contraction[29]. Respiratory muscles are very crucial to alveolar ventilation. These muscles work against increased mechanical load, due to airflow limitation and the geometric changes of the thoracic cage as a result of pulmonary hyperinflation[33].

mMRC is a standardized way of assessing dyspnea in chronic respiratory diseases[34]. In this study, as the performance on the modified MRC dyspnea scale worsened from Grade 0 to Grade 4, the mean PImax and mean PEmax of the COPD patients decreased. Additionally, the difference between the mean PImax and mean PEmax at the different levels of the modified MRC dyspnea scale was statistically significant. The mechanisms of dyspnea in COPD are complex and multifactorial[35]. Respiratory muscle dysfunction has been shown to contribute to dyspnea in patients with COPD[36]. Khalil et al.[37] measured maximal inspiratory pressure and maximal expiratory pressure in stable COPD patients, and correlated it with degrees of airway obstruction and the mMRC dyspnea scale. A significant negative correlation was found between PImax, PEmax and mMRC dyspnea scale grades.

Health-related quality of life (HRQoL) provides a holistic assessment of the impact of a disease on patients as well as the response to treatment[38]. Studies have shown that tools; such as the mMRC dyspnea scale and pulmonary function tests, are not adequate enough to determine the morbidity and limitations in patients with COPD. There is a need to assess the HRQoL of these patients in addition to mMRC and the pulmonary function tests. COPD Assessment Test (CAT) provides a simple, standardized assessment and a numerical estimate of disease impact, which is reliable across patients having various pulmonary measure parameters, and across various population groups[38].

This study showed that there was a negative correlation between the PImax as well as the PEmax and the CAT scores. This signifies worsening Health-Related Quality of Life as the respiratory muscle strength declines. Thus, respiratory muscle weakness has a negative impact on the health status of patients with COPD. This is similar to reports from the literature[4].

The 2021, GOLD guidelines have enumerated 6 guiding principles in the management of COPD. These include: relieving symptoms, improving exercise tolerance, improving health status, preventing disease progression, reducing mortality, preventing and treating exacerbations[1]. A comprehensive assessment of patients with COPD, in both the clinical and research settings, includes the assessment of dyspnea, severity of airflow limitation and HRQoL, and these have been well documented in the GOLD guidelines[1]. However, assessment of respiratory muscle strength has not been included as part of the routine assessment of patients with COPD. In view of the importance of assessment of respiratory muscle strength, it will be of added value if it is incorporated in the existing guidelines.

Similar to findings reported in many studies, conducted on respiratory muscle strength in COPD patients in other parts of the world, this study showed that there was a positive correlation between the PImax and PEmax and the severity of airflow obstruction. Therefore, the PImax as well as the PEmax can serve as prognostic indicators for patients with COPD.

Furthermore, in this study, the dyspnea grade was noted to increase as the PImax and PEmax declined. This corroborates the fact that respiratory muscle weakness may contribute to the dyspnea seen in patients with COPD.

Assessment of maximal respiratory mouth pressures (PImax and PEmax) should be an essential part of evaluation of COPD patients that have persistent dyspnea, despite optimal pharmacological treatment.

In addition, the PImax and PEmax had a negative correlation with the CAT scores of the COPD patients in this study. This further strengthens the fact that respiratory muscle weakness adds to the negative impact COPD has on the health status of these patients.

In view of the above findings, measurement of the PImax and PEmax can aid in the early identification of respiratory muscle weakness in patients with COPD. Subsequent therapeutic intervention can improve respiratory muscle strength and also improve the quality of life in these patients.

Findings from this study have clearly demonstrated that assessment of PImax and PEmax can further add value in the assessment of COPD patients.

Limitation of the study

The presence of a standard reference value for maximal inspiratory and expiratory pressures for the African population would have added more strength to this study.

A multicenter study would have also improved the strength of this study.

Conclusion

This study showed that there is significant respiratory muscle weakness, as evidenced by reduced maximal respiratory mouth pressures (PImax and PEmax), in patients with COPD compared with the controls. In addition, the respiratory muscle weakness seen in COPD patients worsened as the airflow obstruction and the level of dyspnea worsened. Furthermore, respiratory muscle weakness may also add to the negative impact COPD has on the health status of patients with COPD. It is therefore recommended that for comprehensive assessment of COPD patients, tools such as the mouth pressure meter should be used as part of the routine clinical evaluation of patients. In view of the prevalence of the burden of respiratory muscle weakness among the COPD patients, the pulmonary rehabilitation inclusive of inspiratory muscle training should form part of the routine management of patients with COPD.

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Conflict of interest

The authors declared no conflicts of interest.

References

1. 2021 global strategy for prevention, diagnosis and management of copd [homepage on the Internet]. Illinois: GOLD Inc; [cited 2021 May 27]. Available from: https://goldcopd.org/2021-gold-reports/
2. Mannino DM, Gagnon RC, Petty TL, Lydick E. Obstructive lung disease and low lung function in the united states:data from national health and nutrition examination survey, 1988-1994. Arch Intern Med 2000;160:1683–9.

3. Agusti AG. Systemic effects of Chronic Obstructive Pulmonary Disease. Proc AM Thorac So 2005;2:367–70.
4. Decramer M. The Respiratory Muscles in COPD: regulation of trophical status. Verh K Acad Geneeska Belg 2001;63:577–602.
5. Wouters E. Muscle wasting in chronic obstructive pulmonary disease to bother and to measure! Am J Respir Crit Care Med 2006;173:4–5.
6. Kabitz H-J, Walterspacher S, Walker D, Windisch W. Inspiratory muscle strength in chronic obstructive pulmonary disease depending on disease severity Inspiratory muscle strength in chronic obstructive pulmonary disease depending on disease severity. Clin Sci 2007;113:243–9.
7. Hamnegard C-H, Wragg S, Kyroussis D, Aquilina R, Moxham J, Green M. Portable measurement of maximum mouth pressures. Eur Respir J 1994;7:398–401.
8. Black LF, Hyatt RE. Maximal respiratory pressures: normal values and relationships to age and sex. Am Rev Respir Dis 1969;99:696–702.
9. ATS/ERS Statement on respiratory muscle tesing. Am J Respir Crit Care Med 2002;166:518–624.
10. Terzano C, Ceccarelli D, Conti V, Graziani E, Ricci A, Petroianni A. Maximal respiratory static pressures in patients with different stages of COPD severity. Respir Res 2008;9:1–7.
11. Leech J, Ghezzo H, Stevens D, Becklake M. Respiratory pressures and function in young adults. Am Rev Respir Dis 1983;128:17–23.
12. Bruschi C, Cerveri I, Zoia M, Fanfulla F, Fiorentini M, Casali L, et al. Reference values of maximal inspiratory mouth pressures: a population based study. Am Rev Respir Dis 1992;146:790–3.
13. Spruit MA, Singh SJ, Garvey C, Zuwallack R, Nici L, Rochester C, et al. American thoracic society documents an official american thoracic society/european respiratory society statement: key concepts and advances in pulmonary Rehabilitation Executive Summary. Am J Respir Crit Care Med 2013;188:e13–64.
14. Graham BL, Steenbruggen I, Miller MR, Barjaktarevic IZ, Cooper BG, Hall GL, et al. Standardization of spirometry 2019 update. An Official American Thoracic Society and European Respiratory Society Technical Statement. Am J Respir Crit Care Med 2019;200.
15. Rochester D. Malnutrition and the respiratory muscles. Clin Chest Med 1986;7:91–9.
16. Van Balkom RH, Zhan W-Z, Prakash Y, Richard Dekhuijzen P, Sieck GC. Corticosteroids effects on isotonic contractile properties of rat diaphragm muscle. J Appl Physiol 1997;83:1062–7.
17. Nishimura Y, Tsutsumi M, Nakata H, Tsunenari T, Maeda H, Yokoyama M. Relationship between respiratory muscle strength and lean body mass in men with copd. Chest 1995;107:1232–6.
18. Heijdra Y, Dekhuijzen P, Van Herwaarden C, Folgering H. Effects of body position , hyperinflation , and blood gas tensions on maximal respiratory pressures in patients with chronic obstructive pulmonary disease. Thorax 2014;49:453–8.
19. Agusti A, Calverley PMA, Celli B, Coxson HO, Edwards LD, Lomas DA, et al. Characterisation of COPD heterogeneity in the ECLIPSE cohort. Respir Res 2010;11:122–36.
20. Martinez-Garcia M-A, de la Rosa D, Soler-Cataluna J-J, Donat-sanz Y, Serra CP, Lerma MA, et al. Prognostic value of bronchiectasis in patients with moderate-to-severe chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2013;187:823–31.
21. Nambiar VK, Ravindra S, Nanda Kumar BS. Maximal inspiratory and expiratory pressures in men with chronic obstructive pulmonary disease: a cross-sectional study. Indian J Respir Care 2018;7:88–92.
22. Nam-Sik K, Jeong-Hwan S, Myoung-Hwan K, Sung-Hee P, Yu HW. Respiratory muscle strength in patients with chronic obstructive pulmonary disease. Ann Rehabil Med 2017;41:659–66.
23. Polkey M, Green M, Moxham J. Measurement of respiratory muscle strength. Thorax 1995;50:1131–5.
24. Sze MA, Dimitriu PA, Suzuki M, Mcdonough JE, Campbell JD, Brothers JF, et al. Host response to the lung microbiome in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2015;192:438–45.
25. Hogg JC, Timens W. The Pathology of chronic obstructive pulmonary disease. Annu Rev Pathol 2009;4:435–59.
26. Barrett Kim E, Barman Susan M, Boitano Scott, Brooks Heddwen L. Ganong's Review of Medical Physiology. 23rd ed. Singapore: McGraw-Hill; 2010. p.587–607.
27. Hogg JC, Macklem PT, WM T. Site and nature of airways obstruction in chronic obstructive lung disease. N Engl J Med 1968;278:1355–60.
28. Saetta M, Turato G, Baraldo S, Zanin A, Braccioni F, Mapp CE, et al. Goblet cell hyperplasia and epithelial inflammation in

peripheral airways of smokers with both symptoms of chronic bronchitis and chronic airflow limitation. Am J Respir Crit Care Med 2000;161:1016–21.

29. Dyspnea. Mechanisms, assessment and management: a consensus statement. American Thoracic Society. Am J Respir Crit Care Med 1999;159:321–40.
30. Hyatt R. Expiratory flow limitation. J Appl Physiol 1983;55:1–7.
31. Vyas PK, Bindroo PS, Gondha M, Mathur RS, Ghtawat G, Lakkappan V. Measurement of maximal respiratory static pressure in stable copd patients and their co-rrelation with various physiological anthropometric and spirometry parpmeters and arterial blood gas tension. Arch Pulmonol Respir Care 2020;6:33–42.
32. Rochester D, Braun N. Determinants of maximal inspiratory pressure in chronic obstructive pulmonary disease. Am Rev Respir Dis 1985;132:42–7.
33. Orozco-Levi M. Structure and function of the respiratory muscles in patients with COPD: impairment or adaptation? Eur Respir J 2003;22:S41-51.
34. Launois C, Barbe C, Bertin E, Nardi J, Perotin J, Dury S, et al. The modified Medical Research Council scale for the assessment of dyspnea in daily living in obesity: a pilot study. BMC Pulm Med 2012;12:61.
35. O'Donnell DE, Banzett RB, Carrieri-Kohlman V, Casaburi R, Davenport PW, Gandevia SC, et al. Pathophysiology of dyspnea in chronic obstructive pulmonary disease a roundtable. Proc Am Thorac Soc4 2007;4:145–68.
36. Charususin N, Dacha S, Gosselink R, Decramer M, Leupoldt A Von, Reijnders T, et al. Respiratory muscle function and exercise limitation in patients with chronic obstructive pulmonary disease: a review. Expert Rev Respir Med 2018;12:67–79.
37. Khalil M, Wagih K, Mahmoud O. Evaluation of maximum inspiratory and expiratory pressure in patients with chronic obstructive pulmonary disease. Egypt J Chest Dis Tuberc 2014;63:329–35. doi: 10.1016/j.ejcdt.2014.01.010
38. Bove DG, Lavesen M, Lindegaard B. Characteristics and health related quality of life in a population with advanced chronic obstructive pulmonary disease, a cross- sectional study. BMC Palliat Care 2020;19:1–9.