Original Research

Diaphragmatic Ultrasound for Functional Assessment in Chronic Obstructive Pulmonary Disease: A Clinical Study

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***Running Head*: Diaphragm Ultrasound Assessment in COPD**

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Abstract

Objective: To evaluate diaphragm ultrasound as an assessment tool in COPD versus healthy controls, and secondarily to explore changes after a 12-week breathing exercise program.

Methods: We enrolled 80 COPD patients and 80 age- and sex-matched healthy volunteers. The primary endpoint was baseline between-group difference in maximal diaphragmatic excursion (DEmax). Secondary outcomes included tidal excursion (DE), diaphragm thickening fraction (DTF), FEV_1/FVC, RV/TLC, DLCO/KCO, and emphysema burden (LAA%-950, Perc15). COPD patients received a 12-week breathing program; measurements were repeated.

Results: At baseline, COPD patients had lower DEmax, DTF, FEV_1/FVC, and DLCO/KCO, and higher RV/TLC and emphysema burden (all $P \leq 0.004$). After training, DEmax increased from 3.58±0.55 to 5.02±0.48 cm, DTF from 53.72±6.45% to 66.18±8.12%, FEV_1/FVC from 56.49±6.83% to 62.34±6.78%, RV/TLC decreased from 51.57±5.26% to 45.67±6.23%, DLCO from 61.34±8.42% to 65.21±8.71% predicted, and KCO from 67.75±9.22% to 71.43±9.56% predicted (all $P = 0.018$–0.032; DE unchanged, $P = 0.376$). Post-training DEmax, DTF, FEV_1/FVC, and RV/TLC remained worse than controls ($P = 0.009$–0.044); DLCO/KCO were not separately compared. DEmax and DTF correlated with FEV_1/FVC, RV/TLC, DLCO, KCO, LAA%-950, and Perc15 ($P < 0.001$). Analyses were not adjusted for multiple comparisons.

Conclusion: Diaphragm ultrasound parameters are associated with pulmonary function, diffusion impairment, and emphysema burden, and changed after breathing training in this uncontrolled cohort. However, these findings should be considered exploratory and do not

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establish efficacy, routine implementation, or a minimal clinically important difference. Diaphragm ultrasound may be a feasible adjunct to spirometry pending validation in randomized controlled trials.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a major global health burden, affecting over 300 million people worldwide and ranking as the third leading cause of death [1]. It is characterized by persistent, often progressive, airflow limitation resulting from chronic airway inflammation and parenchymal destruction [2]. The disease not only impairs pulmonary ventilation but also induces systemic consequences, among which respiratory muscle dysfunction—particularly of the diaphragm—plays a pivotal role in disease progression and disability [3]. The diaphragm, as the principal inspiratory muscle, undergoes significant mechanical disadvantage in COPD due to chronic hyperinflation, which flattens the muscle, shortens its fibers, and reduces its force-generating capacity [4]. This leads to a vicious cycle of increased work of breathing, dyspnea, and reduced exercise tolerance, ultimately contributing to poor quality of life and increased mortality.

Conventionally, pulmonary function testing (PFT) is the gold standard for diagnosing and staging COPD, yet it has notable limitations: it requires maximal patient effort, does not provide regional information, and cannot specifically assess diaphragmatic function [5]. In recent years, ultrasound has emerged as a validated, radiation-free, and bedside-available technique for evaluating diaphragmatic structure and function [6-8]. Diaphragmatic ultrasound can quantify excursion (DE), thickening fraction (DTF), and maximal excursion (DEmax), which correlate with respiratory muscle strength and pulmonary function in various patient populations [9]. Several studies have demonstrated that diaphragmatic ultrasound parameters are reduced in

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COPD and may improve after rehabilitation [10-12]. More recent systematic reviews have confirmed the utility of diaphragm ultrasound in assessing respiratory muscle dysfunction and monitoring training effects in COPD [6, 10]. However, evidence regarding the responsiveness of these parameters to breathing exercises and their correlation with standard spirometric indices remains limited, and many studies lack detailed methodological reporting.

In addition, previous studies have often characterized COPD using spirometry alone, without incorporating diffusion capacity or quantitative emphysema burden. DLCO and KCO provide information on alveolar-capillary membrane function and gas exchange efficiency [13, 14], whereas quantitative emphysema burden, including LAA%-950 and Perc15, reflects the extent and distribution of parenchymal destruction [15,16]. Both may be independently associated with diaphragm function and hyperinflation [17]. We therefore added DLCO/KCO and quantitative emphysema burden to the physiological characterization of the cohort.

Therefore, the primary aim of this study was to evaluate the value of diaphragmatic ultrasound as an assessment tool for diaphragmatic function in COPD patients compared with healthy controls. The secondary aim was to explore changes after a 12-week breathing exercise program. Because the study was uncontrolled and non-randomized, the longitudinal component was considered exploratory and hypothesis-generating. Additional aims included exploring correlations between ultrasound parameters and pulmonary function indices (FEV_1/FVC, RV/TLC, DLCO/KCO, LAA%-950, and Perc15) and assessing the potential feasibility of diaphragm ultrasound as a complementary monitoring tool. We hypothesized that

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diaphragmatic ultrasound would detect significant impairment in COPD, show measurable changes after training, and correlate with spirometric, lung-volume, diffusion, and emphysema measures.

Materials and Methods

1. Study Design and Participants

This prospective observational study was conducted from April 2023 to March 2024. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

Sample size calculation: Based on a pilot study (n = 10 per group), we assumed a between-group DEmax difference of 1.0 cm and an SD of 2.14 cm. Using a two-sample t-test with a two-sided α of 0.05 and 80% power, 72 participants per group were required. Allowing for a 10% dropout rate, we planned to enroll 80 participants per group and ultimately enrolled 80 COPD patients and 80 age- and sex-matched healthy controls. This calculation applied only to the baseline cross-sectional comparison; no formal sample-size planning was performed for longitudinal changes, and all longitudinal analyses were exploratory. The pilot study provided the basis for sample-size planning, but its effect size estimate differed substantially from the final cohort; therefore, the calculation should be interpreted as a planning estimate only.

2. Inclusion and Exclusion Criteria

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COPD diagnosis followed the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2023 criteria, confirmed by post-bronchodilator $FEV_1/FVC < 0.70$. Additional inclusion criteria for the COPD group: (1) clinically stable (no exacerbation or change in medication within the prior 4 weeks); (2) able to cooperate with ultrasound and PFT; (3) no severe psychiatric or life-threatening comorbidities. Exclusion criteria for both groups: (1) unstable vital signs or inability to complete assessments; (2) history of lung surgery; (3) pregnancy; (4) evidence of diaphragmatic paralysis (assessed by M-mode ultrasound showing paradoxical motion or absent excursion on the right side, leading to exclusion).

Healthy controls were defined as non-smokers or former smokers with <10 pack-years (no current smokers), with no respiratory symptoms, no known cardiopulmonary disease, and normal spirometry ($FEV_1/FVC \geq 0.70$ and $FEV_1 \geq 80\%$ predicted).

3. Baseline Clinical Data Collection

For all COPD participants, we recorded age, sex, BMI, smoking history (pack-years), current smoking status separately from former smoking, GOLD stage (I-III), Charlson Comorbidity Index, mMRC dyspnea score, current medications (long-acting bronchodilators, inhaled corticosteroids, oxygen use), and number of exacerbations in the past year. The exact numbers of current and former smokersare now reported in Table 1 (current smokers: 18 [22.5%] in COPD vs. 0 [0%] in controls, $P < 0.001$; former smokers: 36 [45.0%] vs. 28 [35.0%], $P = 0.198$). DLCO/KCO and quantitative emphysema burden (LAA%-950 and Perc15) were added to the baseline characterization.

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4. Diaphragmatic Ultrasonography Protocol

All ultrasound examinations were performed by two experienced sonographers (with >5 years of thoracic ultrasound experience) using a LOGIQ E9 system (GE Healthcare, USA) with a 3.5-5 MHz curvilinear probe (for excursion) and a 9-12 MHz linear probe (for thickness). The sonographers were blinded to the participants' group assignment (COPD vs. control) but not to the time point (pre- vs. post-training) for logistical reasons. Complete blinding to disease status is difficult in practice when scanning symptomatic COPD patients, and blinding to time point was not feasible in this study. Future studies should use independent sonographers blinded to both group and time point where possible. To assess reliability, we performed intra- and inter-observer reproducibility analyses on 10 randomly selected subjects; the intraclass correlation coefficients (ICC) for all parameters were >0.85, indicating excellent agreement. Examination time, technical failure rate, and the training required for non-ultrasonographers were not systematically recorded; this is acknowledged as a limitation for evaluating routine clinical feasibility.

Patients were examined in a supine position with the head elevated 30°. To reduce the potential influence of dynamic hyperinflation caused by posture change, participants rested in the supine position for at least 5 minutes before scanning, and all measurements were performed using a standardized sequence and timing. The right hemidiaphragm was used because the liver provides a consistent acoustic window [18].

4.1. DE and DEmax Measurement:

Using the curvilinear probe placed in the subcostal region at the mid-clavicular line, M-mode

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was activated to record diaphragmatic displacement during tidal breathing (DE) and during deep inspiration to total lung capacity (DEmax). The vertical distance of the bright echogenic line was measured in centimeters. Three consecutive cycles were averaged. Representative M-mode images of DE and DEmax measurements are shown in Figure 1.

4.2. Tdi and DTF Measurement:

With the linear probe placed perpendicular to the chest wall at the 8th-9th intercostal space in the anterior axillary line, diaphragm thickness was measured at end-expiration (Tdi_exp) and at end-inspiration (Tdi_insp). DTF was calculated as [(Tdi_insp – Tdi_exp) / Tdi_exp] × 100%. This approach, using end-expiratory thickness as the denominator, is the most commonly reported method in the literature and reflects the fractional change in thickness during inspiration, which correlates with diaphragmatic contractile activity [19, 20]. Three measurements were averaged. Representative B-mode images of Tdi and DTF measurements are shown in Figure 2.

5. Pulmonary Function Testing, DLCO/KCO, and Quantitative Emphysema Burden

PFT (Jaeger MasterScreen, Germany) was performed according to ATS/ERS standards, measuring FVC, FEV_1, RV, and TLC. Lung volumes, including RV and TLC, were measured by body plethysmography using the Jaeger MasterScreen system, not derived from conventional spirometry. Daily calibration was performed according to the manufacturer's instructions. Technical acceptability criteria followed ATS/ERS standards. Reference equations used were those built into the Jaeger MasterScreen system according to the laboratory's standard

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protocol.DLCO and KCO were measured using the single-breath carbon monoxide diffusing capacity technique (Jaeger MasterScreen, Germany) according to ATS/ERS standards. KCO was calculated as DLCO divided by alveolar volume (VA). Results were corrected for hemoglobin according to standard equations. The reference equations used were those built into the Jaeger MasterScreen system according to laboratory routine.

Quantitative emphysema burden was assessed by chest computed tomography (CT) using 1 mm slice thickness and a soft tissue reconstruction algorithm. Two metrics were derived: the percentage of low-attenuation areas below −950 Hounsfield units (LAA%-950) and the 15th percentile of the lung attenuation distribution (Perc15). CT scans were analyzed using 3D Slicer v4.11. Scans performed within 3 months before baseline were used. CT was available in 74 COPD patients (92.5%) and 76 controls (95.0%).

All tests were conducted post-bronchodilator (400 µg salbutamol) both at baseline and after the intervention, using the same equipment and protocol. The ratios FEV_1/FVC and RV/TLC were recorded. DLCO/KCO was repeated after the 12-week intervention. Quantitative emphysema burden was not repeated because it was not expected to change within 12 weeks.

6. Pulmonary Rehabilitation Protocol

The intervention was a 12-week structured breathing exercise program, not a comprehensive pulmonary rehabilitation (which typically includes exercise training and education). The 12-week duration, frequency, and session length were adapted from previous inspiratory muscle training and breathing exercise studies [21–23]. However, this exact protocol has not been

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independently validated in a randomized controlled trial. It consisted of:

(1) Diaphragmatic breathing: patients practiced in a comfortable position, placing one hand on the abdomen and the other on the chest. They inhaled slowly through the nose, pushing the abdomen outward, and exhaled through pursed lips with abdominal contraction. Each session lasted 15 minutes, performed three times daily.

(2) Pursed-lip breathing: after nasal inhalation, patients exhaled slowly through slightly pursed lips, prolonging expiration to 2-3 times the inspiratory time. Same duration and frequency.

All patients attended weekly 1-hour supervised group sessions at the outpatient clinic for instruction and correction, and performed home practice with daily logs. This represented approximately 90 minutes of prescribed breathing practice per day, which is a substantial intervention burden. Adherence was monitored by diary review and weekly telephone calls, not by objective electronic monitoring; mean adherence (completed sessions) was 92%. This may overestimate the true delivered dose. No changes in pharmacological therapy were permitted during the study.

7. Statistical Analysis

Data are expressed as mean ± SD. Between-group comparisons used Mann-Whitney U test; within-group pre-vs-post comparisons used Wilcoxon signed-rank test. The effect size r with 95% confidence intervals (95%CI) was reported for nonparametric comparisons. For Mann–Whitney U and Wilcoxon signed-rank tests, r was calculated as $r = Z/\sqrt{N}$, with the sign retained to indicate direction; 95%CIs were obtained by bootstrapping with 1000 resamples. Spearman's

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correlation was used for associations between ultrasound and PFT parameters using baseline data only to avoid repeated-measure dependency, with 95%CI calculated for each correlation coefficient using Fisher's z transformation.

The original analysis did not include a linear mixed-effects model or formal adjustment for multiple comparisons. Therefore, all longitudinal findings are exploratory, and P values should be interpreted with caution. Effect sizes and 95% confidence intervals were now added for all primary comparisons and correlation analyses. The primary endpoint was defined as the baseline between-group difference in DEmax; secondary endpoints were changes in DTF, FEV_1/FVC, RV/TLC, and DLCO/KCO. The longitudinal change in DEmax was an exploratory endpoint. All analyses were performed with SPSS 25.0, and $P < 0.05$ was considered statistically significant before any adjustment; no FDR or Bonferroni adjustment was applied in the original analysis.

Result

1. Baseline Characteristics

The COPD and control groups were well matched for age, sex, and BMI (all P = 0.478–0.832, Table 1). Effect sizes (r) and 95% CIs for primary baseline between-group comparisons are shown in Table 1. In the COPD group, the mean FEV_1% predicted was 55.3 ± 12.6%, with GOLD distribution: stage I (n = 22), stage II (n = 38), stage III (n = 20). Current smokers numbered 18 (22.5%) in the COPD group versus 0 (0%) in controls ($P < 0.001$), and former smokers numbered 36 (45.0%) versus 28 (35.0%) (P = 0.198). Current or former smokers numbered 54 (67.5%) versus 28 (35.0%) ($P < 0.001$). Mean Charlson score was 2.1 ± 1.3,

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mMRC ≥ 2 in 65%, and all patients were on long-acting bronchodilators. No patient used home oxygen. Baseline DLCO/KCO and quantitative emphysema burden are now reported in Table 1. The COPD group had lower DLCO/KCO and higher LAA%-950 and lower Perc15 than controls (DLCO: 61.34 ± 8.42 vs. 93.16 ± 7.68% predicted, $P < 0.001$; KCO: 67.75 ± 9.22 vs. 91.67 ± 8.43% predicted, $P < 0.001$; LAA%-950: 34.13 ± 6.54% vs. 8.43 ± 4.23%, $P < 0.001$; Perc15: −922.35 ± 17.42 HU vs. −856.32 ± 14.46 HU, $P < 0.001$). Corresponding post-hoc effect sizes and 95% CIs are provided in Table 1. CT was available in 74 COPD patients (92.5%) and 76 controls (95.0%).

2. Diaphragmatic and Pulmonary Function Outcomes

At baseline, tidal DE did not differ between groups ($P = 0.325$), but DEmax and DTF were significantly lower in COPD ($P = 0.002$ and 0.003). Representative ultrasound images are shown in Figures 1 and 2. Similarly, FEV_1/FVC was lower and RV/TLC higher in COPD ($P = 0.001$ and 0.004, respectively). DLCO/KCO was lower and quantitative emphysema burden was higher in COPD (Table 1).

The pre- and post-training changes within the COPD group and the post-training comparisons with controls are summarized in Table 2. After 12 weeks of training, the COPD group showed significant improvements: DEmax increased from 3.58±0.55 to 5.02±0.48 cm ($P = 0.023$); DTF increased from 53.72±6.45% to 66.18±8.12% ($P = 0.018$); FEV_1/FVC increased from 56.49±6.83% to 62.34±6.78% ($P = 0.031$); RV/TLC decreased from 51.57±5.26% to 45.67±6.23% ($P = 0.027$). DLCO changed from 61.34 ± 8.42% predicted to 65.21 ± 8.71%

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predicted (P = 0.026). KCO changed from 67.75 ± 9.22% predicted to 71.43 ± 9.56% predicted (P = 0.032). DE remained unchanged (P = 0.376). Effect sizes (r) and 95%CI for within-group changes: DEmax (r = 0.25, 95%CI 0.03–0.45), DTF (r = 0.27, 95%CI 0.05–0.47), FEV_1/FVC (r = 0.24, 95%CI 0.02–0.44), RV/TLC (r = −0.26, 95%CI −0.46 to −0.04), DLCO (r = 0.25, 95%CI 0.03–0.45), KCO (r = 0.24, 95%CI 0.02–0.44). These changes are exploratory and may reflect measurement variability, learning effects, differences in inspiratory effort, regression to the mean, positional change with dynamic hyperinflation, or other procedural effects, in addition to any true treatment effect. No adjustment for multiple comparisons was applied.

Baseline between-group effect sizes and 95%CI: DEmax (r = −0.33, 95%CI −0.49 to −0.14), DTF (r = −0.32, 95%CI −0.48 to −0.13), FEV_1/FVC (r = −0.40, 95%CI −0.55 to −0.22), RV/TLC (r = 0.31, 95%CI 0.12–0.47), DLCO (r = −0.58, 95%CI −0.70 to −0.43), KCO (r = −0.54, 95%CI −0.67 to −0.38). Post-training COPD values remained significantly worse than controls for DEmax, DTF, FEV_1/FVC, and RV/TLC (P = 0.038, 0.042, 0.044, and 0.009, respectively) (Table 2). Post-training COPD vs control comparisons: DEmax (r = -0.24, 95% CI -0.44 to -0.02), DTF (r = -0.21, 95% CI -0.41 to -0.01), FEV_1/FVC (r = -0.20, 95% CI -0.40 to -0.01), RV/TLC (r = 0.29, 95%CI 0.09–0.46). (DE: P = 0.463, r = -0.07, 95% CI -0.28–0.15.) Post-training DLCO/KCO values in the COPD group were not separately compared with controls in the original analysis; this is acknowledged as a limitation.

Exploratory analyses examining the relationship between baseline disease severity and change in DEmax/DTF were limited by sample size. Subgroup analyses by FEV_1, GOLD stage,

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hyperinflation, DLCO/KCO, and quantitative emphysema burden were not performed because of incomplete data and limited statistical power; this is acknowledged as a limitation. The updated correlation analysis in Table 3 provides exploratory evidence that DEmax/DTF are associated with DLCO/KCO and emphysema burden.

Correlation analyses were performed using baseline data only to avoid repeated-measure dependency. The updated correlation results (r and 95%CI) are presented in Table 3. DEmax was significantly correlated with FEV_1/FVC (r = 0.656, 95%CI 0.51–0.76, P < 0.001), RV/TLC (r = −0.568, 95%CI −0.69 to −0.41, P < 0.001), DLCO % predicted (r = 0.592, 95%CI 0.44–0.71, P < 0.001), KCO % predicted (r = 0.556, 95%CI 0.39–0.68, P < 0.001), LAA%-950 (r = −0.624, 95%CI −0.74 to −0.47, P < 0.001), and Perc15 (r = 0.605, 95%CI 0.44–0.72, P < 0.001). DTF was significantly correlated with FEV_1/FVC (r = 0.532, 95%CI 0.37–0.66, P < 0.001), RV/TLC (r = −0.502, 95%CI −0.64 to −0.33, P < 0.001), DLCO % predicted (r = 0.478, 95%CI 0.30–0.61, P < 0.001), KCO % predicted (r = 0.449, 95%CI 0.27–0.59, P < 0.001), LAA%-950 (r = −0.437, 95%CI −0.58 to −0.25, P < 0.001), and Perc15 (r = 0.426, 95%CI 0.24–0.57, P < 0.001). These correlations were not adjusted for multiple comparisons and should be considered exploratory.

Discussion

Our study confirms that COPD patients exhibit significant diaphragmatic dysfunction compared to healthy controls, as evidenced by reduced DEmax and DTF, which is consistent with previous reports [10, 12]. The most plausible mechanism is chronic hyperinflation, which

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flattens the diaphragm, shortens sarcomere length, and places the muscle at a mechanical disadvantage, reducing its force-generating capacity [4, 5]. Additionally, systemic inflammation, malnutrition, and corticosteroid use may contribute to muscle atrophy and weakness [24, 25]. Glucocorticoid-induced myopathy is a well-recognized complication in COPD, characterized by preferential atrophy of type IIx/b fibers and reduced mitochondrial function [24], while nutritional depletion further compromises respiratory muscle mass and contractility [25]. Sarcopenia and generalized skeletal muscle dysfunction were not assessed in this study and may be important contributors to diaphragm thickness and function; this is a limitation. The preservation of tidal DE likely reflects compensatory recruitment of accessory respiratory muscles (sternocleidomastoid, scalenes) to maintain resting ventilation, a well-described phenomenon in COPD [26].

The addition of DLCO/KCO and quantitative emphysema burden strengthens the physiological characterization of the cohort. Lower DLCO/KCO indicates impaired alveolar–capillary gas exchange, whereas higher LAA%-950 and lower Perc15 reflect greater emphysema burden. Both may contribute to hyperinflation, loss of elastic recoil, and altered diaphragm mechanics. In our cohort, DEmax and DTF were significantly associated with DLCO/KCO, LAA%-950, and Perc15, suggesting that diffusion impairment and parenchymal destruction are relevant to diaphragm function in COPD.

The significant post-training improvements in DEmax and DTF indicate that diaphragmatic dysfunction is at least partially reversible with targeted breathing exercises. These gains are

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likely due to enhanced neuromuscular efficiency, improved contractility, and increased endurance, as reported in inspiratory muscle training studies [21, 22]. However, because this was an uncontrolled, non-randomized study without a COPD control group receiving usual care, sham training, or an alternative intervention, regression to the mean, repeated-testing effects, temporal variation, changes in patient behavior, and other unmeasured factors cannot be distinguished from a true treatment effect. Therefore, the post-intervention results should be interpreted as exploratory and hypothesis-generating. The incomplete recovery compared to healthy controls suggests that structural remodeling (e.g., fibrosis, fiber type shift) may limit full restoration, especially in more advanced disease. Our post-training pulmonary function values show modest improvements in FEV_1/FVC and RV/TLC, which are physiologically plausible and align with the magnitude of change observed in prior rehabilitation trials (e.g., Crimi et al. reported a FEV_1/FVC increase from ~54% to ~60%) [10]. However, the possibility that positional change from walking to sitting to entering the examination couch, dynamic hyperinflation, or differences in inspiratory effort contributed to these changes cannot be fully excluded.

It is noteworthy that our breathing exercise program, which consisted solely of diaphragmatic and pursed-lip breathing without whole-body exercise training, yielded significant but modest improvements—smaller than those typically reported in comprehensive pulmonary rehabilitation that includes cycle ergometry or treadmill training. This observation aligns with findings from comparative effectiveness studies, which have demonstrated that comprehensive pulmonary

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rehabilitation produces larger gains in exercise capacity and health-related quality of life than isolated inspiratory muscle training alone [23]. This suggests that while isolated respiratory muscle training can improve diaphragmatic function, the addition of aerobic and resistance exercise may be necessary to achieve larger gains in lung function and exercise capacity. Our results are comparable to those of Beaumont et al. (2018), who found that inspiratory muscle training alone improved diaphragm strength but had less impact on FEV_1 than combined programs [21]. Future studies should compare the incremental benefit of adding physical exercise to breathing training.

Regarding DTF measurement, we adopted the commonly used formula with end-expiratory thickness as the denominator, which reflects the fractional thickening during inspiration. Some authors have advocated using thickness at total lung capacity as the reference to avoid variability due to lung volume changes. However, our method has been validated against invasive transdiaphragmatic pressure measurements and is more practical for bedside use. This approach aligns with the majority of COPD ultrasound literature, facilitating direct comparison with previous work. We acknowledge that standardization of DTF calculation remains a topic of debate [27], and future consensus guidelines would help harmonize methodologies across centers.

We also observed significant correlations between ultrasound parameters and spirometric indices, reinforcing the interdependence between diaphragm function and global respiratory mechanics. The updated analysis further showed that DEmax and DTF were correlated with

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DLCO, KCO, LAA%-950, and Perc15, indicating that diaphragm ultrasound parameters reflect not only airflow obstruction and hyperinflation but also diffusion impairment and emphysema burden. These associations are consistent with recent cross-sectional studies demonstrating moderate to strong correlations between DEmax/DTF and FEV_1/FVC in COPD populations [28]. However, the healthy control group was matched only for age and sex and differed fundamentally from COPD patients in smoking exposure, comorbidity, symptoms, and medication exposure. Therefore, differences in diaphragm parameters cannot necessarily be attributed specifically to COPD severity. Smoking exposure, nutritional status, corticosteroid exposure, physical activity, and comorbidity are plausible confounders. All current smokers were in the COPD group; control participants were either non-smokers or former smokers with <10 pack-years. No formal adjustment for confounders was performed; therefore, cross-sectional comparisons should be interpreted as unadjusted comparisons between clinically different populations. Our findings support the utility of diaphragm ultrasound as a surrogate marker for pulmonary impairment, but they do not imply causation. The inverse correlation between RV/TLC and DEmax/DTF highlights the detrimental effect of gas trapping on diaphragmatic mobility and thickening, consistent with the concept of thoracoabdominal asynchrony. Paulin et al. demonstrated that reduced diaphragmatic mobility is associated with lower exercise tolerance and higher dyspnea scores in COPD patients [29].

Post-hoc calculated effect sizes (r) showed that baseline differences between COPD and healthy controls were mostly moderate-to-large for pulmonary function, DLCO/KCO and

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emphysema burden, while within-group improvements after breathing training were small-to-moderate. All CIs were relatively wide, consistent with the exploratory nature and limited sample size for longitudinal endpoints. These effect sizes should be interpreted cautiously and cannot replace estimates from a dedicated randomized controlled trial.

From a clinical perspective, our findings suggest that diaphragm ultrasound may have potential to be integrated into selected COPD assessment pathways, particularly in rehabilitation settings where trained personnel and standardized protocols are available. However, this study did not demonstrate practical feasibility in routine care. Examination time, technical failure rates, and training requirements for non-ultrasonographers were not systematically recorded and require formal evaluation before routine implementation. Feasibility studies have demonstrated that diaphragm ultrasound can be successfully incorporated into outpatient respiratory care pathways, providing objective feedback that enhances patient engagement and adherence to training programs [30]. Measuring DEmax and DTF at baseline and after intervention provides objective, quantitative feedback on diaphragmatic response to training, which may help tailor exercise prescriptions and motivate patients. The test is well-tolerated, requires minimal patient cooperation (except for maximal inspiration), and can be repeated frequently without radiation exposure. We do not propose a minimal clinically important difference for DEmax based on this study.

Clinical relevance and limitations:

The clinical relevance of our findings remains to be established, as patient-reported and

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functional outcomes were not assessed. Because the study was observational and uncontrolled, longitudinal changes cannot be attributed to the breathing program alone and should be regarded as exploratory. Additional limitations include the single-center design, incomplete adjustment for confounders, partial availability of DLCO/KCO and CT emphysema metrics, subjective adherence monitoring, and limited assessment of muscle function and blinding. Effect sizes and 95% CIs were calculated post hoc, and multiple comparisons were not formally controlled.

Despite these limitations, our study suggests that diaphragm ultrasound can detect functional impairment in COPD and track changes after breathing exercises. It is practical, non-invasive, and radiation-free, and may complement spirometry by providing information on diaphragmatic mechanics. Future randomized controlled trials with longer follow-up, comprehensive outcomes, and adequate sample-size planning are warranted.

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Conflict of Interest

The authors declare no conflicts of interest.

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Table 1. Baseline characteristics of COPD group and control group

Characteristic	COPD Group (n=80)	Control Group (n=80)	P value	Effect size r	95% CI for r
Age (years)	67.45 ± 8.35	66.34 ± 8.78	0.478	–	–
Gender (male/female)	66/14	65/15	0.832	–	–
BMI (kg/m²)	21.72 ± 1.56	20.89 ± 1.78	0.562	–	–
GOLD stage (I/II/III)	22/38/20	N/A	–	–	–
Smoking (pack-years)	28.5 ± 15.2	≤10	–	–	–
Current smokers, n (%)	18 (22.5%)	0 (0%)	<0.001	–	–
Former smokers, n (%)	36 (45.0%)	28 (35.0%)	0.198	–	–
Current or former smokers, n (%)	54 (67.5%)	28 (35.0%)	<0.001	–	–
Charlson Comorbidity Index	2.1 ± 1.3	N/A	–	–	–
mMRC dyspnea score (≥2), n (%)	52 (65%)	0	–	–	–
Long-acting bronchodilators, n (%)	80 (100%)	0	–	–	–
Inhaled corticosteroids, n (%)	56 (70%)	0	–	–	–

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Characteristic	COPD Group (n=80)	Control Group (n=80)	P value	Effect size r	95% CI for r
Home oxygen use, n (%)	0	0	–	–	–
DE (cm)	1.57±0.27	1.63 ± 0.24	0.325	–	–
DEmax (cm)	3.58±0.55	6.56±0.28	0.002	−0.33	−0.49 to −0.14
DTF (%)	53.72 ± 6.45	81.36 ± 5.78	0.003	−0.32	−0.48 to −0.13
FEV_1 % predicted	55.3 ± 12.6	94.7 ± 10.2	<0.001	–	–
FVC % predicted	82.4 ± 10.5	97.2 ± 9.4	0.002	–	–
FEV_1/FVC (%)	56.49 ± 6.83	86.35 ± 5.43	0.001	−0.40	−0.55 to −0.22
RV % predicted	138.6 ± 16.3	92.5 ± 11.2	<0.001	–	–
TLC % predicted	114.7 ± 12.1	98.4 ± 8.7	0.003	–	–
RV/TLC (%)	51.57 ± 5.26	27.18 ± 4.37	0.004	0.31	0.12–0.47
DLCO (% predicted)	61.34±8.42	93.16±7.68	<0.001	−0.58	−0.70 to −0.43
KCO (DLCO/VA, % predicted)	67.75±9.22	91.67±8.43	<0.001	−0.54	−0.67 to −0.38

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Characteristic	COPD Group (n=80)	Control Group (n=80)	P value	Effect size r	95% CI for r
LAA%-950 (%)	34.13±6.54	8.43±4.23	<0.001	–	–
Perc15 (HU)	-922.35±17.42	-856.32±14.46	<0.001	–	–

Footnote: Continuous variables are presented as mean ± standard deviation; categorical variables are expressed as n (%). DLCO/KCO was measured by the single-breath technique according to ATS/ERS standards. Quantitative emphysema burden was assessed by chest CT as LAA%-950 and Perc15. CT was available in a subset of participants; missing data are reported. Current and former smoking status is reported separately. Effect sizes were calculated only for primary comparisons to limit multiplicity; other comparisons are shown with P values only. r: 0.1 small, 0.3 moderate, 0.5 large effect.

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Table 2. Pre–post changes within the COPD group and post-training comparisons with controls

Group	DE (cm)	DEmax (cm)	DTF (%)	FEV_1/FVC (%)	RV/TLC (%)	DLCO (% predicted)	KCO (% predicted)
COPD Pre-treatment	1.57 ± 0.27	3.58 ± 0.55	53.72 ± 6.45	56.49 ± 6.83	51.57 ± 5.26	61.34 ± 8.42	67.75 ± 9.22
COPD Post-treatment	1.59 ± 0.32	5.02 ± 0.48	66.18 ± 8.12	62.34 ± 6.78	45.67 ± 6.23	65.21 ± 8.71	71.43 ± 9.56
P (Pre vs. Post)	0.376	0.023	0.018	0.031	0.027	0.026	0.032
Effect size r (Pre vs Post)	0.10	0.25	0.27	0.24	-0.26	0.25	0.24
95% CI for r (Pre vs Post)	−0.12–0.31	0.03–0.45	0.05–0.47	0.02–0.44	−0.46 to −0.04	0.03–0.45	0.02–0.44
Control Group	1.63 ± 0.24	6.56 ± 0.28	81.36 ± 5.78	86.35 ± 5.43	27.18 ± 4.37	93.16 ± 7.68	91.67 ± 8.43
P (COPD Post vs. Control)	0.463	0.038	0.042	0.044	0.009	—	—
Effect size r (COPD Post vs Control)	−0.07	−0.24	−0.21	−0.20	0.29	—	—
95% CI for r (COPD Post vs Control)	−0.28–0.15	−0.44 to −0.02	−0.41 to −0.01	−0.40 to −0.01	0.09–0.46	—	—

Footnote: P values are unadjusted and exploratory. No correction for multiple comparisons was applied. Effect size r is reported for nonparametric comparisons (Mann–Whitney U / Wilcoxon signed-rank). Baseline between-group comparisons for FEV_1/FVC, RV/TLC, DLCO, and KCO are presented in Table 1; Table 2 focuses on within-group changes and post-training comparisons. Longitudinal changes cannot be attributed solely to the intervention because no randomized COPD control group was included. Post-training DLCO/KCO values in the COPD group were not separately compared with controls in the original analysis. CT-derived emphysema metrics

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(LAA%-950 and Perc15) were assessed at baseline only and are therefore reported in Table 1, not in this pre–post comparison table.

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Table 3. Correlations of diaphragmatic ultrasound parameters with pulmonary function, diffusing capacity, and quantitative emphysema burden

Table 3a. Pulmonary function: FEV_1/FVC and RV/TLC

Parameter	FEV_1/FVC (n=80) r	FEV_1/FVC (n=80) 95%CI	FEV_1/FVC (n=80) P	RV/TLC (n=80) r	RV/TLC (n=80) 95%CI	RV/TLC (n=80) P
DEmax	0.656	0.51–0.76	<0.001	−0.568	−0.69 to −0.41	<0.001
DTF	0.532	0.37–0.66	<0.001	−0.502	−0.64 to −0.33	<0.001

Table 3b. Diffusing capacity: DLCO and KCO

Parameter	DLCO % pred (n=80) r	DLCO % pred (n=80) 95%CI	DLCO % pred (n=80) P	KCO % pred (n=80) r	KCO % pred (n=80) 95%CI	KCO % pred (n=80) P
DEmax	0.592	0.44–0.71	<0.001	0.556	0.39–0.68	<0.001
DTF	0.478	0.30–0.61	<0.001	0.449	0.27–0.59	<0.001

Table 3c. Quantitative emphysema burden: LAA%-950 and Perc15

Parameter	LAA%-950 (n=74) r	LAA%-950 (n=74) 95%CI	LAA%-950 (n=74) P	Perc15 (n=74) r	Perc15 (n=74) 95%CI	Perc15 (n=74) P
DEmax	−0.624	−0.74 to −0.47	<0.001	0.605	0.44–0.72	<0.001
DTF	−0.437	−0.58 to −0.25	<0.001	0.426	0.24–0.57	<0.001

Footnote: Correlations are exploratory and were not adjusted for multiple comparisons. 95% confidence intervals are presented for each Spearman correlation coefficient. FEV_1/FVC, RV/TLC, DLCO, and KCO were available in 80 COPD patients; LAA%-950 and Perc15 were available in 74 COPD patients. LAA%-950 = percentage of low-attenuation areas below −950 Hounsfield units; Perc15 = 15th percentile of the lung attenuation distribution.

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Figure legends

Figure 1: Measurement of diaphragmatic excursion using a low-frequency probe (M-mode). Calipers indicate the displacement amplitude during tidal and deep inspiration.

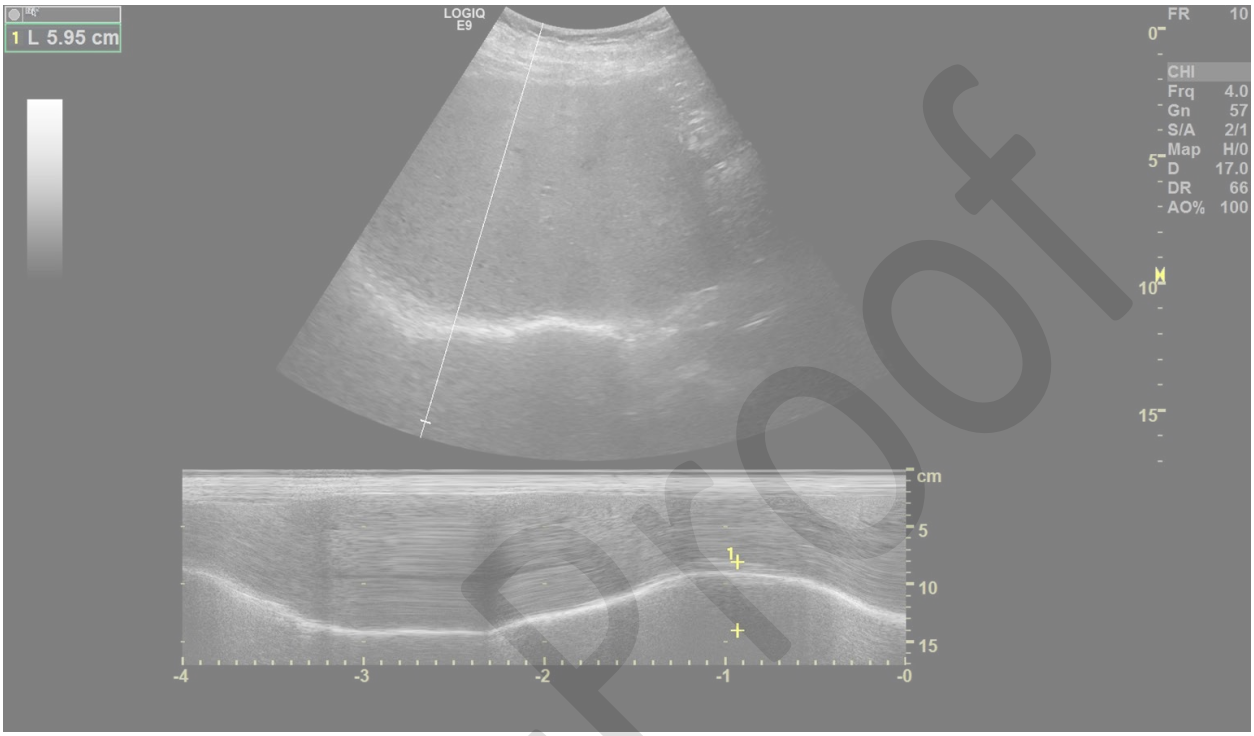

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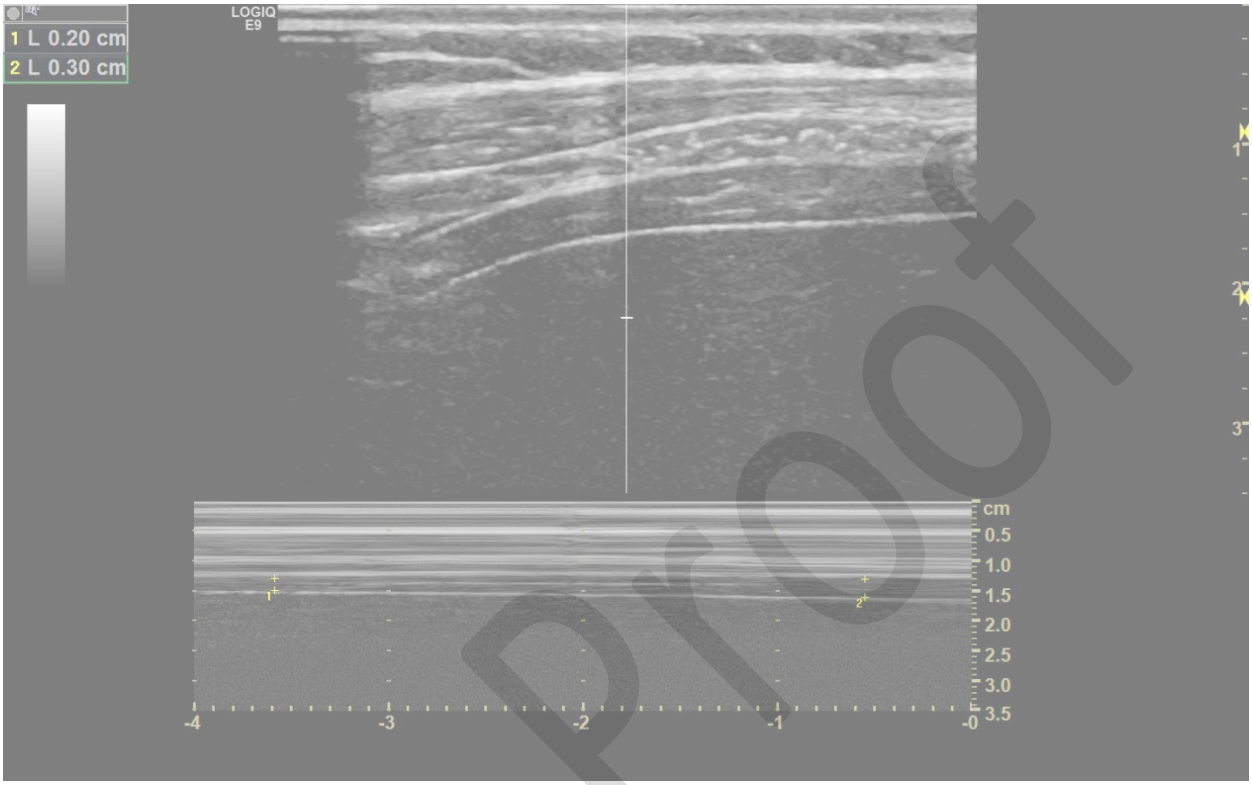
Figure 2: Measurement of diaphragm thickening fraction using a high-frequency probe. Thickness at end-expiration and end-inspiration are shown with on-screen calipers.



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