

Altered Ventilatory Mechanics and Recovery in Long COVID Patients with Dysfunctional Breathing

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ABSTRACT

Introduction: This study investigates the distinct ventilatory pattern and recovery kinetics in Long COVID (LC) patients with co-existing Dysfunctional Breathing (DB) compared to healthy controls.

Methods: We compared respiratory parameters in LC and DB patients against LC patients without DB during the post-exercise recovery period after Cardiopulmonary Exercise Testing (CPET). **Results:** LC/DB patients exhibited a significantly higher RR and lower Vt compared to controls. They also demonstrated lower VO₂/RR in the 3rd minute of recovery. Critically, while VE and PETCO₂ not differ between groups, LC/DB patients displayed poor recovery, sustaining pronounced tachypnoea (~30 breaths/minute) up to the 3rd min of the recovery phase.

Conclusion: The findings suggest that LC/DB patients employ a rapid, shallow breathing strategy. This inefficient ventilatory pattern—characterized by low Vt and, particularly, the sustained post-exertional tachypnoea—indicates a severe disruption in autonomous respiratory control (Erratic Breathing) and a potential contributor to their persistent exertional dyspnea and fatigue.

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INTRODUCTION

Dysfunctional Breathing (DB) is an increasingly well recognized cause of dyspnoea. It is observed in the general population (8%), otherwise "healthy" subjects who report unexplained shortness of breath mainly during fatigue, and in patients with asthma (about 1/3), COPD, heart failure and Long-COVID (LC). The term dysfunctional breathing (DB) refers to a spectrum of respiratory disorders that includes tachypnoea, hyperpnea, irregular respiratory rhythm and volume, use of auxiliary respiratory muscles and thoracic type of breathing, thoraco-abdominal asynchrony, etc. Cardiopulmonary Exercise Testing (CPET) is a valuable diagnostic tool as it reveals the irregular breathing patterns and hyperventilation that characterize DB along with the investigation of pathology that may miss the diagnosis and cause shortness of breath.

The purpose of the present study was to investigate respiratory recovery after maximum exercise in patients with DB and LC and to compare it with LC patients without DB.

METHODS AND MATERIALS

In this retrospective study, we reviewed the CPET data of Long COVID (LC) patients derived from two source studies: the Multifaceted Coordinated Follow-up Study of Patients Suffering from Long COVID Syndrome and the Post Covid-19 Dysautonomia Rehabilitation Randomized Controlled Trial (registered on ClinicalTrials.gov NCT05855356). We compared respiratory parameters in LC and DB patients against LC patients without DB, during the post-exercise recovery period after CPET. Measured variables included Respiratory Rate (RR), Tidal Volume (Vt), Oxygen Consumption per breath (VO₂/RR), Minute Ventilation (VE), and End-Tidal CO₂ (PETCO₂). Comparisons between the two groups were made using the Independent-Samples Mann-Whitney U Test.

RESULTS

Baseline characteristics and cardiorespiratory fitness parameters of study groups did not differ. Of LC/DB patients 20% had asthma, 12% thyroid disease (well regulated), 4% hypertension, 6% diabetes, and 35% dysautonomia. LC/DB patients exhibited **a significantly higher RR** (28.61±5.76vs24.91±3.95 breaths/min) **and lower Vt** (1.15±0.31vs1.39±0.41 lit) compared to LC without DB. They also demonstrated lower VO₂/RR (23.57±6.18vs28.05±7.84 ml/min/breath) in the 3rd minute of recovery. Critically, while VE and PETCO₂ not differ between groups, LC/DB patients displayed poor recovery, sustaining pronounced tachypnoea (~30 breaths/minute) up to the third minute of the recovery phase. Lung function tests were within normal range and not different between groups.

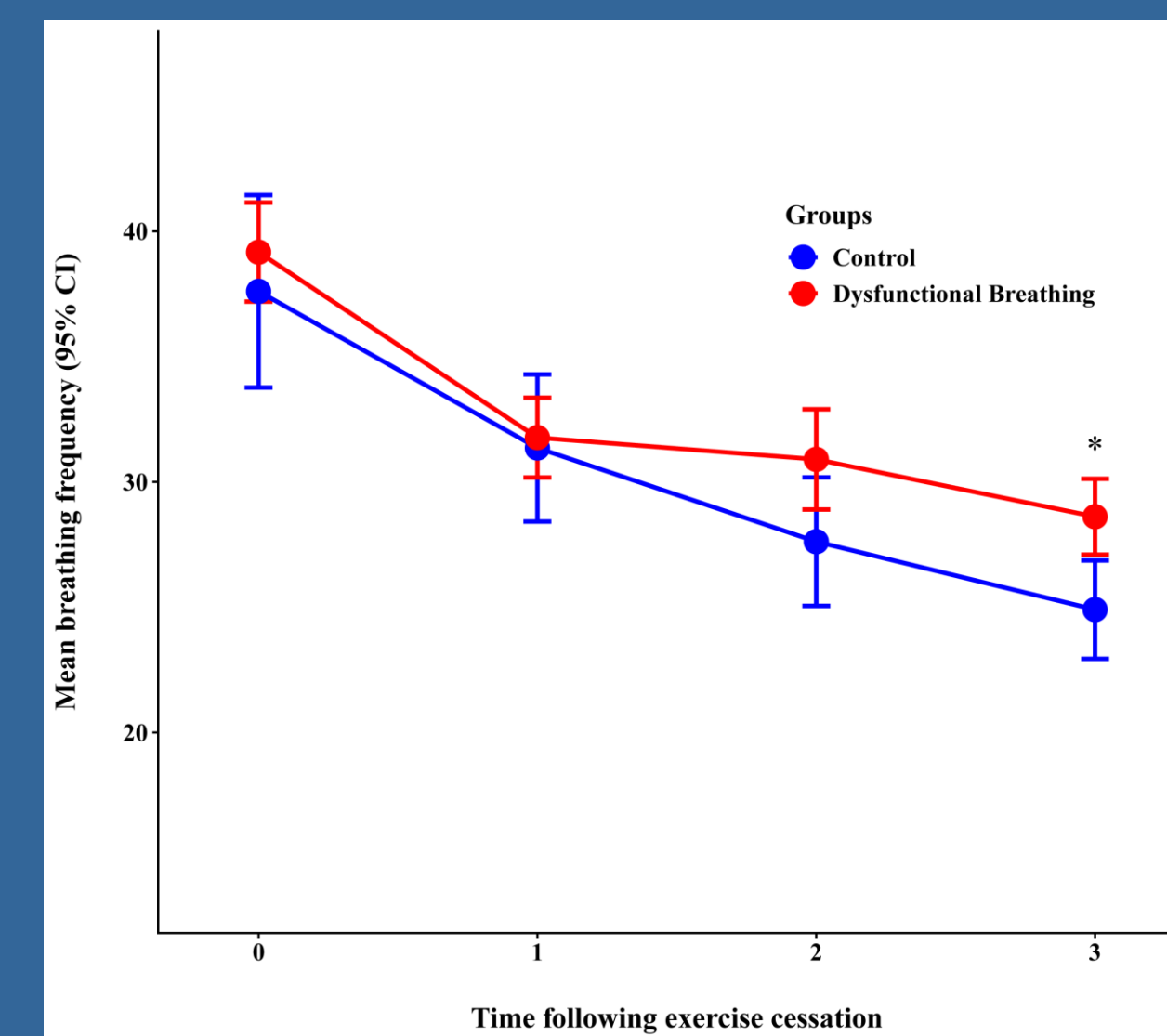


Figure 1. Mean Breathing Frequency following exercise cessation over four time points (min). The asterisk (*) indicates a statistically significant difference between groups (p<0,05)

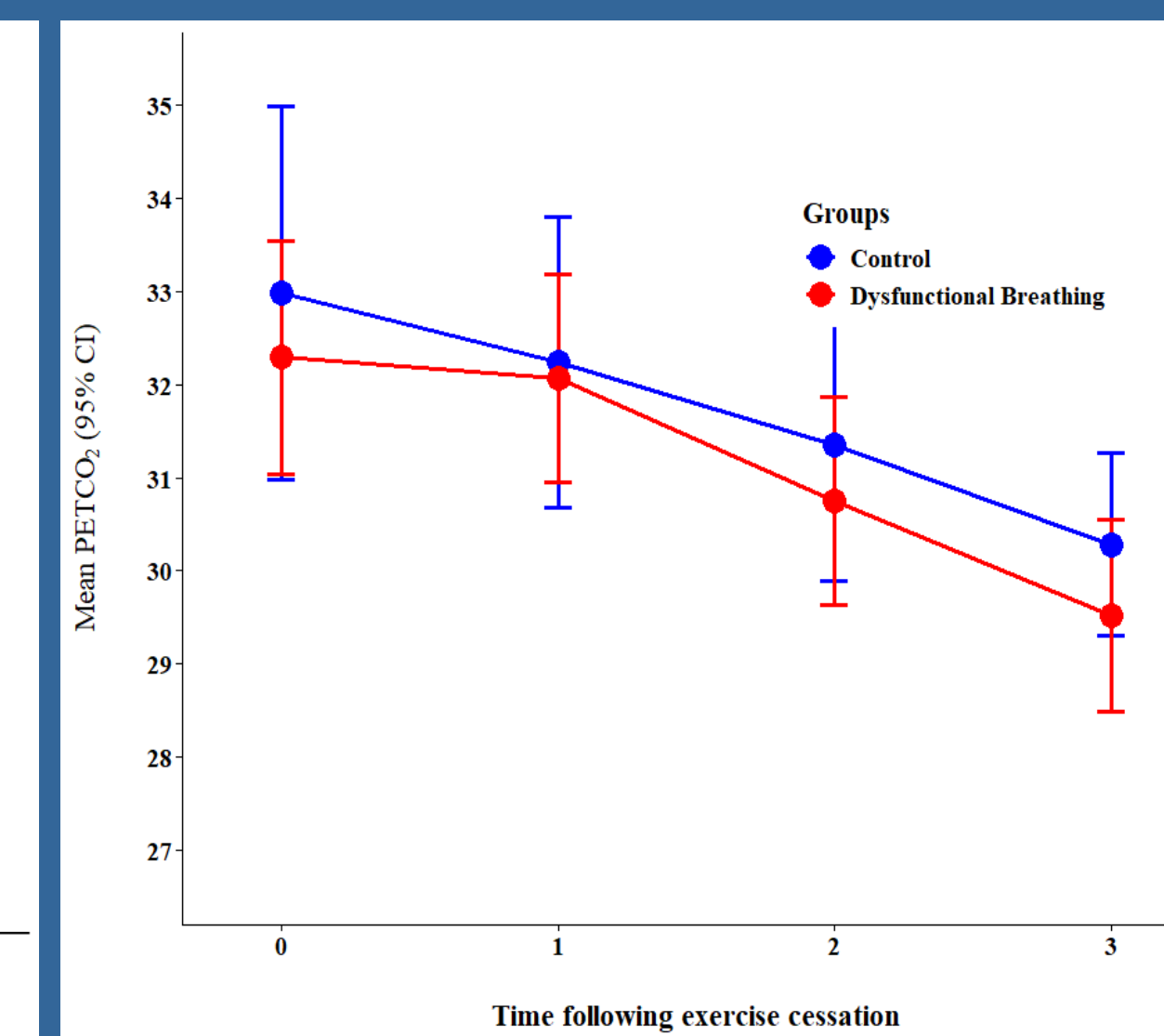


Figure 2. Mean End Tidal CO₂ following exercise cessation over four time points (minutes).

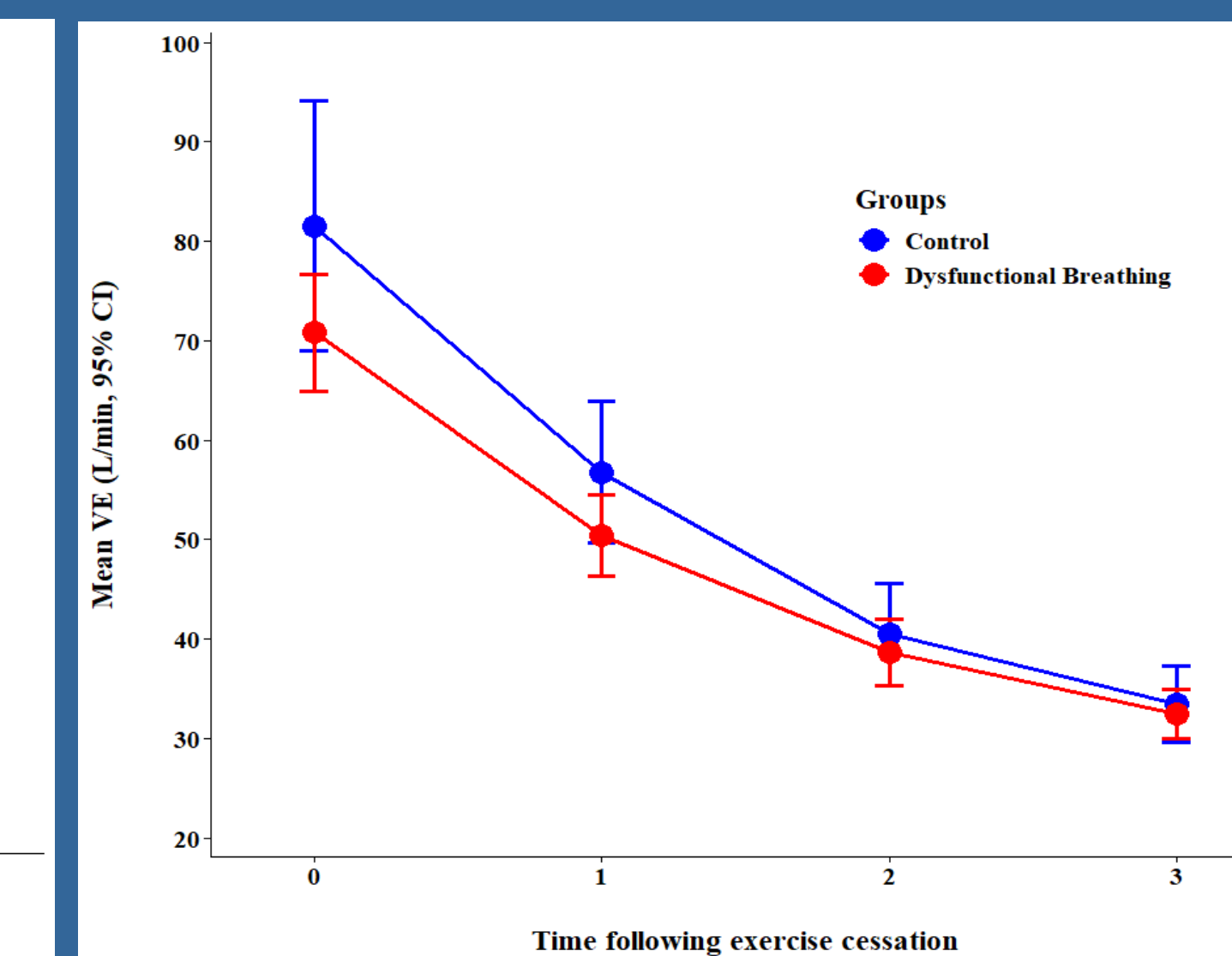


Figure 3. Mean Minute Ventilation following exercise cessation over four time points (minutes).

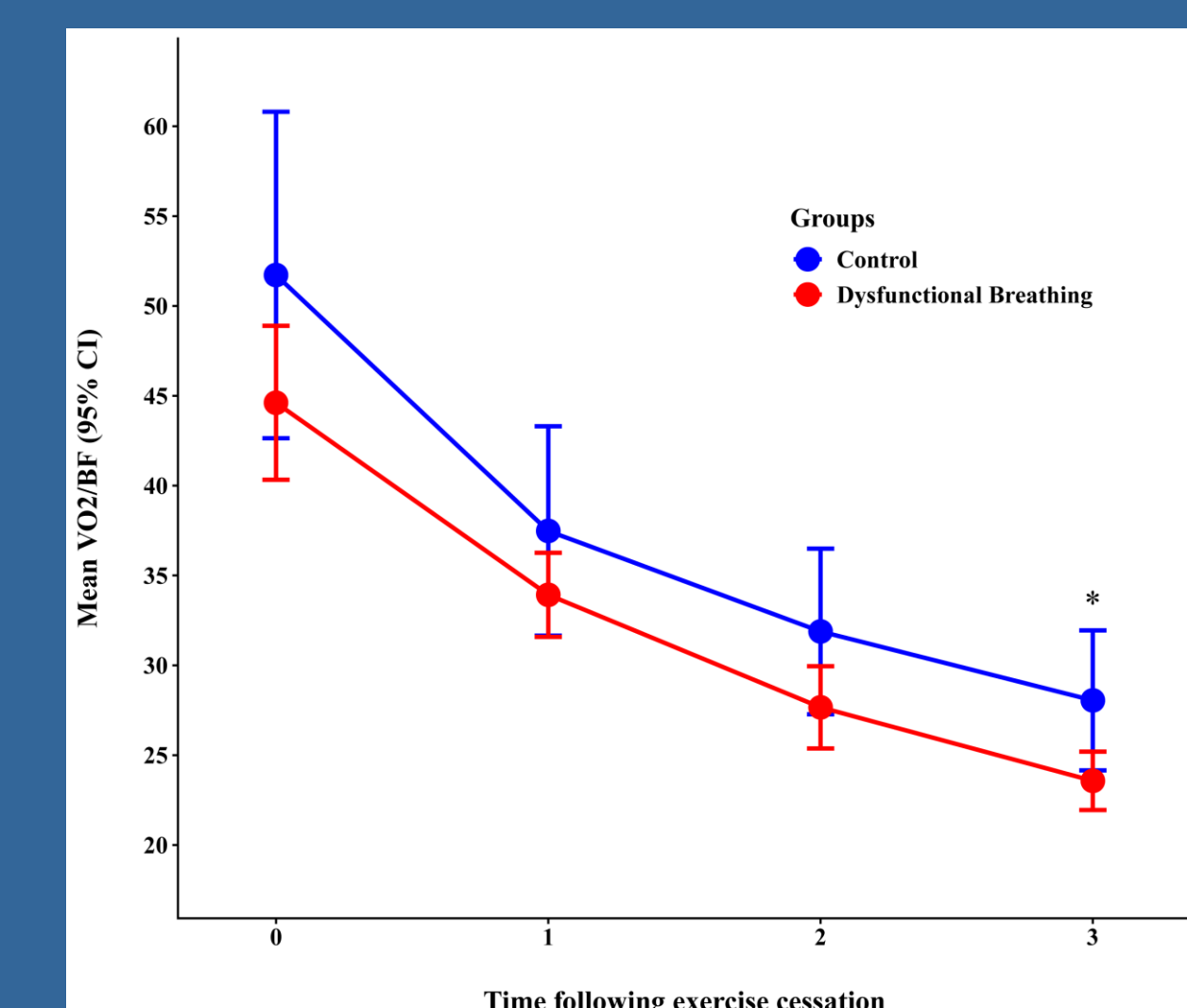


Figure 3. Mean VO₂ to BF following exercise cessation over four time points (min). The asterisk (*) indicates a statistically significant difference between groups (p<0,05)

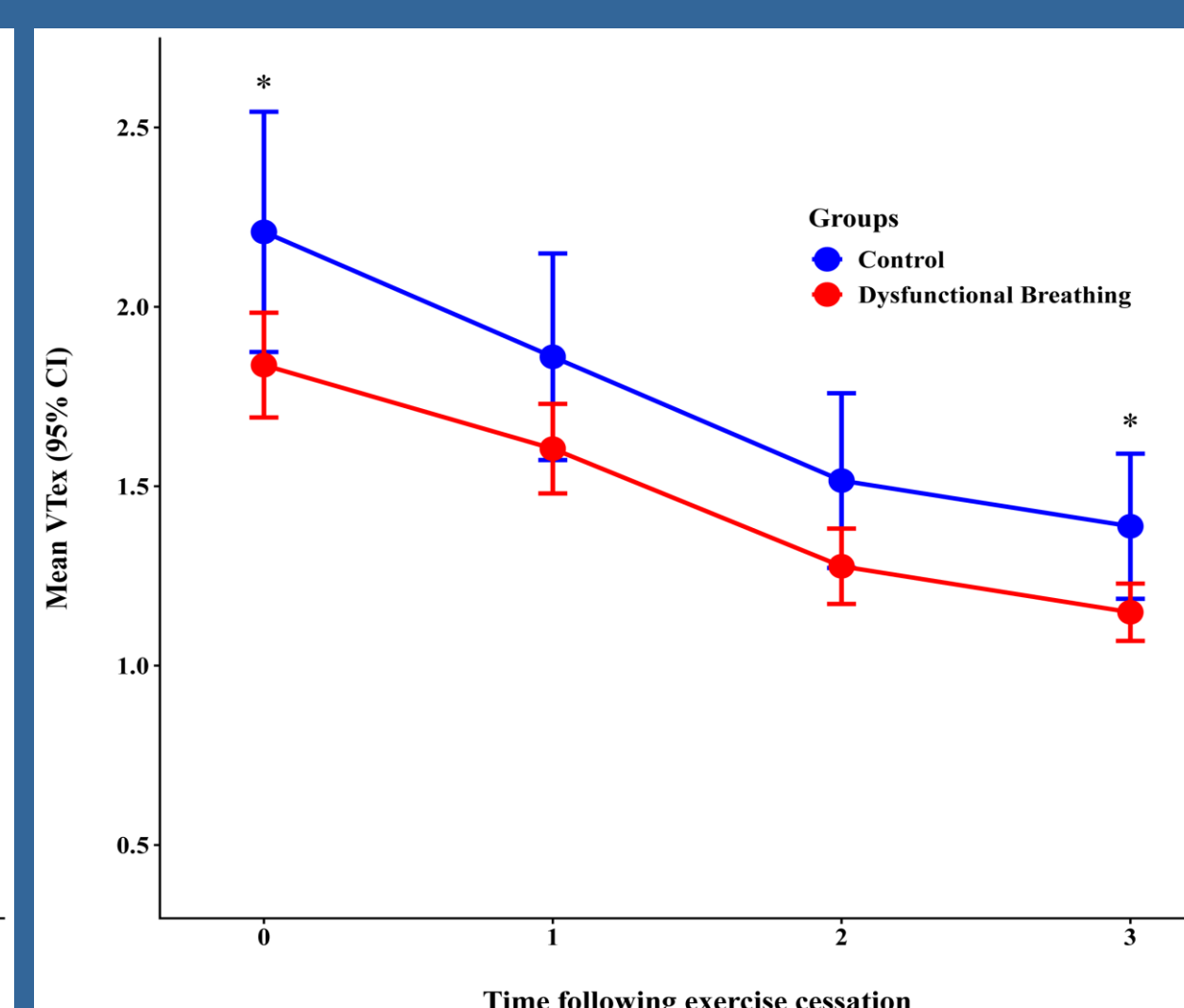


Figure 4. Mean tidal volume following exercise cessation over four time points (min). The asterisk (*) indicates a statistically significant difference between groups (p<0,05)

	Patients with DB (n=65)	LC without DB (Control Group, n=18)
Age	48.65±12,2	47.17±13,44
Sex	71% ♀	50%♀
BMI	27.22±6.66	25.29±3.34
VO ₂ peak%pred	89.48±17.62	92.5±15.95
WRpeak%pred	98.66±25.49	106.28±23.01
Data are presented as mean±SD		

Table. Baseline Characteristics and Cardiorespiratory Fitness Parameters of Study Groups. BMI= Body Mass Index; VO₂peak%pred= Peak oxygen uptake percentage of predicted value; WRpeak%pred= Peak work rate percentage of predicted value.

DISCUSSION

We demonstrate, to our knowledge for the first time, that Long COVID (LC) patients with Dysfunctional Breathing (DB) fail to normalize respiratory mechanics promptly post-exertion. Specifically, this cohort exhibited a sustained 'rapid-shallow' breathing strategy, characterized by an elevated breathing frequency of approximately 30 breaths per minute and constrained tidal volume for up to three minutes of recovery. Crucially, because baseline spirometry and aerobic capacity were preserved, this sustained tachypnoea cannot be attributed to structural lung disease or deconditioning. Instead, our data suggests an underlying alteration in central respiratory drive or autonomic regulation that persists after the cessation of metabolic load.

CONCLUSIONS

The findings suggest that LC/DB patients employ a rapid, shallow breathing strategy that successfully maintains adequate elimination of CO₂ and minute ventilation. However, this inefficient ventilatory pattern—characterized by low Vt and, particularly, the sustained post-exertional tachypnoea—indicates a severe disruption in autonomous respiratory control (Erratic Breathing) and a potential contributor to their persistent exertional dyspnea and fatigue.

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