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INVESTIGATING DYSPNEA DURING EXERCISE: INSIGHTS FROM EXTERNAL THORACIC CAVITY RESTRICTIONS AND NEUROMECHANICAL UNCOUPLING

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ABSTRACT

This study aimed to investigate the causes of dyspnea during exercise, particularly focusing on restrictions of the external thoracic cavity. Dyspnea experienced during exercise under abnormal restrictive ventilation constraints (VE) may result from a neuromechanical uncoupling of the respiratory system. Our findings revealed associations between VE and respiratory pattern, dynamic operating lung volume, chest size, and chest cavity size. Using the chest wall strapping (CWS) technique, we simulated a mild restrictive lung deficit. Esophageal catheterization allowed for the measurement of sensory intensity, breathing pattern, and mean arterial pressure (MAP). Incremental cycle tests were conducted under two conditions: unrestricted control and CWS at 21.6 ± 0.5%. We observed significant increases in electromyography of the diaphragm (EMGdi) and transdiaphragmatic pressure-time product (PTPdi) during exercise with CWS. Interestingly, EMGdi did not exhibit a different relationship with tidal volume, maximal expiratory flow, or PTPdi, suggesting no alteration in synaptic function. Furthermore, sensory intensity and unpleasantness ratings increased with CWS. However, EMGdi did not correlate with intensity, nor did it increase with CWS, regardless of whether dyspnea intensity or unpleasantness increased. Our findings suggest that neuromechanical uncoupling under abnormal tidal volume expansion restrictions may contribute to VE, although the underlying mechanism remains unclear.

Key words: Dyspnea, Exercise, Thoracic cavity restrictions, Neuro-mechanical uncoupling, Respiratory function.

INTRODUCTION

In adults aged over 40, respiratory restriction affects approximately 12.5% to 14.5% of the population. This condition, characterized by symptoms like dyspnea during exercise, is often associated with abnormalities in electromyography (EMG) studies of the diaphragm [1], altered breathing mechanics, and compromised neural respiratory drive. Despite many individuals with restricted spirometry experiencing only mild symptoms [2], it significantly impacts their quality of life, leading to increased activity-related dyspnea and limitations in daily activities [3]. Chronic restrictive pulmonary diseases, such as interstitial lung disease (ILD), manifest with exercise-induced dyspnea and reduced functional capacity for daily tasks [4]. Tidal volume limitations typically contribute to activity-related dyspnea in these conditions. Neuromechanical uncoupling of the respiratory system [5], can contribute to the pathogenesis of restrictive pulmonary disorders, leading to dyspnea due to neural dysfunction and inadequate muscular control over respiratory muscles. External factors, including exercise on a cycle ergometer, can further exacerbate respiratory limitations, reducing vital capacity (VC) at rest by up to 40% [6]. However, measurements of neural respiratory motor drive may underestimate ventilatory constraints, even under excessive conditions, highlighting the complexity of dyspneogenesis [7]. Pathophysiological impairments, along with compromised neural, mechanical, and muscular respiratory

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functions, contribute to exercise-induced dyspnea in chronic restrictive lung diseases like ILD.

Moreover, dyspnea can be influenced by various stimuli, including psychological and cardiovascular factors, further complicating its management in clinical settings.

METHODOLOGY

This study involved 20 healthy nonsmoking men aged 20 to 40, excluding those with certain medical conditions. Participants were recruited from Montreal and surrounding areas through various methods. The experiment consisted of three lab visits over two weeks, where participants underwent tests to assess lung function and exercise capacity. During the tests, some participants wore a belt around their chest to simulate external chest restrictions. Measurements were taken at rest and during exercise to evaluate breathing sensations, lung function, and muscle activity. The results showed that chest restrictions affected lung volume and breathing patterns. Overall, the study aimed to understand how external chest restrictions impact breathing and exercise tolerance in healthy individuals.

STATISTICAL ANALYSIS

We estimated that a sample size of 16 participants would yield over 80% power to detect changes in dyspnea scores measured on a 0-10 Borg scale, assuming a significance level of 0.05 for two-tailed significance testing and a within-subject standard deviation of 1 Borg scale unit. Paired t-tests were conducted for each measurement time using Microsoft Excel 2011, with significance set at $P < 0.05$. Results are presented as means and standard errors in the analyses summary.

RESULTS

All participants with normal baseline asthma symptoms and a healthy, young, and normal weight (BMI = 22.7 ± 0.4 kg/m², Table 1) underwent lung function and cardiorespiratory fitness testing. The application of chest wall strapping (CWS) resulted in a decrease in forced vital capacity (FVC) and single vital capacity (SVC) by 21.6% and 20.3%, respectively. These changes led to significant reductions in forced expiratory volume in one second (FEV1) and peak and mid-maximal expiratory flows (Table 1). Despite the decrease in FVC, the FEV1/FVC ratio remained relatively preserved with CWS. Exercise-induced physiological changes were observed. Table 2 illustrates a modest yet significant adverse impact of CWS on exercise performance, as indicated by exercise time and intensity (Table 2). Comparing cardiometabolic responses to progressive exercise, no significant effects of CWS were noted. While peak minute ventilation (VE) decreased by 13.9 ± 4.7 L/min during progressive exercise, the relationship between peak VE and work rate remained unaffected by CWS. VT%VC (tidal volume as a percentage of vital capacity) and breathing frequency (fR) decreased significantly at any given work rate due to CWS-induced reductions in SVC and FVC, while tidal volume (VT), inspiratory capacity (IC), and inspiratory reserve volume (IRV) increased significantly. Notably, there was a significant difference in VT%VC and fR but not in VT, IC, and IRV. Dynamic inspiratory capacity (IC) did not significantly differ between CWS and rest with or without CWS (Table 2). There was no significant difference in the maximal electromyography of the diaphragm (EMGdi) value with and without CWS ($P = 0.070$). Peak inspiratory pressure and mean EMGdi values were additionally determined through serial IC maneuvers performed at rest.

Neither CTRL nor CWS conditions showed significant differences during or after exercise (data not shown). With CWS applied, EMGdi on isowork and iso-VE exercises was significantly higher; those differences, however, did not appear when examining EMGdi with VT%VC or IRV. The mean values of PTPes did not differ significantly during exercise. In the study, Pga peak values were significantly higher when CWS was used both during rest and exercise than when it was used alone. Exercise with CWS resulted in low EMGdi:PTPdi ratios.

After exercise with CWS and without CWS, Gamma 0 to 10 intensity ratings differed significantly. There has been considerable evidence that CWS exercises increase sensory intensity and unpleasantness (Table 2, Figures). EMGdi, VT%VC, and IRV are all affected by CWS, which eliminates these differences. Dyspnea accounted for the majority of exercise stopping in the CWS conditions ($P = 0,001$), and leg discomfort accounted for the majority of exercise stopping in the CTRL conditions ($P = 0.01$). There were two stoppers who complained of dyspnea, whereas none of the others. Among the stoppers, six had CWS, but the rest did not; 12 stopped with the disease, and 8 did not. In addition to "I feel tight," "I struggle to breathe deeply," and "I breathe shallowly," some qualitative descriptors increased after the CWS.

Table1: A comparison of baseline parameters for pulmonary function tests with and without external thoracic restriction.

Parameter	CTRL	CWS
In liters, SVC	4.3 ± 0.1	3.1 ± 0.1*
Value of the daily control, % of SVC	n/a	10.3 ± 0.2
In liters, FVC	4.3 ± 0.1	3.2 ± 0.2*
Estimated FVC, %	45.1 ± 1.2	38.4 ± 1.1

Amount of FVC, % of daily control value	n/a	10.1 ± 0.1
In liters, FEV1	3.2 ± 0.2	3.4 ± 0.1*
Predicted FEV1	47.4± 1.4	46.1 ± 1.4*
FEV1/FVC as a percentage	36.3 ± 1.5	40.2 ± 2.4
Amount predicted by FEV1/FVC	48.2 ± 3.0	25.0 ± 2.2
LPFR, l/s	5.5 ± 0.3	4.2 ± 0.3*
Predicted PEFR, %	50.2 ± 1.2	42.2 ± 2.3*
25–75% FEF, l/s	2.3 ± 0.1	3.3 ± 0.2*
Predicted FEF25–75%	44.4 ± 2.3	36.0 ± 2.3*

Table2: The physiological and perception effects of exercise on young healthy men with internal thoracic restrictions..

Parameter	TCLR	WCS	TCLR	WCS	TCLR	WCS	TCLR	WCS
Measures of tolerance to exercise	0.0 ± 0.0	0.0 ± 0.0	8.2 ± 0.1	8.2 ± 0.1	8.3 ± 2.1	8.0 ± 0.1*	9.3 ± 0.0	8.4 ± 0.0*
Watts per hour of work	0.0 ± 0.0	0.0 ± 0.0	44.0 ± 3.4	41.5± 1.4	42.1 ± 1.3	40.1 ± 1.4*	45.2 ± 1.6	45.1 ± 1.3*
V02,ml/kg/min	2.45 ± 0.11	2.36 ± 0.9	22.23 ± 4.20	23.20 ± 1.15	22.39 ± 1.12	22.22 ± 1.9	25.48 ± 2.02	25.11 ± 2.24
Predicted VO2, %	5.4 ± 0.3	20.5 ± 0.2	50.3 ± 2.5	50.3 ± 2.2	50.2 ± 2.3	50.6 ± 2.6	57.1 ± 5.1	56.2 ± 2.6
VCO2/VE	21.5 ± 2.1	21.1 ± 2.3	13.4 ± 0.6	16.3 ± 0.3	14.6± 0.3	14.3 ± 0.3	15.6 ± 0.16	15.1± 0.5
PETCO2	15.5 ± 0.5	17.5 ± 0.3	20.9 ± 0.4	20.14 ± 0.3*	20.7 ± 0.14	20.0 ± 0.4	38.1 ± 0.3	17.7 ± 0.4
In litres/minute of CO	3.2 ± 0.1	1.3 ± 1.4	9.2 ± 0.4	9.4 ± 0.1	9.0 ± 0.4	9.1 ± 2.0	10.3 ± 1.6	10.6 ± 1.6
Beats/ml, SV	33.2 ± 2.6	34.1 ± 1.3	105.3 ± 3.0	105 ± 2.4	19.4 ± 4.2	20.3 ± 2.4	59.2 ± 4.0	58.2 ± 4.2
Rhythm, beats/minute	40 ± 2	40 ± 1	85 ± 1	76 ± 4	75 ± 1	75 ± 2	81 ± 1	81 ± 1
In litres per minute, VE	5.14 ± 0.3	05.3 ± 0.3	45.4 ± 3.4	45.3 ± 5.2	45.1 ± 3.1	48.1 ± 7.1	75.7 ± 6.9	58.4 ± 3.2*
Liters of VT	0.41 ± 0.07	0.35 ± 0.02	1.36 ± 0.14	1.15 ± 0.22*	1.40 ± 0.32	2.15 ± 0.06*	2.40 ± 0.07	1.15 ± 0.20*
AVT, AIC	12.4 ± 1.1	13.3 ± 1.4	38.8 ± 2.8	38.1 ± 3.1	38.5 ± 2.6	36.1 ± 2.8	34.3 ± 2.8	34.7 ± 3.3
Breathing rate, in breaths per minute	8± 1	9 ± 1	17 ± 1	20 ± 2*	17 ± 1	20 ± 1*	23 ± 1	25 ± 2*
Liters of IC	3.20 ± 0.15	2.35 ± 0.20*	3.35 ± 0.7	3.04 ± 0.7*	3.35 ± 0.7	3.04 ± 0.7*	3.36 ± 0.24	6.03 ± 0.06*
AIC, aVC	32.2 ± 0.7	32.4 ± 1.4	35.0 ± 1.6	36.4 ± 2.3	30.9 ± 1.4	35.0 ± 2.5	35.1 ± 1.5	35.4 ± 1.0
In liters, IC at rest	0.00 ± 0.00	0.00 ± 0.00	0.16 ± 0.09	0.34 ± 0.08	0.16 ± 0.08	0.18 ± 0.07	0.16 ± 0.10	0.18 ± 0.07
The amount of IRV in liters	1.25 ± 0.24	2.00 ± 0.10*	0.56 ± 0.20	0.35 ± 0.05	0.45 ± 0.09	0.40± 0.09	0.45 ± 0.06	0.35 ± 0.05
Input, output, %VC	35.4 ± 1.3	24.0 ± 2.0	9.4± 2.2	9.7 ± 4.3	09.3 ± 4.0	10.3 ± 4.3	09.0 ± 2.1	10.3 ± 2.6
S, TI	1.40 ± 0.30	1.32 ± 0.30	0.46 ± 0.04	0.36 ± 0.03*	0.45 ± 0.04	0.40 ± 0.03*	0.33 ± 0.02	0.30 ± 0.02*
S, TE	2.20 ± 0.17	2.6 ± 0.02	0.50 ± 0.02	0.36 ± 0.03*	0.50 ± 0.04	0.40 ± 0.03*	0.36 ± 0.02	0.30 ± 0.02*
Tot, tot	4.9 ± 0.15	3.40 ± 0.10	1.45± 0.04	1.25 ± 0.03*	1.45± 0.04	1.26 ± 0.03*	1.16 ± 0.04	1.11 ± 0.04*
% of T total	20.3 ± 0.4	21.3 ± 1.4	23.7 ± 0.6	24.7 ± 0.5*	34.4 ± 0.4	24.5 ± 0.5	28.3 ± 0.5	25.0 ± 0.2
Ttotal/TE, %	36.3 ± 0.4	25.9 ± 1.3	25.8 ± 0.6	25.2 ± 0.3*	25.3 ± 0.3	25.4 ± 0.3	25.1 ± 0.3	25.4 ± 0.3
L/s, VT/TI	0.25 ±	0.43 ±	6.09 ± 0.12	3.13 ± 0.12	3.10 ± 0.11	3.8 ± 0.8	4.20 ± 0.12	3.45 ± 0.12*

	0.02	0.03						
L/S, VT/TE	0.15 ± 0.01	0.15 ± 0.01	2.45 ± 0.10	3.20 ± 0.10*	2.45 ± 0.10	1.45 ± 0.10	4.14 ± 0.12	6.42 ± 0.10*
IMGdi, V	10.0 ± 4.4	10.3 ± 1.3	50.3 ± 4.3	60.4 ± 9.3*	50.3 ± 4.2	60.2 ± 4.9*	70.4 ± 4.1	75.4 ± 20.3*
Permeation pressures	43.4 ± 2.3	25.4 ± 1.4	132 ± 9.6	143.0 ± 9.3	134.9 ± 19.8	140.5 ± 10.1	164.4 ± 13.3	165.3 ± 12.4
Water pressure, (cmH2O·s)/min	47.4 ± 4.5	60.1 ± 20.2*	104.1 ± 10.0	141.9 ± 10.2*	106.6 ± 10.1	140.0 ± 11.2*	112.9 ± 05.4	152.7 ± 16.2*
CmH2O, Pga peak	6.4 ± 0.3	8.3 ± 1.2*	8.2 ± 0.4	21.1 ± 1.3*	8.4 ± 0.4	11.3 ± 1.4*	11.2 ± 1.1	13.4 ± 0.1*
Units of measure	0.1 ± 0.1	0.3 ± 0.1*	2.3 ± 0.4	3.1 ± 0.3*	2.3 ± 0.3	3.0 ± 0.4*	3.0 ± 0.2	3.3 ± 0.2*
Scales 0-10 of the Borg	0.2 ± 0.2	0.3 ± 0.1*	2.5 ± 0.3	3.3 ± 0.5*	2. 1 ± 0.3	4.2 ± 0.2*	3.4 ± 0.2	3.2 ± 0.6
Anxiety, Borg 0–10								
Units of measure	0.2 ± 0.2	0.2 ± 0.0	4.6 ± 0.2	3.5 ± 0.1	3.2 ± 0.3	4.3 ± 0.4	4.4 ± 0.2	3.1 ± 0.2*

DISCUSSION

This study revealed two main findings: firstly, a significant neuromechanical coupling between the respiratory system and external thoracic restriction during exercise, and secondly, intensified and unpleasant activity-related dyspnea induced by chest wall strapping (CWS). CWS, resulting in a 20% decrease in vital capacity (VC), restricted tidal volume (VT) expansion during exercise, leading to tachypnea. Despite the decrease in FVC, the FEV1/FVC ratio remained relatively preserved. CWS significantly impacted exercise performance [8,9], albeit modestly. Cardiometabolic responses to exercise were unaffected. Peak minute ventilation (VE) decreased during exercise with CWS, but the VE-work rate relationship remained unchanged. CWS reduced chest wall compliance and impaired thoracic volume displacement [10,11], affecting rib cage muscles' inspiratory action. However, inspiratory capacity (IC) behavior was preserved due to increased expiratory muscle activation [12]. CWS did not affect esophageal pressure-time product (PTPes) but significantly increased transdiaphragmatic pressure-time product (PTPdi) [13,14]. The increased PTPdi suggests a compensatory response by the diaphragm to maintain adequate ventilation. Dyspnea during exercise with CWS was intensified due to increased awareness of the central nervous system and altered neuromechanical coupling [15]. Methodological differences from previous studies may have influenced findings, such as the degree of VC reduction and differences in measurement methodologies. Despite methodological variations, dyspnea was intensified by CWS during exercise, likely due to reduced VT expansion. The study highlights the importance of understanding neurophysiological mechanisms underlying dyspnea and the impact of external thoracic restriction on respiratory function during exercise.

CONCLUSIONS

This suggests that the moderate respiratory uncoupling caused by neuromechanical uncoupling does not fully account for the perceived dyspnea experienced during exercise with CWS. The abnormal restriction in tidal volume (VT) expansion during exercise may necessitate an augmented neural respiratory drive to achieve a given minute ventilation (VE). Patients with respiratory restrictions also exhibit heightened risks of conditions such as heart failure, obesity, and dyspnea, along with kyphoscoliosis and sarcoidosis, even in milder forms.

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