



MANUAL THERAPY AND COPD: FROM THE MUSCULOSKELETAL SYSTEM TO THE LUNG

¹Diego L. Godoy, ¹Paulina A. Gutiérrez, ²Carlos V. Cruz, ³Leandro A. Miret, ⁴Felipe A. Contreras, y ³Jorge J. Mauro.

¹School of Kinesiology, Universidad Metropolitana de Ciencias de la Educación, Santiago, Chile

²School of Kinesiology, Universidad de Chile, Santiago, Chile

³Unit of Kinesiology, Complejo Hospitalario San José, Santiago, Chile.

⁴Career in Kinesiology, Pontificia Universidad Católica de Chile, Santiago, Chile.

Email: diegogodoy.kine@gmail.com

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is defined as an obstructive type respiratory disease, chronic airflow limitation which is progressive, not fully reversible and preventable. This is generated as a result of the hyperreactivity of the pulmonary parenchyma against harmful and irritative agents triggering thereof chronic inflammation [1]. Its multiple physiopathological implications make this a multisystemic disease, ranging from respiratory failure as the first focus, eventually impacting the structure and function of both organs and the musculoskeletal system [2-4].

The changes that occur in the anatomy of the airway and lung parenchyma as bronchial hypersecretion and bronchoalveolar instability by loss of radial traction mainly causes the expiratory flow limitation (EFL), generating air trapping, clinically known as dynamic hyperinflation (DHI). This phenomenon leads to an increase in expiratory reserve volume (ERV) and residual volume (RV), therefore an increase in functional residual capacity (FRC), limiting the tidal volume (VT) and the inspiratory reserve volume (IRV), consequently, affecting the inspiratory capacity (IC) [5]. These changes alter the geometry of the thorax affecting the position of the ribs causing a similar state to a sustained inspiration over time ("inspiratory block"), creating a *barrel chest*, altering the position of the diaphragm, leaving flattened and shortened, losing the ability to generate force and finally working at a mechanical disadvantage [6,7]. As compensatory adaptation is necessary the recruitment of accessory respiratory muscles [6,7], which sustained over time determines a overadaptation, which leads to a shortening and overactivation, as well as the surrounding cervicothoracic fascia, adopting postures as anterior projection head with neck hyperextension, increased thoracic kyphosis, anterior projection and internal rotation of

shoulders, etc. all this with the aim to meet the demand of the respiratory system. Finally this results in chest tightness, with increased of breathing work which decreases the ability to generate inspiratory pressures and thus mobilize inspiratory volumes [7-10]

METHODS

The sample for this study consisted of 12 patients, 9 men and 3 women, aged 46 and 77 years, diagnosed with COPD III and IV through spirometry. All were subjected to one intervention of five manual therapy techniques: Suboccipital Release /Decompression, Anterior Thoracic Myofascial Release and Sternum Liberation, Anterior Cervical Myofascial Release, Muscle Energy Techniques for pectoralis minor, scalene, latissimus dorsi and serratus anterior, and finally Costal Ligament Balance, with a duration of 30 minutes. Were evaluated lung volumes and capacities through plethysmography and hemodynamic parameters of heart rate (HR), respiratory rate (RR) and oxygen saturation pulse, pre and post intervention. Changes were considered significant at $P < .05$.

RESULTS AND DISCUSSION

In this study all patients showed a significant decrease of the CPT ($p = 0.031$), VRE ($p = 0.005$), VR ($p = 0.002$). According to the theory [11-14], these changes are probably due to the chosen techniques that aim to correct pathomechanics patterns acquired in the course of the disease through the intervention of the fascial, muscular and ligamentous tissues, both the rib cage and surrounding regions, to reduce the state of tension in them, which would be transmitted from the outside to the inside of the chest, through structures such as Sibson's fascia and the suspensory ligaments of the pleural dome, which would facilitate the process of passive air output [12-16]. The decrease in VRE and VR directly affect the significant increase in CI ($p =$

0.039) [5], which is in addition to changes in inspiratory muscle condition, that they would return to a length that is close to optimal, increasing mechanical efficiency [6,7]. The most capacity of volumes exchange would renew the gas content of the lungs, eliminating rich air volumes of CO₂ and entering more air and thus increase the concentration of O₂ available to improve alveolar ventilation expressed in highly significant increase of SpO₂ (p = 0.000). The vasodilatory effect of O₂ allows increased blood flow at the capillary level so also there would be more hemoglobin available to capture more O₂ [17]. Moreover breathing rate also showed a highly significant decrease (p = 0.000). One can infer that this change is a result of the increase in inspiratory capacity and efficiency of the respiratory muscles to mobilize larger inspiratory volumes, and at the same time because central and peripheral chemoreceptors sense these pressure variations in both CO₂ and the O₂, sending this signal to the respiratory centers [17,18]. Simultaneously, all patients showed a highly significant decrease in heart rate (p = 0.000), this perhaps because by decreasing CRF also decreases total internal pressure of the lung, which would generate less pressure on the pulmonary artery, which coupled to vasodilation in the pulmonary capillaries, would reduce the resistance to blood flow and thus less contractile work of the heart to generate ejection volumes [19-21].

However, both the CV (p = 0.799) as the airway resistance (Raw) (p = 0.069) showed no significant change, this probably because the decrease of the CPT and the CRF, and the increase of the IC, remains proportionally constant the CV. However, the RAW doesn't change possibly because the intervention of the soft tissues of the respiratory pump doesn't generate changes at structural level both in the airways and lung parenchyma hence its tendency to collapse and lability doesn't change.

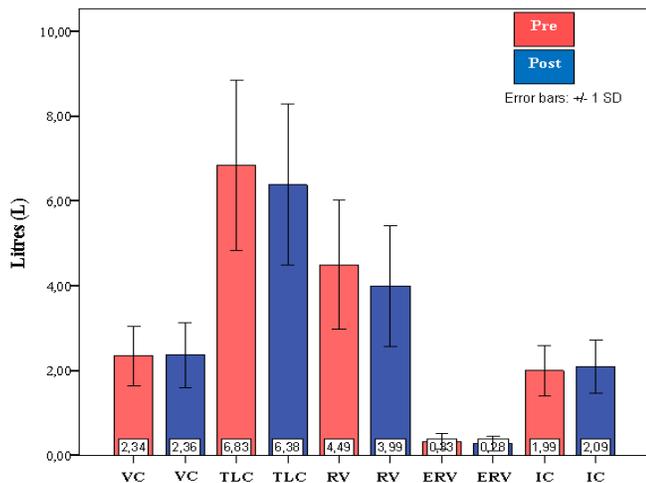


Figure 1: Changes in lung volumes and capacities pre and post intervention.

CONCLUSIONS

Manual therapy techniques showed beneficial changes in lung function, reflected in a significant diminution of VRE and VR with highly significant decrease in HR and RR, and significant increase in CI and highly significant increase in SpO₂. However is important to project this study over time and develop others in the future to help elucidate concretely the real long-term effects and impact on quality of life of patients such as in exercise tolerance or in carrying out their activities in daily life.

REFERENCES

1. *Global Strategy for the Diagnosis, Management and Prevention of COPD, Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2011*. Available from: <http://www.goldcopd.org>
2. Rennard S, Inflammation in COPD: a link to systemic comorbidities, *Eur Respir Rev*; **16**: 105, 91–97, 2007
3. Yawn B, Kaplan A, Co-morbidities in people with COPD: a result of multiple diseases, or multiple manifestations of smoking and reactive inflammation?, *Primary Care Respiratory Journal*; **17(4)**: 199-205, 2008
4. Skeletal Muscle Dysfunction in Chronic Obstructive Pulmonary Disease A Statement of the American Thoracic Society and European Respiratory Society, *Am J Respir Crit Care Med*, **159**. pp S1–S40, 1999
5. Lisboa C, Borzone G and Díaz O, Dynamic hyperinflation in chronic obstructive pulmonary diseases: functional and clinical implications, *Rev. Chilena Enfermedades Respiratorias*; **20**: 9-20, 2004
6. Ottenheim C, Heunks L and Dekhuijzen R, Diaphragm adaptations in patients with COPD, *Respiratory Research*, **9**:12, 2008
7. Orozco-Levi M, Structure and function of the respiratory muscles in patients with COPD: impairment or adaptation?, *Eur Respir J*; **22**: Suppl. 46, 41s–51s, 2003
8. Chaitow L, *Terapia Manual Valoración y Diagnóstico*, Editorial McGraw-Hill Interamericana, 1997
9. Chaitow L, Walker DeLany J, *Aplicación clínica de las técnicas neuromusculares I Parte superior del cuerpo*, Editorial Paidotribo, 2006
10. Kirkwood R et al., Orientation and position of the scapula, head and kyphosis thoracic in male patients with COPD, *Canadian Journal of Respiratory Therapy*, **45**: 30-34, 2009
11. Ercole B, Antonio S, Ann D, Stecco C, How much time is required to modify a fascial fibrosis?, *Journal of Bodywork & Movement Therapies*; **14**, 318e325, 2010
12. Lelean P, The migratory fascia hypothesis, *Journal of Bodywork & Movement Therapies*; **13**, 304e310, 2009
13. Schleip R, Fascial plasticity – a new neurobiological explanation: Part 1 and 2, *Journal of Bodywork and Movement Therapies*, **7(1)**, 11-19, 2003

14. Schleip R, Gitta Muller D, Training principles for fascial connective tissues: Scientific foundation and suggested practical applications, *Journal of Bodywork & Movement Therapies*; **17**, 103e115, 2013
15. Swanson R, Biotensegrity: A Unifying Theory of Biological Architecture With Applications to Osteopathic Practice, Education, and Research—A Review and Analysis, *J Am Osteopath Assoc*; **113(1)**:34-52, 2013
16. Stanbrough M, *Direct Release Myofascial Technique*, Churchill Livingstone, First Edition, 2004
17. West J, *Fisiología Respiratoria*, Séptima Edición, Editorial Medica Panamericana, 2005
18. West J, *Fisiopatología Pulmonar*, Quinta Edición, Editorial Medica Panamericana, 2000
19. Chaouat A, Naeije R and Weitzenblum E, Pulmonary hypertension in COPD, *Eur Respir J*; **32**: 1371–1385, 2008
20. Naeije R, Pulmonary Hypertension and Right Heart Failure in Chronic Obstructive Pulmonary Disease, *Proc Am Thorac Soc*; **2**:20–22, 2005
21. Nieto M, EPOC e hipertensión pulmonar, *Arch Bronconeumol*; **45(4)**:24-30, 2009