

The Importance of the Diaphragm in Neuromotor Function in the Patient with Chronic Obstructive Pulmonary Disease

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Abstract: Chronic obstructive pulmonary disease (COPD) is a constant and chronic narrowing of the respiratory airways, with numerous associated symptoms, not always related to the pathological adaptation of the lungs. Statistical projections show that COPD could become the third leading cause of death globally by 2030, with a significant increase in deaths by 2060. Skeletal muscle dysfunction, including the diaphragm, is one of the causes linked to the increase in mortality and hospitalization. Little emphasis is given by the scientific literature to the importance of the diaphragm towards functional neuromotor pathological expressions. The article reviews the adaptation of the skeletal muscles, with greater attention to the adaptations of the diaphragm, thereby highlighting the non-physiological variations that the main respiratory muscle undergoes and the neuromotor impairment found in COPD. The text could be an important reflection from a clinical and rehabilitation point of view, to direct greater attention to the function and adaptation of the diaphragm muscle.

Keywords: diaphragm, COPD, physiotherapy, rehabilitation, pain, fascia, ageing, osteopathic

Introduction

Chronic obstructive pulmonary disease (COPD) is a complex and constantly evolving pathology, which is characterized by a progressive and constant limitation of the available air volume (airflow obstruction).¹ COPD could become the third leading cause of death for the population by 2030.¹ The Global Initiative for Chronic Obstructive Lung Disease (GOLD) identifies COPD as:

a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.²

GOLD estimates that the number of patients who will die of COPD in 2060 will be approximately 5.4 million deaths annually, compared to 3.2 million deaths in 2015 annually.³ In the USA, the incidence of the disease involves 10.2–20.9% of the population, in Europe, despite a trend of reduction in mortality and a decreased difference between men and women, the percentage of findings remains high (3–26.1%).³ Some data from Africa, few and not covering all geographical areas, show COPD detection rates of 1.6–23.8%; in Latin America, the percentage is between 30% and 31.1%.³ In China, the percentage is about 8.6% (data from 2018), and highly variable and incomplete values for other Asian regions, with an average of 3.5–6.7%.³

COPD is considered as the fifth cause of burden on the economy of the various states due to recurring hospitalizations and pharmacological treatments.⁴ Probably, this global economic aggravation derives from the fact that this pathology does not involve only the pulmonary area but develops many dysfunctions and physiological alterations involving the whole-body system.⁴ Although emphysema and chronic bronchitis are responsible for triggering the chronicity of the disease, the patient does not always go to hospital for respiratory reasons, but for important cardiac problems, fractures,

the onset of lung tumours, mood disorders, muscle complex that prevent independence and severe metabolic alterations.^{1,5} Furthermore, it is not always possible to correctly assess the severity of COPD, as there is only a moderate relationship between pulmonary adaptation and the reduction in the patient's quality of life.⁶ COPD is a systemic disease, and as such it is expressed with multiple symptoms, such as insomnia and drowsiness, dry mouth, anorexia, pain (local or from multiple involved areas), nausea, constipation, gastric reflux, dysphagia, sleep apnea, cough, fatigue and shortness of breath, wheezing and dyspnea, temporomandibular disorders and urinary incontinence.^{2,5,7–10} Some symptoms related to the presence of COPD and which have a negative impact on the mortality and morbidity of patients, are the finding of anxiety and depression, with values ranging from 8% up to a maximum of 80% of patients.^{1,5,11} Functional and structural alterations of the skeletal muscles (almost all patients), lack of coordination and an increase in accidental falls cause a decrease in active movement, leading to a decline in quality of life, an increase in mortality and in the number of hospitalizations.^{12–14}

The article reviews the adaptation of the skeletal muscles, with greater attention to the adaptations of the diaphragm, trying to highlight the relationship between the non-physiological variations that the main respiratory muscle undergoes and the neuromotor impairment found in COPD. The text could be an important reflection from a clinical and rehabilitation point of view to direct greater care to the function and adaptation of the diaphragm muscle.

Peripheral Skeletal Muscle Adaptation in the Presence of COPD

Patients with COPD experience skeletal muscle alterations, causing an increase in the rate of morbidity and mortality, as well as in disease exacerbation events and in the number of hospitalizations.¹⁵ Respiratory disease does not always perfectly reflect peripheral muscle adaptation.^{13,14} The following figures highlight, for the same patient, a slower pathological adaptation of the lungs of an average degree, compared to an accentuated sarcopenic adaptation (Figures 1 and 2).

Muscle functional impairment is found more with emphysematous patients than with chronic bronchitis, and for about 55% of patients with stable COPD; this process probably occurs faster than the pathological adaptation of the lungs.^{13,14} There are different causes, single or superimposable, which can induce skeletal muscle dysfunction. Cigarette smoke, the presence of diabetes, malnutrition, advanced age, polluted air and a sedentary lifestyle combine to produce non-physiological muscular behaviors.^{16,17} COPD is a further cause of muscle dysfunction. We can find systemic inflammation, oxidation, multiple drug intake, nocturnal and diurnal hypoxia, hypercapnia. The patient often presents co-

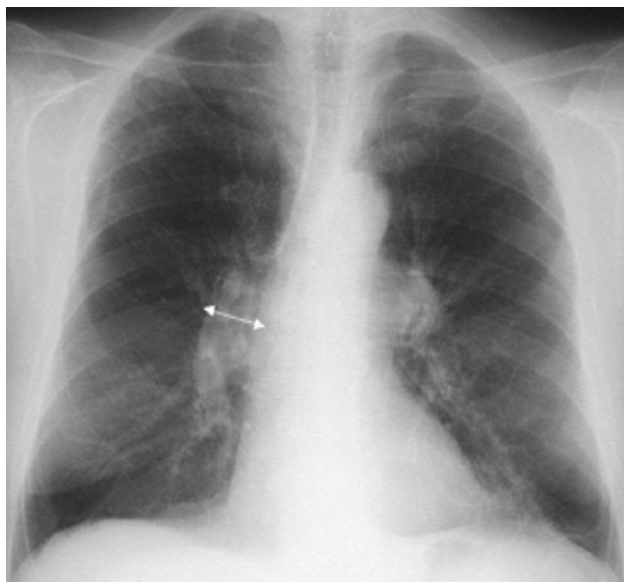


Figure 1 Moderate functional obstruction on respiratory function tests of a 70-year-old patient. X-ray picture of emphysema with pulmonary hyperinflation and thinning of the vascular pattern (arterial deficiency) with hypertension of the small circulation documented by the dilatation of the descending branch of the right pulmonary artery (arrow).



Figure 2 MRI of the same patient with moderate degree of obstruction, but evident sarcopenia. In the cross section of the subject's leg it is possible to notice a reduced muscle mass (light gray area), and a pronounced area of subcutaneous and intramuscular fat (dark gray area).

morbidities such as polyneuropathy, osteoporosis, cardiovascular pathologies, bronchiectasis, obstructive sleep apnea, chronic kidney disease, anxiety and depression, nonalcoholic fatty liver disease, nonspecific pain and gastroesophageal reflux.^{5,9,11,18–20} These co-morbidities negatively affect muscle adaptation.

Changes in Muscles in COPD: Males versus Females

Females with COPD suffer a loss of lean mass in a greater percentage than males, and with a lower value of the expressed strength.²¹ Limb musculature undergoes a phenotypic transition in patients, with a tendency for loss of number and volume of aerobic fibers or type I fibers, and an increase in anaerobic fibers or type II fibers, compared to age-matched healthy subjects.²¹ Women have slightly fewer type IIx fibers (more glycolytic muscle cells than type II fibers), and a higher number of hybrid fibers. The latter information highlights a difficulty of the musculature in women to implement a correct regeneration and with more dilated times (compared to men).²¹ Women lose more muscle mass and are weaker than men. In patients with COPD, we can find sarcopenia, decrease in mass and strength, and concomitant atrophy, weakness and disappearance of lean material.²² Skeletal muscle areas, particularly of the extremities, have reduced capillary architecture, and increased mitochondrial and ribosomal dysfunction.^{14,23}

Stable Disease vs Exacerbation

Exacerbation of respiratory symptoms causes an increase in non-physiological adaptations of skeletal muscles. The expression of muscle strength after 3 days of hospitalization, compared to stable patients, is lower; after 5 days of hospitalization, muscle strength is reduced by 5%, compared to outpatients.²⁴ Hospitalization reduces muscle strength not only in the lower limbs, but also in the upper limbs; this decrease is greater for patients subject to multiple exacerbations during the year.²⁴ The acute phase of the disease causes a reduction in the lean mass of the parasternal/intercostal muscles, which in parallel reflect the decrease in leg volumes; chest muscle weakness corresponds to spirometric severity.^{25,26} The severity of the presence of sarcopenia/atrophy in patients with COPD determines a worse prognosis,

with increased mortality.^{16,25,27} We do not know the detailed pathological reasons that cause such muscular adaptations, and further studies will be necessary to better understand the therapeutic procedure.

Balance Impairment

Non-physiological muscle adaptation in patients with COPD, as measured by surface electromyography (sEMG) and electromyographic activity (EMG) under stress, determines a decrease in neuromotor coordination and early fatigue.^{28–32} This impaired neuro-coordination of the limbs and trunk during active motor activity of patients causes accidental falls and increased fear of falling, creating a behavioral framework that further degenerates into motor dysfunction, physical deconditioning and increased mortality.³³ Patients with COPD have an approximately 51% higher rate of falling than healthy people of the same age.³⁴ Females are more prone to accidental falls than males.³⁴ During the acute phase, with hospitalization, the percentage of falls increases up to 55% of patients, and always with a higher finding value than in non-COPD and hospitalized elderly subjects (35%).³⁴ The risk of falls in COPD is not related to forced expiratory volume in the 1st second (FEV1).³⁴ Impaired neuro-coordination causes a disturbance of body balance during daily activities, a decrease in limb and trunk control during walking, regardless of the degree of severity of lung function.^{34,35}

There are several reasons given in order to understand the problem of lack of balance. The same causes that lead to muscle dysfunction, mentioned above, are pointed out as important genesis of balance deterioration.³⁵ Other causes leading to an increase in the incidence of falls could be related to the presence of depression and anxiety, the use of portable oxygen devices, dyspnoea, altered biomechanics of the chest, decreased muscle elasticity, reduced neurocognitive function, obesity.^{16,34–38} A possible decline in the components that help maintain balance, the vestibular area and the proprioceptive system, can increase the number of accidental falls.^{16,39} Visual dysfunctions in COPD patients would not result in a balance disorder.⁴⁰

Patients with COPD and neuromuscular dysfunctions show a slower recovery in restoring the center of body balance, in the presence of external stresses, and with accentuated trunk rigidity.⁴¹ There is greater body sway during limb movements, a slower pace than in people of the same age but not with COPD: the pattern of movement and gait in patients is altered.^{41,42}

Another possible cause that would determine a dysfunction of balance, placing COPD among the chronic pathologies with the highest incidence of accidental falls (behind only osteoarthritis), comes from the non-physiological adaptation of the diaphragm muscle.^{40,42,43}

Diaphragm Muscle Adaptation in the Presence of COPD

Currently, we do not have sufficient clinical attention directed to the diaphragm muscle in COPD patients; diaphragmatic dysfunction is present at every stage of the disease.³

Effect of Dynamic Hyperinflation

The maximal inspiratory pressure (P_{Imax}) is lower than in healthy subjects, as is the transdiaphragmatic pressure (P_{di}) generated by the diaphragm.^{40,44} The contractile fibers are shortened, giving an inspiratory attitude and a flatter morphology.⁴⁴ This morphological dysfunction corresponds to a lower functionality and expressed strength; the latter reflects the presence of hyperinflation and dyspnoea.⁴⁴ Figure 3 highlights a diaphragm of a COPD patient in an inspiratory attitude, with flattening of the diaphragm; in the healthy subject, the right area is about 1.9 centimeters higher.⁴⁵

In COPD patients, the elasticity of the lung parenchyma decreases exponentially with the chronicity of the disease. In obese patients, in particular (especially in the android form), lipofibroblasts can accumulate in the lung tissue, secreting pro-inflammatory substances (adipocytokines) and transform into myofibroblasts, increasing pulmonary fibrosis.⁴⁶ As the disease progresses, the chest expands to a lesser extent, the intercostal musculature will become stiffer, and the respiratory accessory musculature will be more ineffective.⁴⁷ The diaphragm will be forced to work harder to overcome the resistances of the more rigid chest, with consequent morphological change (more “flat”), structural change (shorter fibers), positional change (inspiratory attitude), phenotypic change (phenotypic shift) and functional (greater stiffness).



Figure 3 Chest x-ray of a COPD patient showing pulmonary hyperinflation characterized by parenchymal hyperdiaphania and flattening of the diaphragm.

The diaphragm undergoes a flattening with mechanical disadvantages. There is a reduction of its area of apposition, a decline in the coordination between the diaphragm itself and the rib muscles, an increase in the radius of curvature (less ability to maintain the tension produced).^{3,44,48} The phrenic nerve undergoes myelin damage, with a slowing of the conduction velocity, with higher neuropathy values for subjects with hyperinflation and, in particular, for the left phrenic area.^{48,49} Neuropathic adaptations correlate positively with FEV1.⁴⁹ Such neuropathy is a direct indicator of the risk of accidental falls.⁵⁰

Change in Contractile Function and Fiber Phenotype

The diaphragm undergoes an unphysiological phenotypic adaptation, with an increase in type I fibers and a decline in type II fibers. Such metabolic change appears to occur faster than the phenotypic change of limb musculature.⁴⁴

Contractile fibers show signs of atrophy, myolysis, sclerosis and fibrosis.^{44,47} We can find a decline in endoplasmic reticulum function, which dysfunction causes an accumulation of intracellular calcium (Ca²⁺).⁵¹ The accumulation of Ca²⁺ in the sarcoplasm is cytotoxic for the cell, stimulating biochemical reactions that can lead to apoptosis.⁵² Furthermore, Ca²⁺ can bind to inorganic phosphorus (Pi), slowing and preventing the formation of actomyosin bridges, causing a weaker diaphragmatic contraction.^{44,53} The force expressed by the diaphragm is about 35% lower in patients, in particular, in patients with severe COPD.³

The amount of myosin decreased by about 50% less than in healthy subjects, with a decreased Ca²⁺ binding sensitivity, with further decline in contractile strength.⁴⁴ The amount of the nebulin protein is reduced, making the muscle fiber more fragile to mechanical stress.^{44,54} Another sarcomeric protein, titin, does not seem to decrease in quantity, but undergoes a decline in elastic capacity, negatively altering the mechanotransductive capacity of the fiber (less regenerative capacity and greater stiffness).⁴⁴

The volume of fibers, both glycolytic and oxidative, has a reduced volume (atrophy) of about 40–60% compared to healthy subjects, with increases in the ubiquitin-proteasome pathway. There is an accumulation of sarcomere area Z proteins, a misalignment of the sarcomeres and loss of serial sarcomeres (about 10–15% of the total), typical of a myopathy.^{3,14,44,55} There are several pathways that disrupt diaphragm fiber structure and function in the presence of

oxidative stress and inflammation. Activation of the canonical nuclear factor kappa-light-chain-enhancer of activated B cells (NF- κ B) pathway, the activation of the non-canonical NF- κ B pathway, and activation of the myostatin-mothers against decapentaplegic homolog 3 (Smad3) pathway.⁵⁶

The fiber is shortened by about 28% compared to healthy subjects, with an increase in collagenous tissue.^{3,57} The regeneration capacity of the fibers is reduced (increased myostatin values), as is the ability of the satellite cells to repair the various muscle components.^{3,44} FEV1 is inversely related to diaphragm hypotrophy.⁴⁴

Changes in Blood and Lymph Supply

We do not know the adaptation of the lymphatic vessels of the diaphragm in the presence of COPD. Generally, with inflammation involving the body system, from the chest to the abdomen, the diaphragm undergoes lymphangiogenesis.⁵⁸

The blood volume and capillary scaffolding affecting the diaphragm musculature appear to be preserved and, despite the increase in the number of type I fibers, the diaphragm shows reduced endurance capacity.⁴⁴ It appears that the patient stops physical activity due to diaphragmatic weakness, despite an increase in phrenic electrical activity; dyspnea is the response to diaphragm fatigue and not necessarily to pathological pulmonary adaptation.^{3,59}

Diaphragm weakness, hypotrophy (diaphragm thickening fraction), reduced range of motion, are correlated with symptom exacerbation and re-hospitalization.⁶⁰ The diaphragm predicts the possible exacerbation of symptoms in COPD patients, and it becomes essential to evaluate the diaphragm for an in-depth clinical assessment.⁶⁰

Another step forward that the clinician should take is to consider the importance of the diaphragm as a determinant for the neuromotor expression of the patient with COPD.

Role of the Diaphragm in Neuromotor Function and Balance

The diaphragm is an important muscle not only in the respiratory field, but also in the neuromotor field.⁶¹ The areas that manage the breath and non-respiratory actions of the diaphragm, and which serve as an informational crossroads between the brain areas and the spinal cord, are located in the pons, midbrain and medulla, ie, the central pattern generator (CPG).⁶¹ Within the CPG, we find the pre-Bötzinger complex (preBötC), the caudal ventral group (VRGc), the rostral ventral group (VRGr), the parabrachial/Kölliker-Fuse complex and the nucleus of the solitary tract (NTS) of the vagus nerve (Figures 4).⁶¹

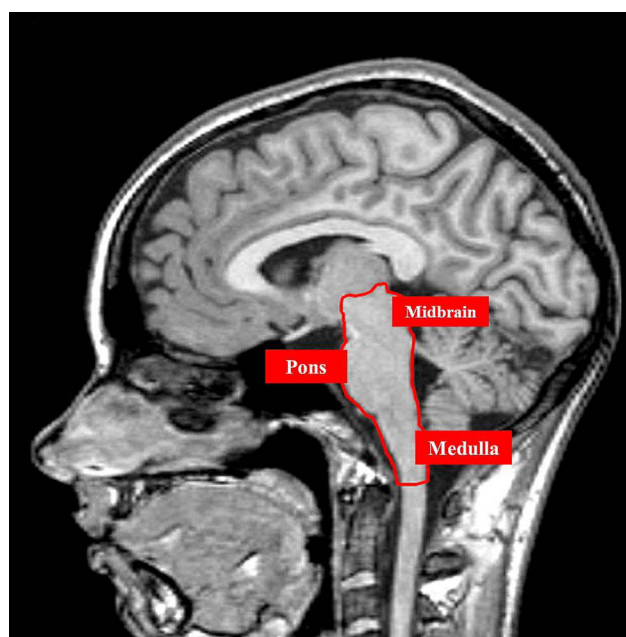


Figure 4 The figure illustrates an MRI image without contrast, highlighting, in a sagittal plane, the midbrain area, pons and medullary area. This anatomical area encloses the kernel of the respiratory network.

Proprioceptive Response from the Diaphragm

The movement of the diaphragm generates a Pdi, which is the difference between gastric and intrapleural pressure.⁶² These pressure changes and redistribution of body fluids activate multiple body receptors (mechanoreceptors, visceroreceptors, chemoreceptors, baroreceptors, etc.), which are part of the exteroceptive and interoceptive system; the latter two fall within the definition of proprioception.⁶³

The diaphragm itself carries phrenic afferents from mechanoreceptors (30–45% sensory-type fibers), which are activated by specific stimuli (animal studies). Type IA fibers are activated during the exhalation phase (lengthening of muscle fibers) and during constant contraction and in the presence of fatigue.⁶⁴ Type Ib fibers (Golgi tendon organs and simil-Pacinian corpuscles) send information during an increase in the contractile stress of the diaphragm (physical activity), during inspiration. Type III–IV fibers, myelinated and unmyelinated (type C fibers), respectively, and with a smaller diameter than the previous fibers (type IA), are activated by unphysiological breathing patterns and by the presence of metabolites related to contractile fatigue.⁶⁴

The unmyelinated fibers, the most representative of the diaphragm, can have components of a sympathetic type, and on an animal model, they send information in the presence of diaphragmatic fatigue with vasoconstrictive functions; type III fibers are not activated by diaphragmatic contractile fatigue.^{64,65} Studies on cadavers show that the right phrenic nerve has catecholaminergic axons and has a larger diameter, while the left phrenic nerve lacks them and has a smaller overall diameter; the reasons are not known.⁶⁶

There are only a few dozen spindles of the diaphragm but, despite this evident diversity compared to the skeletal muscles of the limbs and trunk, they are sufficient to exert an adequate afferential influence towards the central nervous system.⁶⁷ In a healthy subject, before the diaphragm performs a complete movement of inspiration, low-threshold cutaneous receptors (Merkel, Ruffini) are stimulated.^{68,69} These receptors send afferents to the cortex (somatosensory opercular area or primary somatosensory cortex or area S1).⁶¹ The S1 area is important to prepare the body system to adequately manage proprioceptive stimulations, activating the insula and the anterior cingulate cortex.⁷⁰ In particular, the right insula is fundamental for the conception of the self, that is, the correct elaboration of the bodily stimuli for the expression of the final movement (and the emotional aspect) with respect to our adequacy in executing this movement in the context in which we are, at a given moment.⁷¹ When the diaphragm contracts to complete the inspiration, all the stimulated body receptors and the few diaphragmatic receptors will send information to the midbrain area (about 95% of the information) and to the spinal trigeminal nucleus, via spinothalamus pathways (laminae I–X).^{61,72,73} The nucleus of the solitary tract (NTS) in the midbrain will receive the major afferential information, while only a small portion of the receptor inputs will go to the spinal trigeminal nucleus or trigeminal nucleus caudalis.^{61,74} Likewise, the afferents of the vagus nerve from the crural area of the diaphragm (and from the phrenoesophageal ligaments), stimulated by the respiratory movement, will arrive at the NTS.⁷⁵ The NTS will reciprocally exchange the information received with the cerebellum (from all cerebellar nuclei) and with the vestibular area.^{76,77} The trigeminal nucleus caudalis will exchange information with the vagal system (NTS), with the cerebellum (paramedian lobule) and with the vestibular area.^{78–81} The processing of the data obtained from this neurological network will be sent by the NTS towards the limbic area (periaqueductal gray area, amygdala, thalamus, pituitary) and towards the primary motor cortex or M1 (motor coordination) and the supplementary motor area of the cortex or SMA (movement planning and learning).^{61,73,82–84}

Descending excitatory information will be sent from the limbic area and motor cortex M1-SMA to the NTS.^{61,83,85,86} Finally, NTS sends inhibitory information to the rostral ventrolateral medullary area or premotor area of the sympathetic system.^{61,79,87} NTS will involve the CPG and phrenic neurons.⁶⁶ Inhibition of the sympathetic area will produce an increase in the activity of the parasympathetic system, affecting neuromotor expression, with increases in strength and coordination (in particular, with slow and deep breaths).^{61,86,88}

Intra-Abdominal Pressure and Posture

The diaphragm is essential not only for the force expressed, but also for maintaining body posture during daily activities, controlling the position of the lumbodorsal area allows for better control of limb movement.⁶⁶ The inspiration causes an increase in intra-abdominal pressure (IAP), thanks also to the activation of the abdominal muscles (in particular, the

transversus muscle), and the descent of the pelvic floor.^{61,89} The contraction of the diaphragm precedes the movements of the limbs, that is, for the anticipatory postural regulation.⁸⁹ This pre-contraction allows the various spinal and cortical centers to have information before deciding what movement to make. The greater the demand for muscle strength in the limbs (lifting a load or pedaling), the wider the excursion of the diaphragm will be; in this way, there will be a better stabilization of the trunk (greater IAP) and an optimization of the coordination of the limbs.^{89,90} If the IAP is insufficient, for example, due to shallower breathing, postural control problems, balance alterations and limb dysfunctions will occur.^{89,91} IAP creates a hydraulic effect for the stabilization of the lumbodorsal column with a reduction of the electrical activity of the deep back muscles; the latter event occurs because the posterior spinal musculature is not used to maintain posture.^{9,91} The movement of the diaphragm during postural tasks is not correlated with the contraction function for respiration. The diaphragm is a structure that encompasses two identities and with non-homogeneous movements.⁹¹ If the need to create balance (a demanding bodily action) increases, this situation will decrease the ability of the diaphragm to express itself as a respiratory muscle.⁹¹ Likewise, the diaphragmatic contraction that precedes voluntary (about 20 milliseconds before muscle activity of the limbs), and non-voluntary movements, is independent of respiratory function.^{92,93}

It is the contraction of the diaphragm that informs the central nervous system of body position and postural needs (by sending information from body receptors); this proprioceptive information is conveyed towards the cortex by the activity of the diaphragm.^{92,93}

The Postural Diaphragm

The parasympathetic system plays an important role in the proper functioning of the diaphragm and neuro coordination.⁶¹ COPD patients show an increase in the sympathetic system (sympathoexcitation), and this increase predicts a decrease in exercise tolerance and a poor prognosis.^{94,95} This chronic sympathoexcitation is inversely related to PImax.⁹⁵ During an exacerbation, there is a discrepancy between the activity of the sympathetic and parasympathetic systems.

Lung filling/emptying stimulates mechanoreceptors, such as rapid-adapting pulmonary stretch receptors (RARs), slow-adapting pulmonary stretch receptors (SARs), and C-type fibers. In particular, RARs send signals via the vagus nerve to the NTS to favor diaphragm activation.⁹⁶ Lung hyperinflation and emphysema stimulate more RARs and type C fibers.^{97,98} Overstimulation of these receptors could cause a release of local pro-inflammatory substances, causing bronchoconstriction and perpetuating a pathological pulmonary environment.^{95,99} During an exacerbation, the parasympathetic system increases.

Generally, vagal hyperactivation is measured indirectly by the decrease in heart rate variability (HRV).¹⁰⁰ In reality, HRV does not correctly reflect vagus nerve involvement.¹⁰¹ The parasympathetic system is more active in acute phases at the pulmonary level, but not at the systemic level. In fact, the parasympathetic system in the acute phases fails to activate the diaphragm more.⁶⁰ Data confirm that in the exacerbation phases, the COPD patient is more at risk of falling.³⁴ There is a relationship between diaphragm weakness and the risk of falls.

We know that electromyography of the spinal muscles and that of the external obliques and rectus abdominis is increased compared to healthy subjects; this should be an indirect clue to the dysfunction of the diaphragm as a postural muscle.⁴¹ We know that the ability to manage proprioceptive information in the COPD patient is impaired, and this alteration is connected to diaphragmatic dysfunction in the role of postural muscle.^{40,87} The diaphragm remains in a shortened condition, and with reduced excursion capacity in COPD patients.^{3,25} Another relationship between diaphragm weakness and fall risk.

The diaphragm is unable to adequately solicit the spino-cortical and cortico-spinal neurological pathways, as its contractile capacity is decreased; the result is a balance impairment, increased risk of accidental falls and increased mortality/morbidity. We can strongly speculate that the impaired postural balance, the neuro-motor incoordination of the trunk and limbs, could be related to the diaphragm.

We know that rehabilitation inspiratory training improves postural balance in patients.³³ The same literature is very sparse on the relationship between diaphragm training and neuro-coordination response in COPD patients.³³

In light of the above, the clinician and rehabilitation field and scientific research should place greater emphasis on the assessment and training of the diaphragm, not only with the goal of slowing the progression of the disease or helping the patient in the acute phases, but as a target to prevent the occurrence of accidental falls.

Conclusions

Chronic obstructive pulmonary disease (COPD) causes deterioration of the airways, with persistent and non-reversible airflow limitation. COPD could become the third leading cause of death for the population by 2030. There are several causes that lead to structural and functional alterations of the lungs, many of which could be counteracted before developing the disease, such as lifestyle and increased attention in avoiding some daily habits, such as physical activity and cigarette smoking, respectively.

The skeletal muscles of the trunk, the limbs and the diaphragm muscle undergo non-physiological adaptations over time, which worsen the patient's clinical picture and lead to an increase in accidental falls. The article reviewed the literature concerning the pathological adaptation of the diaphragm, placing emphasis on the spino-cortical and cortico-spinal neurological relationships that influence the management of proprioceptive information, and how diaphragmatic dysfunction can alter the neuro-coordination of the COPD patient. A greater interest of the clinician and the physiotherapist should be directed towards the diaphragm to counteract the genesis of unwanted falls.

Ethics Statement

The people involved in the figures to accompany the article have consented to the publication of the images.

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Disclosure

The authors report no conflicts of interest in this work.

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