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LETTER

Updated Perspectives on the Role of Biomechanics in COPD: Considerations for the Clinician [Letter]

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Dear editor

I read with interest the article by Yentes et al.[1] The text emphasizes the importance of the extrapulmonary component in chronic obstructive pulmonary disease (COPD), and directs the clinician's interest towards biomechanics ("forces that act upon a body and the reactions produced").[1] Skeletal musculature in COPD patients adapts with phenotypic, morphological and functional non-physiological changes, which adaptations do not always reflect the speed of pulmonary changes, or do not always correlate with the severity of the disease.[1] Skeletal muscle mass reduces volumes (atrophy), and there is a shift in contractile fibers from an aerobic to anaerobic metabolism, where white or type II fibers increase, with parallel decrease in red or type I fibers.[2] Although type II fibers have a greater ability to increase volume (hypertrophy) and a greater ability to express contractile force than type I fibers, such adaptation in COPD does not occur.[2]

One of the possible causes is the presence of a local and systemic inflammatory environment, which stimulates canonical nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) pathway and non-canonical NF-κB pathway. In the presence of inflammation, oxidative stress or antigens, NF-κB facilitates the nuclear transcription of its heterodimers (p65 and p50); this step will stimulate protein degradation and inhibition of correct mechanotransductive responses.[3] Non-canonical NF-κB pathway uses, in particular, B-cell lymphoma 3-encoded protein (Bcl3) to induce alterations to the cell nucleus, along with p50 and p52.[3]

Pathological adaptations occur not only in the skeletal muscles of the limbs. The diaphragm muscle, the main muscle respiratory, exhibits numerous non-physiological changes in COPD. The diaphragm has an inspiratory attitude compared to healthy subjects, a greater thickness (probably due to a progressive shortening of the contractile fibers), and a decrease in white fibers with a parallel increase in red fibers.[4] The ability to produce strength and resistance is decreased, with an alteration of the electrical activity of the phrenic nerve, reflecting neuropathic adaptations.[4]

A fundamental concept that the authors of the article did not take into consideration is that of remembering that the force expressed by the limbs depends on the diaphragm and, therefore, secondarily influencing the values expressed by biomechanics.

Each respiratory act stimulates the sending of information deriving from receptors linked to the proprioceptive and exteroceptive sphere, passing through the nucleus tractus solitarius (NTS).[5] This information, essential for the correct representation of the body at the cortical level (and for adequate motor control), is shared with the cerebellum and the vestibular area to finally send enriched afferents towards the limbic area and the cortical motor area M1.[5] From the same higher centers, efferences for NTS return. New information will start from the NTS that will induce inhibition for the sympathetic system (at the level of the ventrolateral medullary rostral area) and stimulate the parasympathetic system.[5] The result is better neuro-coordination and a better expression of muscle strength.[5]

The diaphragm muscle should be considered as the first option (and not underestimated or forgotten) to correctly frame the neuro-motor behavior of the limbs and trunk.

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Disclosure

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