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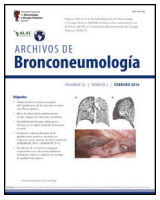

Editorial

Inspiratory Muscle Training: Back to Basics Must be the First Step?

The diaphragm is considered the primary respiratory muscle as it is responsible for generating the intrathoracic negative pressure that facilitates ≈2/3 of lung volume change during inspiration.[1–3] However, a broad range of pathologies and clinical situations may alter its composition and function, impeding normal ventilation. Respiratory muscle wasting consists of a devastating neuromuscular, both peripheral nerve alterations and myopathic injury, which can be triggered by several factors such as inflammatory response, malnutrition, neuronal axon degeneration, changes in muscle histology and immobilisation.[4] For instance, in the intensive care unit (ICU) environment, critically ill patients on mechanical ventilation often develop significant and rapid diaphragmatic atrophy, with diaphragm thickness reductions of up to 10% in the first 4 days from admission,[5] and this continues after weaning from mechanical ventilation.

In recent years, there is increasing evidence suggesting that inspiratory muscle training (IMT) may counteract diaphragm deterioration and improve clinical outcomes, especially in critically ill patients or with COPD where it has been shown to be safe. Indeed, studies report that incorporating inspiratory training by the use of a threshold valve as a complementary intervention in pulmonary rehabilitation could improve muscle strength in COPD patients although key outcomes are varied.[7] Nevertheless, it is important to emphasise that the physiological basis of training is often not considered among current evidence and there is no consensus on how to train the diaphragm adequately. Indeed, for example, exercise prescription and optimal approach to dose–response adaptation, breathing pattern (in terms of volume and time), and progression in ICU patients remains unclear[6,8] due to the heterogeneity of training regimens. High-intensity threshold interval or endurance training methods are often used, with loads set between 20 and 50% of the patient's inspiratory strength or maximum tolerated value. Unfortunately, there is no consensus or clear physiological basis for choosing one training programme over another based on both inspiratory force and load, sets and frequency.

Muscle motor units are classified based on their mechanical, metabolic and fatigue properties, and fibre type composition. The diaphragm is known to have a higher proportion of oxidative muscle fibres since ≈80% of the fibres are type I and IIa fibres, with type I constituting up to 50% of the fibres.[9,10] This leads to a marked functional difference in terms of neural activity, metabolism, and energetic demands, being a muscle with high endurance capacity and fatigue resistance,[10] which correlates with the cyclical task of diaphragm recruitment and activation during low-load activities such as resting breathing. Indeed, during normal breathing, type I and IIa fibres are more active (approximately 40%) than type IIb fibres (1% of the total).[2]

According to Henneman's size principle on the characteristics of motor neurons and their muscle fibre size, low to moderate intensity activities recruit mainly type I fibres, whereas type IIa and IIb fibres are progressively recruited as intensity increases.[11] The neural recruitment pattern among the different diaphragmatic fibres follows this key order to generate different motor behaviours ranging from normal ventilation up to intense efforts.[1,2] However, it is worth noting that in the diaphragm, maximum diaphragm fibre recruitment, and therefore activation of all motor fibres, occurs sporadically during forced expiratory manoeuvres, which play a minimal role compared to normal diaphragmatic function.[1,2] During exercise, in healthy adults, neural activation gradually increases in incremental tests, whereas diaphragm activation typically plateaus at submaximal values ≈<80%max. Indeed, different metabolic demands during submaximal testing produce different amplitudes and plateaus of diaphragm neural activation, with activation at the end of incremental exercise greater than that seen at the plateau at both 80% and 60% constant loads.[12] This suggests that the recruitment output of the respiratory motor is matched to the metabolic demands of the diaphragm. Therefore, theoretically, the intensity and frequency of diaphragmatic exercise should result mainly in modulation of type I and IIa fibre recruitment. Instead, overloading due to higher ventilatory demands may recruit non-diaphragmatic primary and accessory inspiratory muscles, which have different proportions of fibre types and functional tasks.

Following this information, time under load (TUL) is a key element in exercise prescription. Low-load training, compared to high-load training, maintains a constant duration of repetition, and achieves a longer TUL. Low-load training with a high TUL, is essential to activate the entire motor unit and maximise the growth of all muscle fibres.[13] As you approach failure, fast fibres are progressively recruited to help maintain strength. Using a low or moderate load (<60% 1RM) with enough repetitions to approach technical failure results in a similar increase in muscle mass as using high loads (>60% 1RM).[14]

These two elements are crucial for the prescription of peripheral muscular training, which differs from the approach of muscular training by applying different intensities and repetition ranges depending on the type of fibre. We must not forget the muscular failure and the duration of the load, which disturbs the slow fibres. This is of special concern considering the risk for failure of the cyclic

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ventilatory duty of the diaphragm. Therefore, why don't we use the principles to design a respiratory training programme? It is imperative to consider how this complex clinical pathophysiological approach alters the neuromechanical and metabolic principles of the respiratory muscles to optimise IMT. These principles, focused on healthy or stable clinical situations, must be combined with the basic principles of training methods to design, and prescribe tailored IMT protocols in different settings. In peripheral muscle training, different combinations of resistance and/or endurance training have been more extensively studied in terms of load, sets and/or weekly frequency and the effect on fibre neuromechanical or morphological adaptations.[15] However, are the IMT protocols previously evaluated or based and adapted on these solid principles of the inspiratory muscles and the framework of each setting? For instance, recently, Jenkins et al. demonstrated the metabolic cost of IMT in mechanically ventilated patients.[8] This novel approach reflects that there is a progressive VO_2 dose–response relationship with respiratory load during IMT, which is modulated by respiratory strength. Similarly, higher resting VO_2 was associated with better inspiratory strength. This may help to identify patients with better tolerance to high loads or stratify patients in the ICU who can tolerate higher respiratory loads during IMT and avoid respiratory muscle task failure.

Based on the information gathered, IMT appears to play a significant role in improving diaphragm strength and function although heterogeneity in IMT programs is present in most studies nowadays. Exercise prescription, considering the basics of muscle physiology, with TUL and training close to muscle (or each fibre type) failure, seem to be key in improving muscle performance conditioning. Thus, should these principles be used also during IMT? To date, there is not enough evidence to train respiratory muscles in that manner since previous studies have not considered these physiological dimensions or were limited by methodological design limitations in applying or tailoring diverse types of intervention. Additionally, special attention should be paid as forcing the diaphragm to work close to failure can be detrimental, particularly in the event of over-exertion. Are we overtraining or undertraining the diaphragm? It is impossible to robustly answer this question, but for sure we must consider the physiological muscle basis in the methodology of an inspiratory muscle training study. The challenge for an effective tailored IMT prescription in terms of set load and frequency will be to identify the responder with a better tolerance to appropriate loads, and to reduce or control overloading of fibre fatigability. Therefore, going back to basics must be the first step.

Statement of Ethics

We complied with the guidelines for human studies and our research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Information revealing the subject's identity is to be avoided.

Authors' Contributions

We were all involved in writing and reviewing the manuscript.

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Conflicts of Interest

The authors have no conflicts of interest to declare.

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