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# Inspiratory muscle training in patients with obstructive sleep apnoea

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Dear Editor:

We have read with great interest the article written by Huei-Chen Lin and colleagues entitled “The effects of threshold inspiratory muscle training in patients with obstructive sleep apnea: a randomized experimental study” [1]. The authors designed an intervention study in 22 patients with moderate-to-severe obstructive sleep apnoea (OSA). Sixteen patients were randomly assigned to a threshold inspiratory muscle training (TIMT) group and 6 to a control group. The target threshold of TIMT was adjusted every week to maintain the threshold to reach 30% of each patient’s maximal inspiratory pressure (MIP). Post-treatment apnoea-hypopnea index (AHI) and Epworth sleepiness scale (ESS) scores were significantly lower: 6% and 20.2%, respectively ( $p < 0.05$ ).

In the last decade, comprehensive treatments that include physical training or oropharyngeal exercises [2] or both [3] have been suggested for lowering the AHI in OSA patients. Possible benefits of these exercises include a reduction in neck circumference, snoring, subjective sleepiness, and AHI, as well as an improved quality of life [2]. Especially, the genioglossus and pharyngeal muscles, that play an essential role in OSA, can be trained through oropharyngeal exercises [3].

The authors described a training protocol of inspiratory muscles; however, it is essential to consider that the respiratory muscles training must be followed with physiological

principles of overload, specificity, and variability to achieve favourable results. Nevertheless, the frequency of training and duration continue to be a matter of discussion. The key to prescribing training is the correct dosage of its intensity or load, as well as the total time or volume of training [4].

The maximal respiratory mouth pressure is the most used test to assess respiratory muscles. From this assessment, reference values have been established for a healthy population and countless publications that provide values for different diseases. Maximum respiratory pressure assessment is necessary to establish the initial intensity of training and to adjust the load during the training weeks [4].

Lin and colleagues did not assess the MIP value, so the degree of muscle weakness or the training load cannot be determined. They reported that the initial setting range of target threshold was between 11 and 21 ( $14 \pm 6$ ) cmH<sub>2</sub>O. This load was programmed with a protocol which measured 10 repetitions maximum (RM), with intensity reported by the patient between 13 and 15 of exertion rating [1]. The authors cited Kline and colleagues for this assessment method [5], but these authors assess the MIP value, as the literature recommends [4]. Moreover, the device used to assess of RM has a maximal pressure of  $-41$  cmH<sub>2</sub>O, well below the values shown by OSA patients [5] and values of healthy subjects [4]. Hence, these instruments are recommended for the measurement of the entire spectrum of healthy people and patients.

Another critical point is the protocol used. The authors described that the training was done with 30% of each patient’s MIP. Furthermore, the literature has consistently shown in different types of pathologies that the threshold type devices with loads higher than 30% produce improvements in the strength and resistance of the respiratory muscles [4]. However, Lin and colleagues used a target threshold between 11 and 21 cmH<sub>2</sub>O. If we think that these loads correspond to a training load of 30% as described by the authors, 14 of the 16 subjects had MIP less than 50 cmH<sub>2</sub>O (only two subjects trained with a load higher than 15 cmH<sub>2</sub>O). Although the authors mention the non-measurement of muscle strength as a limitation, they admit that they could not explore the

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mechanisms of TIMT with respiratory muscle function. However, they did not consider an important aspect: that without measuring MIP, the training, that is the main intervention of this study, cannot be correctly prescribed.

Different authors assessed the MIP in patients with OSA. The values reported were much higher than those described by Lin and colleagues. For example, Kline reports a mean of  $94.2 \pm 7.6$  cmH<sub>2</sub>O [5]. If we hypothesise that patients of the Lin group had similar MIP pressures, the programmed training load was extremely low to produce any physiological effect. Consequently, the analysis of responsiveness, responders vs non-responders, could not be attributed to the TIMT effects.

Moreover, the outcome of the results is very heterogeneous. The literature describes that general and specific training is dosage-dependent, but when we analyse the results of Lin and colleagues, we observed unexpected results [1]. For example, subject 16 (42 years old) was trained for 106 days and showed an increase of 35.5% of AHI, while subject 2 (21 years old) was trained for 73 days and showed an increase of 55.8% of AHI. On the other hand, subject 10 (42 years old) was trained for 28 days and showed a decrease of AHI of 30.8%. We understand that there may be responders and non-responders, but we think that to obtain reliable results, we need to compare equivalent training loads.

We think that training the respiratory muscles in patients with OSA and incorporating physical training associated with comprehensive rehabilitation programmes have an enormous potential. Consequently, they should be part of the therapeutic strategies of people who have OSA, especially those with associated cardiovascular comorbidity. However, the

prescription of exercise should always be based on the scientific basis of training and the use of objective methods to achieve the best possible effect.

## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

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