Editorial Comment

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Fit Yourself and Take Your Lungs to Heart

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It has been estimated that physical inactivity is worldwide responsible for 6–10% of the major non-communicable diseases. Furthermore, sedentary lifestyle causes 9% of premature mortality [1]. Moreover, recent studies have consistently shown that messages emphasizing the benefits of being active are more effective at changing physical activity behaviour than those stressing the consequences of inactivity [2].

Chronic obstructive pulmonary disease (COPD) is a complex disease mainly characterized by structural abnormalities of the airways and lungs, but it is very often associated with concomitant comorbidities. The presence of comorbidities strongly influences not only the severity of symptoms, but also the risk of hospitalization and death [3]. The relationship between COPD and cardiovascular disease is particularly notable and of clinical relevance, as cardiovascular disease represents the most common comorbidity and the leading cause of hospitalization in patients with mild-to-moderate COPD [4].

Exercise-based pulmonary rehabilitation is a well-established intervention for patients with COPD. Physical training, by improving skeletal muscle function, positively influences exercise tolerance and symptoms; this is mainly due to a reduction in lung dynamic hyperinflation but also to a desensitization to central dyspnea [5]. There is now emerging evidence to support the efficacy of exercise-based pulmonary rehabilitation also in the management of COPD exacerbations. An up-to-date Cochrane meta-analysis shows that pulmonary rehabilitation significantly reduces odds of hospital admissions and deaths following acute exacerbations, as well as consistently improves the quality of life and exercise tolerance of COPD patients [6]. However, no prospective studies on the role of physical rehabilitation in influencing the number and severity of future exacerbations have been performed yet.

The paper by Ramponi et al. [7], published in this issue of *Respiration*, aims to assess whether a 9-week pulmonary rehabilitation program may affect cardiovascular response to exercise in COPD patients. In an observational prospective trial, according to the ATS/ERS recommendations, 27 patients with COPD were referred to a rehabilitation program consisting of 3-hour sessions, three times a week, with a minimum of 21 sessions required. Data obtained show a significant improvement in maximal exercise tolerance, such as peak oxygen uptake, and in some cardiovascular parameters following rehabilitation. Leg fatigue was also significantly reduced, supporting the reported positive effect of physical exercise on muscle function. The novel aspect of the study is to compare cardiopulmonary exercise variables at 'submaximal' exercise levels, isometabolic and isoventilatory levels ('isolevels') before and after rehabilitation. Of note, the O_2 pulse (peak oxygen uptake/heart rate) and tidal volume were significantly higher after rehabilitation. Furthermore, tidal volume changes correlated significantly with changes in O_2 pulse. The authors concluded that the most likely explanation for their observations was an improvement in cardiovascular function due to a reduction

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in lung hyperinflation induced by exercise training. The O_2 pulse, as originally described many years ago, is the product of the stroke volume times the arterial mixed venous O_2 differences ($CaO_2 - CvO_2$) [8]. Thus, an improvement in O_2 pulse may be due to an increase in stroke volume, $CaO_2 - CvO_2$, or both. As suggested by the authors, the observed increase in O_2 pulse very likely reflects an improvement in cardiovascular function, although a small contribution to an increased O_2 peripheral muscle extraction after rehabilitation cannot be excluded. In their paper, the authors also reported significant changes, after rehabilitation, in the oxygen uptake efficiency slope and heart rate recovery following exercise; these improvements, although difficult to be interpreted, may also indicate an amelioration of cardiovascular function induced by pulmonary rehabilitation. Further studies are needed to clarify these aspects.

The study by Ramponi et al. [7] has the merit to highlight the relevance of cardiopulmonary exercise testing while simultaneously recording integrated parameters of ventilatory, cardiac and metabolic responses to exercise. Such a comprehensive evaluation appears particularly useful not only for diagnostic purposes, but also for monitoring the effectiveness of pharmacological and non-pharmacological interventions, as well as for providing prognostic indexes [9].

Apart from the need for further studies to assess the carry-over positive effects of pulmonary rehabilitation in COPD patients, as correctly outlined by the authors, we strongly suggest to address future investigations on the role of different COPD phenotypes and the presence of comorbidities in influencing patient responses to physical training programmes. In fact, as already shown by our group in emphysematous patients [10], cardiopulmonary responses may differ in specific clinical phenotypes, suggesting that a personalized medical approach might also be extended to pulmonary rehabilitation in COPD patients.

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