Differences between exercise capacity and physical activity in COPD: from the lungs to the periphery

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SUMMARY

Chronic Obstructive Pulmonary Disease (COPD) is a progressive disease affecting primarily the lungs. However, an important proportion of patients present deleterious extra-pulmonary effects, the so-called “systemic effects”. Muscle dysfunction and wasting constitute one of the most relevant systemic effects associated to exercise tolerance limitation, increase in cost of the disease and an independent morbi-mortality prognosis factor.

Abnormalities in lung and muscle function contribute to limit exercise capacity in COPD. This increment the ventilator equivalents during exercise, which transform exercise into an unpleasant experience due to symptoms such as dyspnoea and muscle fatigue. This situation, together with psychological factors such as depression – affecting around 20-40% of patients with COPD – and behavioural aspects contribute to establish a sedentary habit.

Inability to perform exercise (reduce exercise capacity) is therefore related to different physiological aspects related to the lung, peripheral muscles and heart function. Among lung factors contributing to reduce COPD patients exercise capacity are ventilatory limitation, the development of dynamic hyperinflation during exercise and gas exchange abnormalities. We know today that the reduced exercise capacity, classically attributed almost exclusively to the altered lung function characteristic of the patients with COPD, can also be determined by other extra-pulmonary factors. Muscle dysfunction is one of these factors. This is characterised by loss of muscle strength and endurance. The latter is inversely related to the increase fatigability of the muscle of these patients. These physiological factors are related with a number of pathogenic mechanisms such as reduced oxidative capacity of the muscle, re-distribution of muscle fibres (reduction of the proportion of Type I fibres with increase of Type II fibres), reduced activity of oxidative enzymes, altered capillarization of the muscle and net loss of muscle mass. As a result of this, Type II fibres are early recruited during exercise once most of Type I fibres, the first to be recruited during exercise, are active. As a result of this and other factors, patients with COPD are characterised by an early lactate release during incremental exercise. This acid is rapidly buffered by the carbonic acid tampon system increasing the availability of CO2 during exercise. As a result of this the ventilator demand is increase in patients with COPD at equal work-load in comparison to healthy controls, which accelerate the consumption of the ventilator reserve and result in an early termination of exercise.

Other factors contributing to a reduced exercise capacity in COPD are cardiovascular factors. Cardiovascular comorbidities are one of the most frequent comorbid conditions associates with COPD. When present, Heart failure contributes to the exercise intolerance. In turn, interaction between lung and heart during exercise have also been described in patients with COPD, particularly in those developing dynamic hyperinflation during exercise. The increase in intra-thoracic pressure can compromise the venous return to the right heart chambers reducing the end-dyastolic volume and, consequently, the stroke volume.
Exercise intolerance is an independent prognosis factor for survival, frequency of hospitalisations and outcomes after lung volume reduction surgery in COPD.

**Physical activity in COPD**

It is important to differentiate physical activity from exercise capacity. An interesting example of this difference is highlighted by the fact that changes in one of these concepts can be dissociated from the other. We can successfully increase exercise capacity with interventions such as exercise training, the cornerstone component of pulmonary rehabilitation (PR) programmes, with little or no effect on changes in physical activity levels (PAL). In fact, only 50% of PR studies evaluating the effect of PR on physical activity levels showed increments in PAL. Other factors are relevant to explain why, beside the fact that exercise capacity increase after PR, patients “choose” to remain inactive. An interesting study by Pitta et al showed that only after 6 month of PR effects in exercise capacity are being translated to increments in PAL. This suggest that changes in behaviours require more time that PR induced physiological changes in the muscle.

Clearly exercise intolerance limits the ability of patients to exercise during daily life having an impact on PAL. However, other factors such as mood, motivation or depression influence PAL in patients with similar exercise capacity.

All these factors contributing to limit exercise capacity (e.g. lung, muscle or heart factors) and physical activity (e.g. same factors plus mood, motivation and depression), contribute to the vicious cycle of inactivity – deconditioning – increase symptoms, characteristic of COPD.

Physical activity is conventionally defined as any bodily movement produced by skeletal muscles that result in energy expenditure. It is a complex behaviour characterised by type, intensity, duration, patterns, and symptoms experience. It includes, but it is not limited to leisure-time, domestic and occupational activities.

In turn, exercise constitutes a subtype of physical activity that is planned, structured, repetitive and purposeful.

We conventionally measured exercise capacity in patients by pushing patients to sustain exercise throughout time using different exercise protocols (e.g. incremental or constant work-rate protocols). While we can assess the capacity to exercise, these protocols give very little information on what the patient is actually doing in real life in terms of activity levels.

If we measure PAL (e.g. using questionnaires, monitors or hybrid tools such as the PROactive tools) we find that PAL are reduced in patients with COPD. Moreover, PAL is marker of disease severity and health status. Patients suffering from exacerbations of the disease (ECOPD) further reduce their PAL. Interestingly, studies assessing PAL recovery after ECOPD found that PAL after 30 days of an ECOPD remain far below PAL in stable patients.

PAL constitutes a prognosis factor for mortality and hospitalisations. These classical studies have been more recently reproduced.

A more recent study has shown that PAL are possibly the most powerful predictor of mortality before lung function, exercise capacity, body composition, or quality of life measurements. These support the need to develop strategies to increase PAL in patients with COPD. Pulmonary rehabilitation programmes should, therefore, include strategies to increase PAL and translate the improvements in muscle function and exercise capacity achieved into a more active life style.