Introduction: During whole body exercise, respiratory muscle work may affect locomotor muscle oxygenation and local blood flow and consequently reduce exercise capacity [1]. Respiratory muscle training using normocapnic hyperpnoea exercise (NHE) is a promising strategy to improve exercise tolerance in patients with COPD.

Aims: To investigate the acute effects of NHE on intercostal and locomotor muscle oxygenation in COPD.

Methods: Eight patients with COPD (FEV1 = 55 ± 8% of pred) performed a NHE at 50-60% of peak ventilation (VEpeak) assessed during maximal cycling test. The NHE was conducted to exhaustion, using a respiratory device (SpiroTiger®) connected to a gas analyzer to monitor (VE, VO2 and PETCO2). Cardiac output (CO) and intercostal and vastus lateralis muscle oxygenation were continuously measured during exercise by means of finger photoplethysmography and NIRS, respectively. Arterial blood gases (PaCO2) and inspiratory capacities (IC) were obtained at rest and at end-exercise.

Results: The NHE was maintained for 653 ± 397s, at a VE averaging 25 ± 10 l/min or 52 ± 5% of VEpeak and with a VO2 corresponded to 52 ± 6% of VO2peak. Intercostal muscle deoxyhemoglobin and total hemoglobin increased by 14 ± 6% and 13 ± 8% respectively (p<0.05) while they remained unchanged in the vastus lateralis muscle (p=NS). Compared to resting values, there was no change in PETCO2, PaCO2, IC and CO during NHE (p=NS).

Conclusions: NHE promotes muscle deoxygenation in the intercostal muscles but not in the vastus lateralis but it was not accompanied by changes in CO or dynamic hyperinflation in patients with COPD. Our results suggest that NHE stimulates the intercostal muscles, which could potentially improve exercise tolerance following a NHE training.

References: