CHANGES IN NEUROMUSCULAR RECRUITMENT DURING EXERCISE IN ACUTE HYPOXIA

Ansley Les 1, St Clair Gibson Alan 2, Hunter Angus 3, Neary Patrick 4, Skowno Justin 5, Noakes Timothy 6

(Kingston University 1, United Kingdom, University of Northumbria 2, United Kingdom, University of Stirling 3, United Kingdom, University of Regina 4, Canada, Great Ormond Street Hospital 5, United Kingdom, University of Cape Town 6, South Africa)

Physical work capacity is significantly impaired in hypoxia. Two opposing theories have been advanced to explain this. One theory holds that this results from a reduction in maximal cardiac output since hypoxia limits the intrinsic pumping capacity of the heart (2). But there is no evidence that myocardial function is impaired even during maximal exercise at extreme altitude (5). Alternatively, Kayser et al. (3) have reported that the electromyographic (EMG) activity in the exercising muscle is reduced during maximal exercise in hypoxia. More recently Amann et al. (1) showed that EMG activity in the rectus femoris muscle during 5km cycling time trials falls with increasing levels of hypoxia. These studies suggest that skeletal muscle recruitment is reduced during exercise in hypoxia.

To evaluate the effect of inspired oxygen fraction (F1O2) on cardiac output and muscle recruitment eight highly trained cyclists performed two 20-km cycling time trials in an atmospherically-sealed chamber in which inspired O2 concentration was altered mid-way through the exercise bout. The F1O2 either decreased from 0.21 to 0.14 (21-14) or increased from 0.14 to 0.21 (14-21). The order of the trials was randomised and single-blinded. Trials were completed on an electrically-braked cycle trainer (Computrainer Pro), which provided continuous power output (PO) data. Real-time cardiac output (Q) was measured using bolus lithium indicator dilution (LiDCOTM) (4). EMG activity of the rectus femoris muscle (RF), as a measure of central activation, was sampled during a 10-s epoch every 1km.

Mean Q in the 14-21 trial was significantly higher than during the 21-14 trial (33.9 ± 5.1 L/min vs 29.6 ± 6.0 L/min; p=0.007). Power output for the first 10km was higher in the 21-14 trial (254 ± 29W) compared to the 14-21 trial (210 ± 32 W) (p=0.016) and decreased significantly from the first 10km to the second 10km in the 21-14 trial (205 ± 37 W); (p=0.008). Although PO increased in the second 10 km of the 14-21 trial (230 ± 37 W), this change was not significant. EMG of the RF tracked the changes in PO during the time trials.

Cardiac output was higher in the first 10km in hypoxia when PO was lower. Hence cardiac output was neither constrained by hypoxia nor did the magnitude of the cardiac output determine performance. Rather changes in PO tracked the changes in EMG activity. This suggests that PO during the time trials was regulated principally by changes in the neural recruitment of the motor units in the exercising muscles as also shown by Amann et al. (1).


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