Effect of graded hypoxia on supraspinal contributions to fatigue

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Fatigue, defined as exercise-induced impairment in the ability to produce muscle force, can originate from peripheral (intramuscular) and central (neural) mechanisms. Supraspinal fatigue, a component of central fatigue, has been defined as an exercise-induced decline in force caused by suboptimal output from the motor cortex (1). Supraspinal fatigue accounts for about one-quarter of the force loss after fatiguing contractions of the knee extensors during normoxia (2). The aim of this study was to evaluate the relative contribution of supraspinal processes to fatigue in response to different severities of acute hypoxia. We hypothesised that the supraspinal contribution to fatigue would be elevated in line with increasing severities of hypoxia. On separate days, 11 healthy men performed sets of intermittent (5 s contraction, 5 s relaxation), isometric, quadriceps contractions at 60\% MVC to task failure during normoxia (FIO\textsubscript{2}/SpO\textsubscript{2}[%] = 0.21/97.8), mild hypoxia (0.16/93.2), moderate hypoxia (0.13/84.8) and severe hypoxia (0.10/74.2). Twitch responses to femoral-nerve stimulation and transcranial magnetic stimulation were obtained to monitor peripheral force-generating capacity and cortical voluntary activation (VA), respectively (2). Arterial and cerebral/muscle oxygenation were estimated using pulse oximetry and near-infrared spectroscopy, respectively. Force-generating capacity and cortical VA were unaffected by hypoxia at baseline. Time-to-task failure (mean ± SD) ranged from 24.7 ± 5.5 min in normoxia to 15.9 ± 5.4 min in severe hypoxia (P<0.05 vs. normoxia and mild hypoxia). MVC and quadriceps twitch force were significantly reduced in all conditions immediately post-exercise. The decrease in quadriceps twitch force was less in severe hypoxia vs. normoxia (20 ± 20 vs. 34 ± 17%; P = 0.02). Cortical VA was significantly reduced after exercise in all conditions; however, the deficit in cortical VA was larger in severe hypoxia versus normoxia (P<0.05). Of the ≈30\% decrease in MVC observed in all conditions, supraspinal fatigue accounted for 18\% in normoxia, 25\% in mild hypoxia, 24\% in moderate hypoxia and 54\% in severe hypoxia. The dominance of supraspinal fatigue in severe hypoxia may have been due to cerebral deoxygenation (73 to 57\% pre vs. end-exercise; P<0.01). In conclusion, peripheral mechanisms of fatigue contribute relatively more to the reduction in force generating capacity of the knee-extensors in normoxia and mild-to-moderate hypoxia, whereas supraspinal fatigue plays a greater role in the force reduction during severe hypoxia.

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References