Respiratory muscles, O_2 transport and fatigue

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Healthy fit humans were studied to determine the effects of exercise-induced Hb O_2 desaturation and respiratory muscle work on: a) endurance exercise performance; b) locomotor muscle and diaphragm fatigue; and c) limb blood flow. Cycle exercise to exhaustion at 90% VO_{2max} (10 – 16 min) caused: a) time-dependent but variable HbO_2 desaturation (SaO_2 85 – 93%), due primarily to metabolic acidosis and increased temperature; b) significant fatigue of the diaphragm and of the quadriceps muscles as determined by supramaximal paired magnetic (1-100 Hz) and/or electrical (1 – 30 Hz) phrenic femoral or nerve stimulation; and c) progressive increases in perception of limb and breathing discomfort. Exercise performance time was enhanced (+ 12 to 20%) and effort perception reduced by preventing HbO_2 desaturation (when SaO2 was ≤ 93%) via raising F_IO_2 to .25 to .29 or by reducing the work of breathing by 40 to 60% via use of a proportional assist ventilator. When comparisons are made among experimental and control conditions at equal exercise times and work rates, the increased performance and reduced effort perceptions (when SaO_2 was maintained or ventilatory work reduced) coincided with: a) less locomotor muscle fatigue; b) prevention of diaphragmatic fatigue; and/or c) reduced plasma lactate concentrations. Alternatively, increasing inspiratory airway resistance, while maintaining SaO_2, reduced performance time and exacerbated limb muscle fatigue. We conclude that exercise-induced arterial desaturation (SaO_2 ≤ 93%) or high levels of respiratory muscle work and diaphragm fatigue that normally accompany high intensity endurance exercise under normoxic ambient conditions in healthy subjects, contribute significantly to the limitation of endurance exercise performance and to the perception of limb discomfort and dyspnea. In turn, these performance limiting influences are due in part to locomotor muscle fatigue secondary to reduced O_2 transport to limb muscles, because of either: a) sympathetically-mediated limb vasoconstriction and reduced blood flow due to a metaboreflex elicited from fatiguing respiratory muscles; or b) reduced SaO_2. (Funded by NHLBI and the American Heart Association.)