

Muscle oxygenation during prolonged electrical stimulation-evoked leg cycling in paraplegics

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Abstract

This study investigated cardiorespiratory responses and muscle oxygenation during prolonged electrical stimulation (ES)-evoked leg cycling in individuals with paraplegia (PARA). Four PARA and 6 able-bodied persons (AB) participated in this study. Subjects performed 10-min of passive cycling and 40-min of active cycling (PARA: ES-cycling, AB: voluntary cycling) at workloads selected to elicit an equivalent oxygen uptake between groups. Oxygen uptake was similar in PARA (737 ± 177 ml·min⁻¹) and AB (840 ± 90 ml·min⁻¹) during exercise. The average cycling power output for PARA was initially 9W, but varied considerably over 40-min. In contrast, power output at similar oxygen uptake was 43W in AB. PARA demonstrated lower gross mechanical efficiency (~1.3%) during ES-cycling compared to AB performing voluntary exercise (~12.6%). During ES-cycling, muscle oxygen saturation (SO₂) decreased to approximately $72 \pm 19\%$, while SO₂ during volitional cycling was unaltered from resting levels. Deoxygenated haemoglobin initially rose during the first 5-min of ES-cycling, and remained elevated by 28% thereafter. During ES-cycling, muscle oxygenation followed a pattern similar to that observed in prior studies that have involved prolonged volitional cycling. However, because forces during ES-cycling were low, inferred intramuscular pressures were also small, perhaps allowing blood volume in the legs to increase.

1. INTRODUCTION

Computer-controlled electrical stimulation (ES-evoked) leg cycling enables dynamic exercise of the paralyzed lower limbs in persons with SCI. During prolonged exercise, effective oxygen delivery and extraction within the muscles is necessary to maintain aerobic metabolism. However, muscle atrophy of the paralyzed lower

limbs, also results in a poor lower limb blood flow, a decreased vascular bed, and reduced venous ‘muscle pump’ activity [1]. Therefore, it has been speculated that persons with paraplegia might have impaired oxygen delivery and extraction within their working muscles during prolonged ES-cycling, compared with prolonged voluntary cycling by able-bodied individuals.

The purpose of this study was to quantify muscle oxygenation in the lower limbs of individuals with paraplegia during prolonged ES-cycling. To accomplish this, the following objectives were proposed: 1) To compare muscle oxygenation between passive and ES-cycling in persons with paraplegia; and, 2) To compare muscle oxygenation during ES-cycling in persons with paraplegia to voluntary cycling by able-bodied subjects.

2. METHODS

Four males with neurologically-complete paraplegia (PARA; ASIA-A, T₅-T₁₂) participated in this study and each subject had been post-injury for at least 3 years. The mean age (\pm SD) of PARA was 35 (\pm 11) yr. Six able-bodied males (AB; 30 \pm 10 years) were also recruited for comparison. Following medical screening, all subjects gave their written informed consent, as required by the University of Sydney Human Research Ethics Committee.

PARA and AB were assessed during four measurement phases, carried out in the following order; 1) REST, 2) passive leg cycling at 50 rev·min⁻¹ for 10 min (PAS), 3) prolonged active cycling, comprising either ES-leg cycling (ES-LCE) by PARA or voluntary cycling by AB (VOL-LCE) for 40-min, and, 4) 10-min of post exercise recovery (REC). All leg exercise trials were conducted using an ERGYS-1 leg cycle ergometer (Therapeutic Alliances, Dayton, OH). In PARA, the power output of ES-cycling was set to 6, 12 or 18 W depending on individual’s

muscle strength and cycling ability. The power output of voluntary cycling by AB was preset to 43 W, which elicited an oxygen uptake ($\dot{V}O_2$) similar to PARA during ES-cycling.

During all trials, oxygen uptake ($\dot{V}O_2$), expired ventilation (VE), and respiratory exchange ratio (RER) were measured by computerised open circuit spirometry. Heart rate (HR) was monitored continuously from the electrocardiogram via an ECG. Stroke volume (SV) was measured using trans-thoracic impedance cardiography using techniques previously described [2]. The product of SV and HR determined cardiac output (CO). Total peripheral resistance (TPR) was calculated as $MAP \div$ cardiac output.

A spatially resolved spectroscopy-based NIRS photometer (OM-200, Shimadzu, Tokyo, Japan) was employed to assess the muscle oxygenation and blood volume in the portion of the exercising muscles under the NIRS probe. The photometer calculated changes (in arbitrary units; a.u.) in oxygenated hemoglobin and myoglobin (oxy-Hb) and deoxygenated hemoglobin and myoglobin (deoxy-Hb). Total hemoglobin and myoglobin (total Hb) in a.u. and tissue oxygen saturation (SO_2) were calculated as previously described by Muraki et al. 2004. The NIRS probe was firmly attached approximately 10-15 cm above the knee over the belly of the right vastus lateralis muscle.

Before cycling, super-systolic arterial occlusion interrupted arterial blood flow via a pneumatic tourniquet on the thigh at a pressure of approximately 250 mmHg for 6 to 10 min. The resting levels of oxy-Hb and SO_2 were arbitrarily defined as 100%, and the nadir values during the occlusion were set to cipher. Similarly, the resting level of deoxy-Hb was defined as 0% and the plateau value during the occlusion as 100%. Thus, arbitrary units of photometer-calculated changes in NIRS data were 'normalized' within the 0% to 100% range.

For the normalised values of muscle oxygenation, a one-way ANOVA with repeated-measures was conducted to detect the effect of time or mode within groups. If a simple main effect or significant interaction was present, a paired t-test was performed to compare the difference between groups at corresponding time points. Statistical significance was accepted if $p < 0.05$. All data are presented as mean \pm S.D.

3. RESULTS

During 40-min active cycling, $\dot{V}O_2$, VE and cardiac output in both PARA and AB were significantly increased compared to REST, and reached a steady-state plateau after 10-min of cycling (Figure 1). Although $\dot{V}O_2$ during steady-state exercise in AB was about $100 \text{ ml}\cdot\text{min}^{-1}$ higher than in PARA, the increases of $\dot{V}O_2$ above resting levels were almost the same by 40 min (PARA: $546 \pm 139 \text{ ml}\cdot\text{min}^{-1}$; AB: $575 \pm 51 \text{ ml}\cdot\text{min}^{-1}$). In PARA, RER increased until about the 10th min of cycling then decreased thereafter until the end of exercise. In contrast, HR in PARA increased progressively throughout ES-LCE, and still had not returned to resting levels after 10 min of REC.

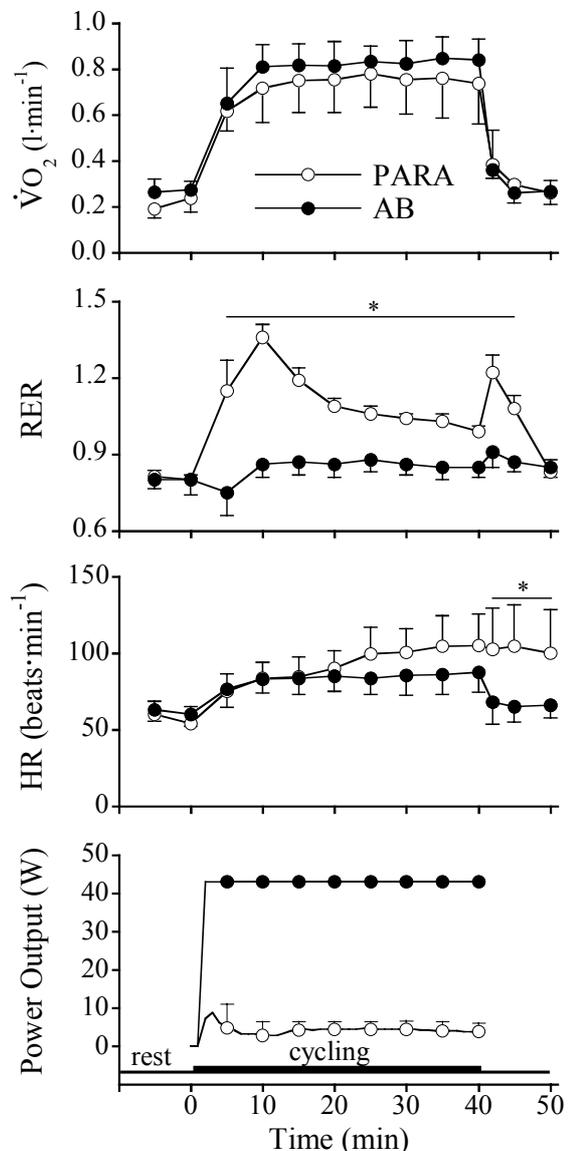


Figure 1. Oxygen uptake ($\dot{V}O_2$), respiratory exchange ratio (RER), heart rate (HR) and power output (PO) during cycling in PARA (ES-LCE) and AB (VOL-LCE). * denotes significant difference ($p < 0.05$) between PARA vs. AB. Data are mean \pm S.D.

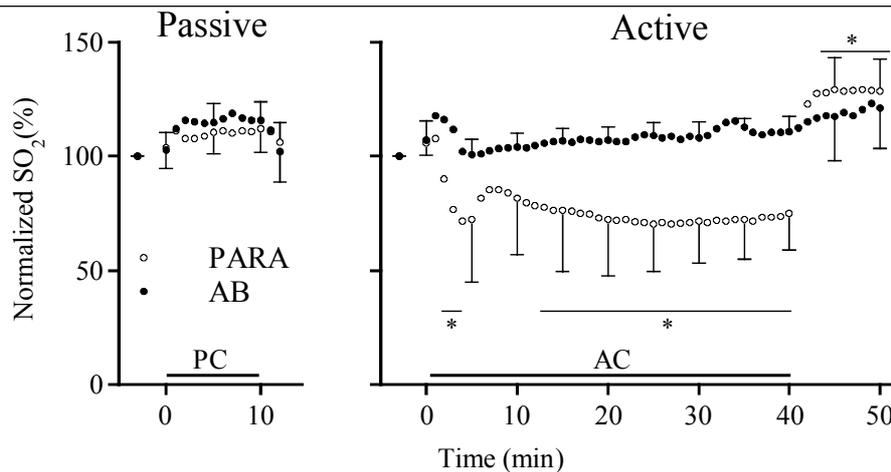


Figure 2. Normalized SO_2 during PAS and active cycling in AB (N = 5) and PARA (N = 4). In PARA active cycling was elicited via ES-LCE. * denotes significant difference ($p < 0.05$) between PARA vs. AB. The horizontal bars (labelled PC & AC) refer to when passive or active cycling took place. Data are mean \pm SD.

Significantly greater power outputs and higher mechanical efficiencies (AB: 43W, 12.6% vs. PARA: 8.8W, 1.3%, $P < 0.05$) were produced by AB compared to PARA during cycling. In PARA, the power output during ES-LCE dropped until the 10th min and then rose to a steady-state value after 15 min (Figure 1).

For the majority of active cycling, significant differences were observed in muscle oxygenation between PARA and AB (Figure 2). Normalized SO_2 during ES-cycling in PARA dropped to 72% ($\pm 28\%$) at the onset of the exercise, thence increased from the 5th until the 7th min to 86% ($\pm 23\%$), but decreased again to a plateau around 72% ($\pm 19\%$) of resting muscle oxygenation. After an initial peak, the AB SO_2 remained near or above resting levels from the 4th min of VOL-LCE. Between the 2nd to 4th min and from the 13th min of cycling, SO_2 was significantly greater in AB than PARA.

4. DISCUSSION AND CONCLUSIONS

ES-cycling by PARA induced significant increases in $\dot{V}O_2$, RER, HR, expired ventilation and cardiac output, but a significant decrease in total peripheral resistance compared to resting values. The cardiorespiratory responses observed during ES-cycling in the current study are similar to those that have been previously reported [2, 3]. The originality of the current study was that muscle oxygenation was measured using NIRS for a prolonged duration of ES-cycling. Scrutiny of the muscle oxygenation responses offered further clues to explain the disparate findings observed

between ES-evoked cycling with paralyzed muscles versus voluntary leg cycling.

Muscle oxygenation was significantly lower during ES-cycling by PARA (70-75%) than during voluntary exercise by AB (105-115%). The lower SO_2 in PARA clearly suggested an insufficient oxygen supply and/or sustained anaerobiosis within the working muscles during ES-LCE. Interestingly, similar decreases in SO_2 have been observed in able-bodied individuals during prolonged voluntary cycling at high exercise intensities, which has been attributed to a complementary decrease in oxy-Hb and increase in deoxy-Hb, reflecting imbalance between oxygen supply and oxygen demand in working muscles.

In conclusion, PARA displayed a different muscle oxygenation pattern during prolonged ES-cycling compared to AB performing voluntary cycling at an equivalent $\dot{V}O_2$. After an initial delay, an equilibrium between oxygen demand and oxygen delivery was eventually reached during prolonged ES-cycling, despite the lack of neural adjustments of leg vasculature in the paralyzed lower limbs.

References

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