

# CARDIOVASCULAR RESPONSES DURING FES LEG CYCLING IN PARAPLEGICS VERSUS ABLE-BODIED INDIVIDUALS

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**Abstract** — This study investigated the cardiovascular responses engendered during functional electrical stimulation-induced leg cycling (FES-LCE) in people with paraplegia (PARA) compared to voluntary leg cycling (VOL) at similar levels of oxygen consumption in able-bodied (AB) individuals. Six PARA with complete spinal cord lesions (T5-T9) and 6 AB participated in this study. Oxygen consumption ( $\text{VO}_2$ ), stroke volume (SV), heart rate (HR), cardiac output (Q) and blood pressure (BP) were measured at rest and during submaximal leg cycling. At the highest cycling intensity achieved ( $9.2 \pm 2.4$  W for PARA and  $42.8 \pm 1.0$  W for AB),  $\text{VO}_2$  was augmented above resting levels to  $0.75 \pm 0.11 \text{ l}\cdot\text{min}^{-1}$  in PARA and to  $0.74 \pm 0.07 \text{ l}\cdot\text{min}^{-1}$  in AB. HR and SV were also increased during FES-LCE in PARA (to  $92.1 \pm 8.6 \text{ b}\cdot\text{min}^{-1}$  and  $93.9 \pm 11.3 \text{ ml}\cdot\text{beat}^{-1}$ ) and during VOL in AB (to  $83.9 \pm 9.2 \text{ b}\cdot\text{min}^{-1}$  and  $89.7 \pm 9.0 \text{ ml}\cdot\text{beat}^{-1}$ ). At an equivalent submaximal  $\text{VO}_2$ , HR and SV were not different between the two groups, however Q was higher in PARA ( $6.6 \pm 0.7$  vs.  $4.1 \pm 0.9 \text{ l}\cdot\text{min}^{-1}$  per litre  $\Delta\text{VO}_2$ ). With increasing  $\text{VO}_2$ , BP rose in AB but not in PARA. These data confirm the importance of central command and skeletal muscle afferent feedback for the regulation of cardiovascular responses during leg exercise. In the absence of these two neural pathways (i.e. in PARA), non-neural mechanisms may be implicated in the control of HR and Q during FES-LCE. These results have important implications for FES-induced mobility whereby non-neural regulation of cardiovascular responses may impose physiological constraints upon functional outcomes.

**Keywords:** Electrical Stimulation, exercise, cardiovascular, spinal cord injury

## 1. Introduction

Functional electrical stimulation (FES) leg cycling presents an alternative mode of exercise to FES-induced gait or upper body voluntary activities for people with spinal cord injuries. Functional electrical stimulation-induced leg cycling (FES-LCE) incorporates a larger muscle mass than arm exercise alone and may also improve venous return and cardiac volume loading [1]. Consequently, during FES-LCE oxygen uptake ( $\text{VO}_2$ ), stroke volume (SV) and heart rate (HR) are increased above resting levels in people with paraplegia (PARA) [2].

FES-LCE presents an interesting paradigm for the regulation of typical cardiovascular responses during exercise. The involuntary nature of computer-controlled FES-LCE precludes the influence of 'central command' to the cardiovascular control centre(s). In addition, the ascending signals arising from ergoreceptors in the active leg muscles are unlikely to transcend the spinal cord lesion and their input to supraspinal regulation of cardiovascular events is abolished. Considering the importance of these neural mechanisms in the control of the cardiovascular responses during voluntary exercise, how are the cardiovascular responses during FES-LCE mediated?

The purpose of this study was to investigate the cardiovascular responses during FES-LCE in PARA and voluntary leg cycling (VOL) in able-bodied individuals (AB) at similar levels of oxygen uptake.

## 2. Methods

Six male PARA ( $38 \pm 7$  yr) with clinically complete spinal lesions between the T5-T9 and 6 male AB ( $38 \pm 6$  yr), volunteered to participate in this study. Informed consent was obtained from all subjects prior to their participation and the University of Sydney Human Ethics Committee had previously approved this study.

All PARA were trained using FES-induced leg muscle contractions, with the majority of subjects having habitually utilised FES for at least 2 months prior to testing. PARA performed FES-LCE trials with electrode

placements and neuromuscular stimulation parameters that we have previously described [9]. AB performed leg cycling on the same ergometer, but under voluntary control at 50 rev•min<sup>-1</sup>.

Physiological responses were determined for PARA at REST and during 4 to 6 trials of FES-LCE, at varying power outputs from 0 W to 12 W. The number of cycling trials performed by PARA was determined by the workloads at which the subject could cycle, unassisted, for the duration of the trial (~12 min). AB underwent assessments at REST and VOL at 6 W, 18 W, 30 W and 42 W. Due to the high levels of muscle fatigue associated with FES-LCE, the trials were performed over 2 test sessions with at least 2 days separating each session. Subsequently, all day 1 and day 2 data were pooled for the PARA and AB groups since there were no differences for any variable between days

During all trials, steady state VO<sub>2</sub> was measured via open circuit spirometry. The mean value for the final 2-min of collection during each 12-min trial was taken to represent the steady-state VO<sub>2</sub> for that power output. The heart rate (HR), beat-to-beat changes in systolic (SBP), diastolic (DBP) and mean arterial (MAP) pressures, stroke volume (SV) and cardiac output (Q) were assessed as described by Raymond and colleagues [9]. Arterial-venous oxygen difference (a-vO<sub>2</sub>diff) was calculated by dividing VO<sub>2</sub> by Q, and total peripheral resistance (TPR) was calculated as MAP/Q.

Linear regression analysis and t-tests were employed to explore relationships between the dependent variables and cycling time between the PARA and AB groups. All data are presented as mean ± SD and analyses were performed using a statistical software package (SPSS Version 8.0) on a personal computer. To protect against an inflated effect size following the pooling of data, the 99% confidence interval was used

### 3. Results

#### Oxygen uptake and cycling efficiency

At the highest cycling power output (PO), VO<sub>2</sub> was raised above resting levels to 0.75 ± 0.11 l•min<sup>-1</sup> in PARA and to 0.74 ± 0.07 l•min<sup>-1</sup> in AB. PARA achieved 9.2 ± 2.4 W PO<sub>peak</sub> during FES-LCE and AB completed 42.8 ± 1.0 W by VOL.

The linear relationship between PO and VO<sub>2</sub>, was VO<sub>2</sub> (l•min<sup>-1</sup>) = [0.022 x PO (W)] + 0.58 for PARA, and VO<sub>2</sub> (l•min<sup>-1</sup>) = [0.009 x PO (W)] + 0.33 for AB. The slope (β<sub>1</sub>) and intercept (β<sub>2</sub>) of the VO<sub>2</sub>-PO relationship were different from cipher in both groups. The β<sub>2</sub> of the VO<sub>2</sub>-PO relationship was significantly different between PARA and AB, however there was no difference in β<sub>1</sub>.

#### Cardiovascular and Haemodynamic Responses during Leg Cycling

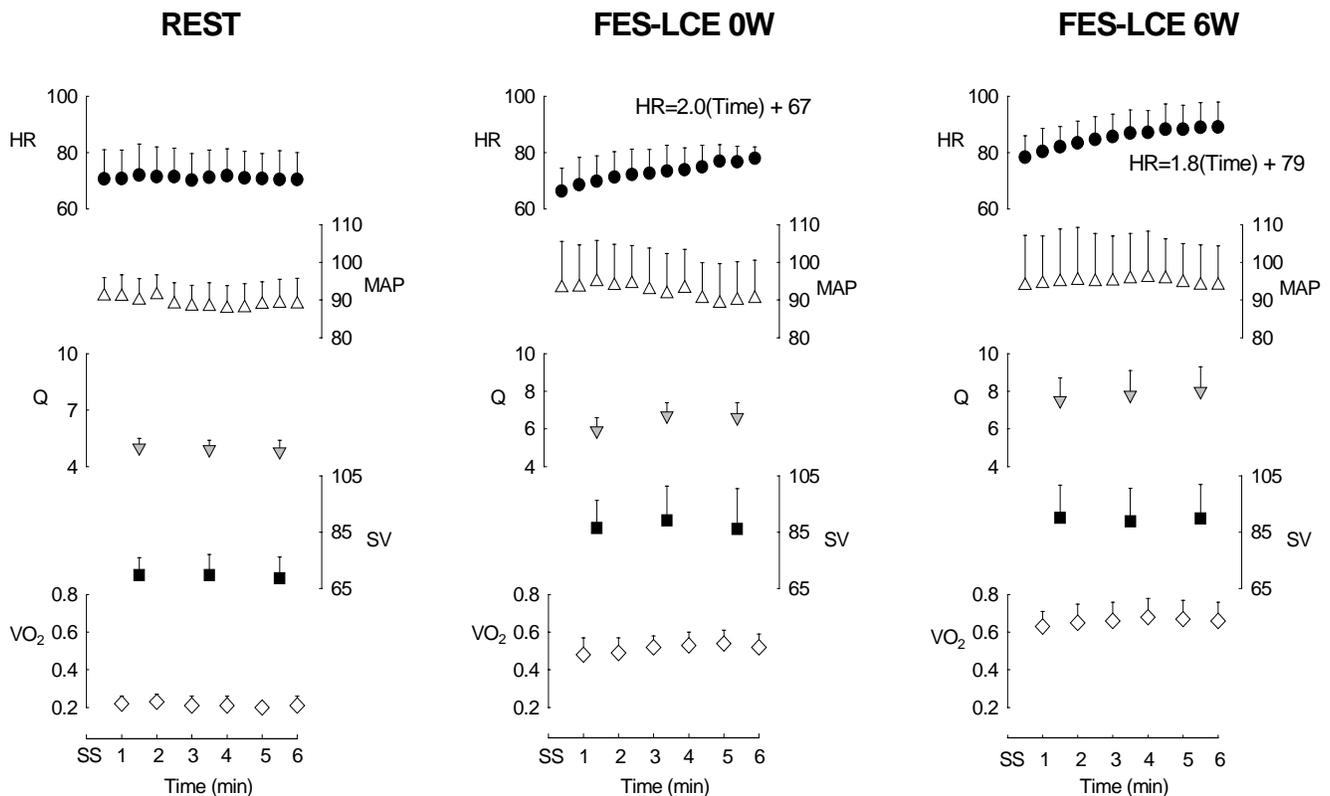
The PARA data for HR, SV, Q and MAP at REST, 0W FES-LCE and 6W FES-LCE are presented in Figure 1. HR and SV increased during FES-LCE in PARA (to 92.1 ± 8.6 b•min<sup>-1</sup> and 93.9 ± 11.3 ml•beat<sup>-1</sup>, respectively) and during VOL in AB (to 83.9 ± 9.2 b•min<sup>-1</sup> and 89.7 ± 9.0 ml•beat<sup>-1</sup>). At an equivalent submaximal VO<sub>2</sub>, HR and SV were not different between the two groups, however Q was higher in PARA (6.6 ± 0.7 vs. 4.1 ± 0.9 l•min per litre ΔVO<sub>2</sub>). With increasing VO<sub>2</sub>, MAP rose in AB but not in PARA

For AB, the dependent variables HR, SV, Q and MAP displayed significant linear trends in relation to VO<sub>2</sub>. In PARA, all variables except MAP displayed a significant linear trend. Comparison of the linear regressions between the two groups revealed that the slope (β<sub>1</sub>) of the Q-VO<sub>2</sub> and a-vO<sub>2</sub>diff-VO<sub>2</sub> relationships and the intercept (β<sub>2</sub>) of the SV-VO<sub>2</sub> regressions were significantly different.

Within each trial, linear regression analysis was also used to determine whether the HR after the subject had achieved metabolic steady state exhibited a linear trend for time (i.e. HR = β<sub>1</sub>•time + β<sub>2</sub>). A one-sample t-test comparing the slope of the HR-time relationship (i.e. β<sub>1</sub>) against cipher revealed that in PARA, β<sub>1</sub> was greater than cipher during each exercise trial (cf. Figure 1). For PARA, the β<sub>1</sub> derived from each exercise trial was different to that derived at REST. However, there were no differences between the β<sub>1</sub> parameters derived from 0W and 6W FES-LCE. When β<sub>1</sub> from all exercise trials were contrasted between PARA and AB, the mean HR-time slope was greater for PARA (Δ3.46 ± 1.1 vs. Δ0.18 ± 0.4 b•min<sup>-1</sup>). Analysis of MAP, SV, Q and VO<sub>2</sub> throughout the trial revealed no difference over time for either group, except for an increase of Q during ES-LCE at 0 W in PARA.

### 4. Discussion

The use of PARA with clinically complete lesions undertaking FES-LCE presented an interesting paradigm whereby central command and neural feedback from the active muscles were absent or attenuated during this form of leg exercise [13]. With this exercise paradigm, cardiovascular adjustments were observed in PARA that were not noted in AB performing VOL. These included a greater Q for a given level of VO<sub>2</sub> (6.6 ± 0.7 vs. 4.1 ± 0.9 l•min per litre ΔVO<sub>2</sub> for PARA and AB, respectively), no change in blood pressure and a gradual rise in HR (~Δ3.5 b•min<sup>-1</sup>) over the exercise trial observed in PARA but not AB. These findings suggest that in the absence of ascending and descending neural traffic, other mechanisms assist in the regulation of cardiovascular responses during FES-LCE. By implication, cardiovascular control during other FES-induced lower-limb activities (e.g. FES-gait) might also be different from able-bodied analogues of leg movement.



**Figure 1.** Time course of cardiorespiratory responses at REST and during 2 FES-LCE workloads in PARA. Data are presented following metabolic steady state (SS). Data are mean  $\pm$  SD. Each HR and MAP data point represents the average over 30 s. In the  $HR = \beta_1 \cdot \text{time} + \beta_2$  equations (top panels), time is represented in 30 s intervals.

### Blood Pressure

Consistent with the viewpoint that an increase in blood pressure is facilitated via neural feedback arising from the chemoreceptors within the active muscles [12], MAP was not elevated during FES-LCE in PARA (Figure 1), despite an increased metabolism. In contrast, there was a significant linear relationship between  $VO_2$  and MAP in AB performing VOL. The absence of any notable increase in mean arterial pressure during FES-LCE suggested that afferent signals arising from the active muscles were unable to reach the cardiovascular control centre(s) and thereby elicit any reflexive change in blood pressure [13].

### Cardiovascular Responses

During voluntary exercise, both central command and skeletal muscle afferent feedback are fundamental in the regulation of exercise-induced cardioacceleration [5,11]. In PARA undertaking FES-LCE, the diminution or absence of central command (because the leg movements were computer-controlled) and skeletal muscle afferent feedback (due to spinal cord lesion) suggested an alternative means of mediating exercise cardioacceleration. Kjær and colleagues [4] observed that the rise in HR

during FES-LCE in subjects with spinal cord injury was attenuated when such exercise was performed under conditions of leg vascular occlusion. This led the authors to propose a humoral mechanism responsible for cardioacceleration during FES-LCE. Our data support this viewpoint. During FES-LCE trials from 0W-9W, a gradual rise in HR was detected (mean =  $\sim\Delta 3.5 \text{ b} \cdot \text{min}^{-1}$ ), despite conclusive evidence that a metabolic “steady state” had been achieved. By contrast, HR remained constant during VOL performed by AB. This observation in PARA is consistent with the viewpoint of a putative humoral stimulus, which may either accumulate over the duration of exercise, or be released when the muscle reaches a certain metabolic ‘threshold’. One possible humoral factor may be circulating catecholamines, which are known to evoke a continual rise in heart rate during constant intensity exercise in patients with transplanted hearts [7].

SV was increased during both FES-LCE and VOL. This observation confirms earlier findings that have demonstrated reactivation of the lower limb muscle pump via the use of FES-induced contractions in individuals with spinal cord injury, improves venous return, ventricular filling pressures and stroke volume [2,3,13]. The augmented stroke volume contributed to an increase in Q

during FES-LCE and VOL. However, the change in Q for a given increase in  $\text{VO}_2$  was significantly greater in PARA than in AB ( $6.6 \pm 0.7$  vs.  $4.1 \pm 0.9$  l·min per litre  $\Delta\text{VO}_2$ , respectively), suggesting a 'hyperkinetic' circulatory response in PARA during FES leg exercise.

How might this higher than expected Q for a given  $\text{VO}_2$  during FES-LCE in PARA be explained? In AB, Q increases in response to the demand for increased blood flow by the exercising leg muscles. Several redundant neural reflexes that influence this circulatory response to exercise tightly regulate the supply-demand relationship. Current evidence strongly suggests that central command, the muscle chemo- and mechanoreflexes and the arterial baroreflex are all instrumental in the control of the cardiovascular responses during leg exercise [12]. For the unique exercise paradigm employed in the current study, the first three reflexes were presumably absent, whilst the arterial baroreflex remained operational. In AB, the baroreflex resets to a higher operating point during exercise [6,8] thus acting as one stimulus to elevate Q in order to maintain blood pressure and muscle perfusion pressure at higher levels. However in PARA, the carotid baroreflex, although functional, is not reset during FES-LCE [10]. Instead, the baroreflex acts to maintain blood pressure at a "set point" similar to that during resting conditions. Accordingly, it is unlikely that the baroreflex might then act to increase Q during FES-LCE unless it detected a fall in systemic arterial blood pressure — a reduction in blood pressure was not observed in this study.

Based on this observation, it is possible that the Q– $\text{VO}_2$  relationship observed during FES-LCE might not have been a function of the tight coupling which is a predominant feature of voluntary exercise. This speculation is supported by the observation that during FES-LCE at 0 W, there was a significant increase in Q throughout the trial. However, this increase was not accompanied by a concomitant change in  $\text{VO}_2$  during the trial once the subject had attained metabolic 'steady state', but appeared to track the slow rise in HR. This would suggest that the hyperkinetic relationship between  $\text{VO}_2$  and Q was not due to an increase in demand for blood flow at the muscle level, but due simply to the combined action of cardioacceleration and augmented venous return and stroke volume.

## 5. Conclusion

This study confirms the important role that central command and skeletal muscle afferent feedback has in the up-regulation of the cardiovascular responses to voluntary exercise. Using an exercise paradigm, which assumed these neural pathways were absent, the normal rise in blood pressure was eliminated and atypical cardiac output and heart rate adjustments during FES leg exercise were observed in PARA. Although it is not clear from this

study precisely what mechanism(s) regulated heart rate and cardiac output during FES-LCE, it is possible that some blood-borne stimulus mediated cardioacceleration. The implications of these findings are significant for individuals with spinal cord injury who may wish to undertake upright FES-induced gait.

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