

EXTERNAL POWER OUTPUT CHANGES DURING PROLONGED CYCLING WITH ELECTRICAL STIMULATION

D. Theisen,¹ C. Fornusek,² J. Raymond² and G. M. Davis²

From the ¹Department of Physical Education and Rehabilitation, Faculty of Medicine, Catholic University of Louvain, Louvain-la-Neuve, Belgium and ²Rehabilitation Research Centre, University of Sydney, Australia

This study analysed external power output and physiologic responses in 5 individuals with paraplegia during 40 minutes of electrical stimulation leg cycle exercise. Cycling was performed on a motor-driven isokinetic ergometer that enabled precise determinations of power output. Electrical stimulation was increased to 120–140 mA within the first 5 minutes and remained constant thereafter. Power output increased to 10.7 ± 3.0 W after 2 minutes, dropped to 5.3 ± 1.8 W after 6 minutes and subsequently recovered to 8.2 ± 2.2 and 6.1 ± 2.3 W after 19.5 and 40 minutes, respectively. Oxygen consumption increased to 0.47 ± 0.09 l/min after 6 minutes and declined during the second half of the exercise bout. Gross mechanical efficiency after 19.5 minutes was elevated compared with the value after 6 minutes. Heart rate was significantly increased at the end of the trial. The time-dependent variability of power output and physiological responses question the concept of steady state for this form of exercise.

Key words: electrical stimulation, leg cycle ergometry, external power output, oxygen consumption, stimulation amplitude, spinal cord injury, cardiovascular adjustments, exercise steady state.

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Correspondence address: Daniel Theisen, Department of Physical Education and Rehabilitation, Faculty of Medicine, Catholic University of Louvain, 1 place Pierre de Coubertin, BE-1348 Louvain-la-Neuve, Belgium. E-mail: daniel.theisen@edph.ucl.ac.be

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INTRODUCTION

Electrical stimulation-induced leg cycle exercise (ES-LCE) for individuals with spinal cord injury (SCI) has gained much popularity in recent years. This mode of exercise has been proposed as an alternative of, or complement to voluntary upper body exercise, in order to enhance training potential and exercise capacity (1–4). Chronic use of ES-LCE may elicit significant increases in peak oxygen uptake (VO_2) for this kind of exercise, increased lean body mass and decreased whole body fat content (5), as well as improvements in bone mineral density (6). To optimize ES-based intervention strategies for individuals with SCI, it is necessary to understand the basic physiologic responses to this form of exercise and to identify the factors that

determine performance. Despite an increasing number of investigations in this field, descriptive studies are sparse and the physiologic control mechanisms acting during ES-LCE, and thus influencing performance, remain poorly understood.

There is little information concerning external power output (PO) production during ES-LCE, which is probably related to methodological aspects. Many studies (2, 5, 7, 8) have used commercially available ES-LCE systems, in which cycling cadence is a system-controlled parameter. ES current output to the leg muscles is regulated by a microprocessor, which aims at maintaining a cycling rate of 50 revolution per minute (rpm). Exercise is automatically stopped if the cycling rate falls below 35 rpm. For obvious safety reasons, the current output delivered is limited to a maximum of approximately 140 mA. Therefore, cycling resistance must be set, or adjusted throughout the exercise bout, to a (low enough) level to enable cycling at 35–50 rpm (2, 5, 9). Thus external PO during ES-LCE is not a well-controlled variable, unlike during voluntary exercise, but may be largely influenced by the underlying physiologic responses.

The purpose of this study was to investigate external PO production during prolonged ES-LCE in individuals with SCI, using a constant stimulation amplitude and a constant pedalling rate. Physiologic responses were analysed concomitantly because of their potential influence on the physical performance during this specific form of exercise.

MATERIALS AND METHODS

Subjects

Five individuals (4 males and 1 female, with a mean age of 33 ± 8 years and a mean body mass of 66 ± 7 kg) participated in the study. The protocol was approved by the Human Ethics Committee of the University of Sydney. The participants had motor and sensory complete spinal cord injuries (ASIA class A) between the level thoracic 4 and thoracic 9, and mean time since injury was 5.5 ± 2.6 years. Prior to participation in the study all subjects underwent medical screening and planar radiography of the pelvis and the lower limbs to ensure that there were no contraindications to their participation in ES-LCE. The subjects were all familiar with ES-LCE and the testing procedures through prior sessions. Four of the 5 subjects had been training regularly 2–3 times per week, 2 for 2 months and 2 for more than 6 months prior to testing. One subject was formerly trained but had stopped training for several months prior to testing. The subjects took anti-spastic medication and showed no apparent side effects of their medication. None encountered significant episodes of spasticity during testing.

Methods

Electrical stimulation. Two gel-backed surface electrodes (Empi, USA)

were placed over the motor points of the quadriceps, hamstring and gluteal muscle groups. Muscle contractions were induced via a custom 6-channel transcutaneous neuromuscular stimulator (ExoStim2; Mr T. Turner, University of Sydney, Australia), delivering 250 μ s monophasic wave pulses at a frequency of 35 Hz. Stimulation amplitude was limited to a maximum of 120–140 mA. The computer-controlled temporal pattern of stimulation of the different muscle groups was synchronized with respect to the crank position of the cycle ergometer (0° = the top dead centre). For the right hand side, the stimulation angles were $300\text{--}30^\circ$ for the quadriceps, $60\text{--}160^\circ$ for the hamstring and $6\text{--}72^\circ$ for the gluteal muscle groups.

Leg cycle ergometer. A customized MOTOMed Viva cycle ergometer (Reck, Germany) was used. This motor-driven isokinetic ergometer evaluated external PO from the negative motor current resulting from the torque exerted by the user on the crank. The device had been calibrated dynamically with weights of known masses. Custom software was used to record the motor current data (~ 60 Hz) and the crank position on a PC laptop, to process and display relevant parameters during the test and to control the neuromuscular stimulator via the parallel port.

Protocol. After the electrode placement, the subjects transferred on to the semi-recumbent leg cycle ergometer and rested for 10 minutes. Exercise started with the motor turning the crank at a constant frequency of 50 revolutions per minute. The first 5–10 crank revolutions were performed with no stimulation of the lower limb muscles to determine and take into account the influence of the leg weight on PO calculations. Subsequently, ES of the lower limb muscles was triggered. Stimulation amplitude was increased within the first minutes of exercise to a maximum of 120–140 mA, a value kept constant thereafter. For safety reasons, the initial rise of stimulation amplitude was progressive and aimed at inducing a preset external target PO, determined individually during previous sessions.

Physiologic measurements. Metabolic and respiratory parameters were measured using a CPX/D[®] MedGraphics metabolic cart (Medical Graphics Corporation, Germany). Subjects breathed through a mouthpiece connected to a pneumotach and an air sample line. Expired air was analysed on a breath-by-breath basis. Values for VO_2 (STPD) carbon dioxide production (VCO_2 , STPD) and expired ventilation (VE, BTPS) were obtained and averaged over 30 seconds periods at rest and throughout exercise. Heart rate was continuously monitored using a Portascope CR55 ECG recorder (Cardiac Recorders, UK). Heart rate values were averaged over the same sampling periods used for the metabolic data. Gross mechanical efficiency was evaluated from aerobic energy sources, assuming an energetic equivalent of 21.1 kJ per litre O_2 .

Statistics

Time-related changes of the different parameters were evaluated using one-way repeated measures analyses of variance and the Student-Newman-Keuls post-hoc procedure when appropriate. The time-points used were identified from the PO curve and were based on the occurrence of extreme values recorded, i.e. at 0 (rest), 2, 6, 19.5 and 40 minutes of exercise. Significant effects were considered at the 5% level. Data are presented as means \pm standard deviation.

RESULTS

As illustrated in Fig. 1, stimulation amplitude was ramped up during the first 5 minutes of ES-LCE and kept constant thereafter. PO demonstrated a strong time-related dependency ($F = 29.141$, $p < 0.001$) and showed significant changes, even during the phase of constant stimulation amplitude (Fig. 1). After 2 minutes of exercise, during the ramping up phase of the stimulation current, PO reached a maximal value, followed by a sharp drop ($p < 0.05$) after 6 minutes of exercise (Table I). Subsequently, PO rose again progressively after 19.5 minutes

($p < 0.05$) and decreased slightly towards the end of exercise (not significantly).

VO_2 showed successive, significant increases from the rest value after 2 and 6 minutes of ES-LCE (Table I). Although VO_2 remained constant thereafter (the values at 19.5 and 40 minutes not being significantly different from the one at 6 minutes), the VO_2 at 40 minutes of exercise was significantly lower than the one at 19.5 minutes. Gross mechanical efficiency after 6 minutes of ES-LCE was $3.3 \pm 1.1\%$ and demonstrated a significant increase to $4.7 \pm 1.2\%$ after 19.5 minutes. The value found at the end of the exercise bout ($4.2 \pm 1.5\%$), however, was not significantly different to the mechanical efficiency at 6 minutes of exercise (Fig. 2).

Respiratory exchange ratio increased during the initial phase of the trial to values > 1 and decreased progressively thereafter (Fig. 3 upper panel). Heart rate showed a slight drop after 2 minutes of exercise, followed by a subsequent rise to a significantly elevated end value as compared to rest ($p < 0.05$) (Fig. 3 lower panel). Oxygen pulse increased from rest until 6

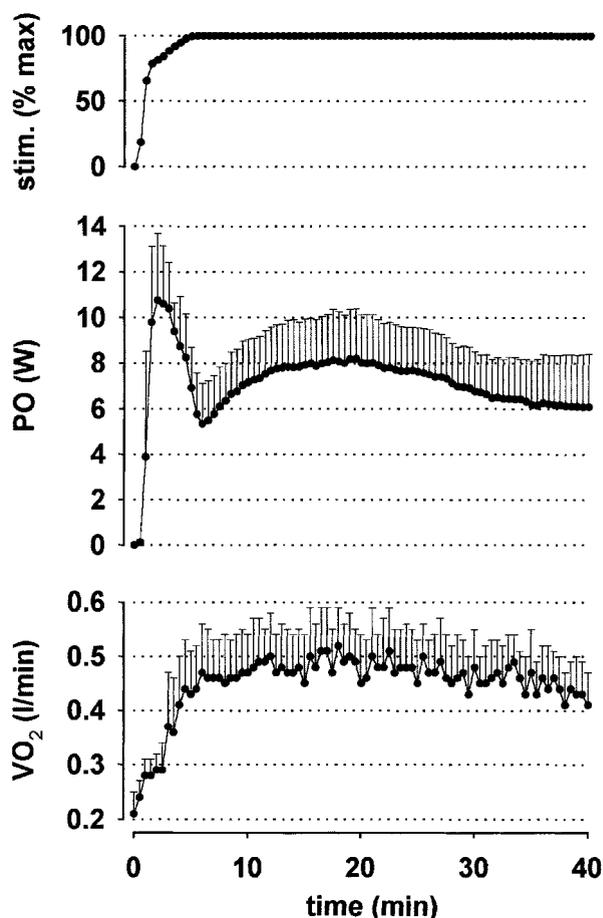


Fig. 1. Stimulation amplitude (stim.), external power output (PO) and oxygen consumption (VO_2) during 40 minutes electrical stimulation leg cycle exercise in individuals with paraplegia (mean values and standard deviations given).

Table I. Main parameters recorded at time-points of extreme power output during 40 minutes electrical stimulation leg cycle exercise in individuals with paraplegia

	0 minutes (rest)	2 minutes	6 minutes	19.5 minutes	40 minutes
PO (W)	0	10.7 ± 3.0*	5.3 ± 1.8*	8.2 ± 2.2*	6.1 ± 2.3
VO ₂ (l/min)	0.21 ± 0.04	0.29 ± 0.03*	0.47 ± 0.09*	0.49 ± 0.06	0.41 ± 0.06*
HR (beats/min)	76 ± 16	70 ± 17	80 ± 14	90 ± 12	93 ± 17
O ₂ pulse (ml/beat)	2.8 ± 0.7	4.3 ± 1.0*	6.0 ± 1.3*	5.5 ± 0.8	4.5 ± 0.5*

Data are means ± standard deviation; PO = power output; VO₂ = oxygen uptake; HR = heart rate; O₂ pulse = oxygen pulse; * significantly different from preceding time-point value.

minutes of exercise ($p < 0.05$) and decreased during the second half of the exercise bout ($p < 0.05$) (Table I).

DISCUSSION

Exercise paradigm

The main finding of this investigation is that external PO production during prolonged ES-LCE is highly variable, despite constant ES amplitude and VO₂. PO during ES-LCE could be largely determined by the particular physiological conditions associated with this form of exercise, which differs from voluntary exercise in many aspects. The cardiovascular adjustments to voluntary exercise are tightly regulated by the autonomic nervous system, which generates a well-matched response to the work intensity through a decrease in parasympathetic, and an increase in sympathetic activity. This neural activity is elicited via descending feed-forward control, or "central command", as well as ascending feedback control from the working muscles, termed the "exercise pressor reflex" (10). Additional feedback control is provided by the baroreflex, which is reset to a higher operating pressure during dynamic exercise (11). These mechanisms work in concert to generate a rapid,

coordinated cardiovascular response by which oxygen supply to the exercising muscles is enhanced. Consequently, skeletal muscle blood flow and VO₂ increase linearly with work intensity, such that pulmonary VO₂ is linearly related to absolute mechanical PO delivered (12).

Compared with voluntary LCE, ES-LCE in individuals with complete SCI represents a very particular exercise paradigm. First, movements are performed via surface stimulation of the paralysed lower limb muscles, i.e. in the absence of intended effort. Second, as a result of the spinal cord disruption, there is no direct neural interference from the central command and the exercise pressor reflex to induce a rapid and well-matched

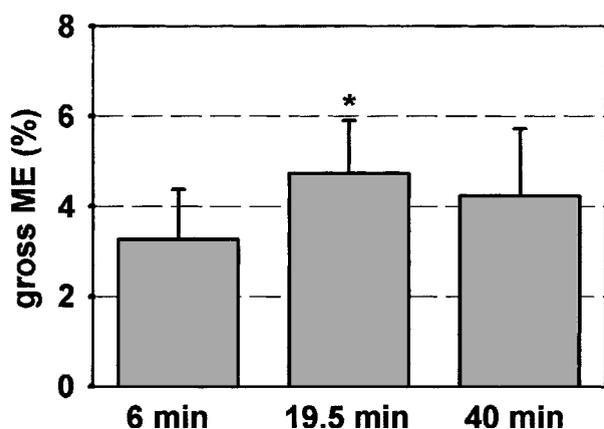


Fig. 2. Gross mechanical efficiency (ME) after 6, 19.5 and 40 minutes of continuous, constant current electrical stimulation induced leg cycling in individuals with paraplegia. * Significantly different from value recorded after 6 minutes (mean values and standard deviations given).

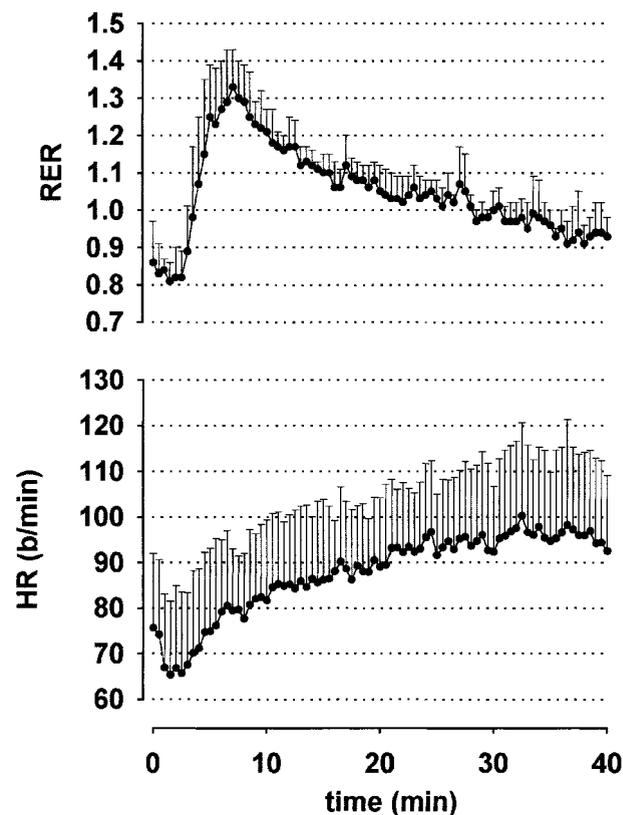


Fig. 3. Respiratory exchange ratio (RER) and heart rate (HR) during 40 minutes electrical stimulation leg cycle exercise in individuals with paraplegia (mean values and standard deviations given).

response to the work intensity. One consequence of this exercise paradigm is that there is no stimulus for carotid baroreceptor resetting during ES-LCE, and the reflex thus operates near resting levels of arterial blood pressure (7). Given this situation, physiologic adjustments probably function through alternative, non-neural pathways, which will influence cycling performance and thus external PO production.

External PO

Time-dependent changes in PO during ES-LCE have been reported previously (9), but not quantitatively analysed. The peak in PO produced during the initial phase of exercise was achieved with a high anaerobic contribution, since VO_2 was not considerably elevated with respect to rest (although significantly so). The levelling-off and the subsequent drop in PO occurred as stimulation amplitude approached maximum and likely no further muscle fibres were recruited. This phase could reflect the depletion of intra-muscular phosphocreatine stores, as well as a gradual increase in intra-muscular lactate accumulation, leading to a decrease in contraction performance after 6 minutes of exercise (13). Although no lactate measurements were performed in this study, a large anaerobic contribution was suggested by the high respiratory exchange ratio, thus confirming previous reports (2, 5, 8). This could be attributed to the de-conditioned state of the atrophied lower limb muscles in individuals with SCI, including increased proportions of type IIb fibres (9), low capillary density (14), and the preferential recruitment of fast glycolytic muscle fibres during ES (2). Taken together, these factors may explain the initial decrease of external PO. An additional explanation could be a possible antagonistic muscle activation through increased stimulation amplitude during the initial exercise phase. This may have decreased cycling efficiency and thus PO, while contributing to the rise in VO_2 .

The reasons for the recovery in cycling ability after 20 minutes are more speculative and could be related to the vascular adjustments during prolonged ES-LCE. In the absence of a neurally mediated exercise pressor reflex, a slowly developing, locally induced vasodilatation in active muscle would enhance the clearance of accumulated, fatigue-inducing metabolites in the stimulated leg muscles. Thus, contraction performance could be improved, allowing a recovery in cycling ability as shown by the increases in PO and gross mechanical efficiency after 19.5 minutes of ES-LCE. However, these assumptions cannot be supported by the mere results here, and further studies are warranted to identify the mechanisms involved.

Metabolic and cardiovascular responses

The initial phase of ES-LCE was characterized by a moderate but significant rise in VO_2 after 2 minutes (Table I). At this stage heart rate had declined slightly while oxygen pulse was enhanced. This suggests that the quick response in VO_2 during the first 2 minutes of exercise was achieved by an increase in cardiac stroke volume and/or oxygen extraction (arteriovenous O_2 difference). After a subsequent increase at 6 minutes of

exercise, VO_2 remained relatively stable, with a decline during the second half of the session. At the same time oxygen pulse decreased, probably as a result of a drop in cardiac stroke volume if one assumes that oxygen extraction remained unchanged during this period of constant stimulation amplitude. This would be consistent with a peripheral vasodilatation, which may be amplified as exercise continues, due to the inability to generate vasoconstrictive sympathetic control over the working muscles (12).

An important finding of this study was the apparent lack of coupling between PO and VO_2 (Fig. 1). This contrasts with previous reports suggesting a close relationship between mechanical PO and VO_2 during ES-LCE (1, 5), like during voluntary exercise (12). However, those studies (1, 5) used graded, intermittent workloads, which may have limited muscle fatigue, thus overcoming decreases in cycling performance and preventing a dissociation between PO and VO_2 . Furthermore, they (1, 5) used commercial leg cycle ergometers in which cycling cadence was maintained by automatic adjustments of stimulation amplitude, a variable thus not controlled by the experimenter. As long as stimulation amplitude was sub-maximal (<140 mA in general), a given PO could be maintained by increases in the current output, despite muscle fatigue occurring.

Considering the absence of direct neural influences from the central command and the exercise pressor reflex, it is not clear what factors are responsible for the heart rate response. The opposite variations in heart rate and VO_2 at the beginning of exercise suggest that the two parameters are not tightly coupled during this phase. The slight decrease in heart rate after 2 minutes compares with previous observations (5) and may have been triggered by an elevation in blood pressure via the intact baroreflex, which operates to maintain blood pressure near its resting level (7). The gradual rise in heart rate with the continuation of exercise may be stimulated by non-neural blood-borne factors that accumulate over time (15). Another explanation may be a progressive peripheral vasodilatation caused by an increase in body temperature (5). Thus, the elevation in heart rate could be due to a thermoregulatory-induced cardiovascular drift, a hypothesis that would be in line with a decreased stroke volume and VO_2 towards the end of exercise.

Implications of the study

The time-dependent variability of external PO production and physiologic variables observed during prolonged ES-LCE raises the problem of defining the steady state conditions of this form of exercise. It is necessary to evaluate which of the parameters like stimulation amplitude, cycling cadence, PO, VO_2 , heart rate, etc. are to be considered in this respect. It is even possible that the concept of steady state does not apply to ES-LCE. Neural feed-forward and peripheral feedback controls are interrupted in individuals with complete SCI, which directly influences physiological adjustments to exercise and performance capacity (external PO production). Physiologic adjustments (e.g. increase in heart rate, vasodilatation in active muscles)

are achieved via other, non-neural mechanisms (e.g. humoral pathways, local factors), and the occurrence of a steady state would theoretically require a longer exercise time, often compromised by the occurrence of early muscle fatigue. More studies are warranted to identify the physiologic mechanisms underlying short-term and prolonged ES-LCE in individuals with SCI.

In terms of maximizing the training effects of ES-LCE, current practices have individuals to exercise as hard and as long as possible during a single session (5, 8, 9). These conditions are associated with a high contribution of anaerobic energy components (2, 5, 8) and may indeed be necessary to provide an appropriate training stimulus, e.g. with respect to cardiac workload. Such an approach might however not be optimal to augment muscle endurance or to make it possible to maintain a given PO for a certain duration, which may have implications for ES-LCE training regimens or the safety of ES-assisted ambulation. Future longitudinal investigations should be directed towards the effects of different training modalities and thus contribute to a better understanding of the physiologic adjustments during chronic ES-induced exercise.

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