

Effect of adjusting pulse durations of functional electrical stimulation cycling on energy expenditure and fatigue after spinal cord injury

Ashraf S. Gorgey, MPT, PhD, FACSM;^{1–2*} Hunter J. Poarch, BS;¹ David R. Dolbow, DPT, PhD;¹ Teodoro Castillo, MD;¹ David R. Gater, MD, PhD³

¹Spinal Cord Injury and Disorders Service, Hunter Holmes McGuire Department of Veterans Affairs Medical Center, Richmond, VA; ²Department of Physical Medicine and Rehabilitation, Virginia Commonwealth University, Richmond, VA; ³Department of Physical Medicine and Rehabilitation, Penn State Milton S. Hershey Medical Center, Hershey, PA

Abstract—The purpose of the current study was to determine the effects of three different pulse durations (200, 350, and 500 microseconds [P200, P350, and P500, respectively]) on oxygen uptake, cycling performance, and energy expenditure (EE) percentage of fatigue of the knee extensor muscle group immediately and 48 to 72 h after cycling in persons with spinal cord injury (SCI). A convenience sample of 10 individuals with motor complete SCI participated in a repeated-measures design using a functional electrical stimulation (FES) cycle ergometer over a 3 wk period. There was no difference among the three FES protocols on relative oxygen uptake or cycling EE. Delta EE between exercise and rest was 42% greater in both P500 and P350 than in P200 ($p = 0.07$), whereas recovery oxygen uptake was 23% greater in P350 than in P200 ($p = 0.03$). There was no difference in the outcomes of the three pulse durations on muscle fatigue. Knee extensor torque significantly decreased immediately after ($p < 0.001$) and 48 to 72 h after ($p < 0.001$) FES leg cycling. Lengthening pulse duration did not affect submaximal or relative oxygen uptake or EE, total EE, and time to fatigue. Greater recovery oxygen uptake and delta EE were noted in P350 and P500 compared with P200. An acute bout of FES leg cycling resulted in torque reduction that did not fully recover 48 to 72 h after cycling.

Key words: cycling performance, energy expenditure, exercise, fatigue, functional electrical stimulation, oxygen uptake, pulse duration, rehabilitation, spinal cord injury, torque.

INTRODUCTION

Functional electrical stimulation (FES) exercise is an essential rehabilitation component to attenuate several of the chronic conditions following spinal cord injury (SCI) [1–3]. Different forms of FES protocols have proven to be effective in reversing the process of skeletal muscle atrophy and the percentage of leg lean mass [1,4–6], as well as attenuating the decline for bone mineral density and bone architecture and restoring activities similar to standing and walking [5]. In persons with SCI, applications

Abbreviations: AIS = American Spinal Injury Association Impairment Scale, ANOVA = analysis of variance, BP = blood pressure, C = cervical, CSA = cross-sectional area, EE = energy expenditure, FES = functional electrical stimulation, HR = heart rate, LCE = leg cycle ergometry, P200 = pulse duration of 200 μ s, P350 = pulse duration of 350 μ s, P500 = pulse duration of 500 μ s, RPM = rotations per minute, SCI = spinal cord injury, T = thoracic, VA = Department of Veterans Affairs, VCO_2 = carbon dioxide production, VO_2 = oxygen uptake.

*Address all correspondence to Ashraf S. Gorgey, MPT, PhD, FACSM; Spinal Cord Injury and Disorders Service, Hunter Holmes McGuire VA Medical Center, 1201 Broad Rock Blvd, Richmond, VA 23249; 804-675-5000, ext 3386; fax: 804-675-5223. Email: ashraf.gorgey@va.gov
<http://dx.doi.org/10.1682/JRRD.2014.02.0054>

have extended to include reduction in ectopic adipose tissue and improvement in glucose tolerance, insulin sensitivity, and lipid profile [4]. Additionally, FES cycling is commonly used as a rehabilitation intervention to enhance venous return and cardiovascular capacity in persons with SCI. Optimizing stimulation parameters that are commonly used is essential to maximize the benefits during FES cycling [7–10].

An inherent problem to surface neuromuscular electrical stimulation is the rapid onset of muscle fatigue [7–11]. This well-studied phenomenon is known to interfere with optimizing neuromuscular and cardiovascular performance in persons with SCI. Rapid onset muscle fatigue is characterized by rapid decline in performance during repetitive activity [8–11]. Previous work documented that peak torque was reduced in the group with SCI 1.7 times more than in controls after isometric activations with electrical stimulation and remained declined by 22 percent on day 3 in the group with SCI [11]. Mahoney et al. examined the magnitude and recovery of low-frequency fatigue in the quadriceps muscle after electrically stimulated contractions in people with SCI compared with controls [12]. The controls showed a gradual recovery of torque, whereas the group with SCI did not show recovery 24 h after stimulation [12]. This phenomenon has yet to be studied or established during FES cycling.

We and others have previously demonstrated that altering electrical stimulation parameters can influence motor unit recruitments and reduce neuromuscular fatigue [10,13–16]. Increasing the amplitude of the current and pulse duration has been shown to increase skeletal muscle recruitment but does not interfere with the extent of fatigue during stimulation [10]. Increasing pulse duration was previously shown to increase torque production during isometric action of the knee extensor. A pulse duration of 450 μ s increased torque by 55 percent and the amount of muscle activated by 40 percent compared with 150 μ s [13–14]. Notably, increasing pulse duration from 150 to 450 μ s did not appear to influence percentage of fatigue [10]. This increase in pulse duration from 150 to 500 μ s was previously shown to increase the torque in a nonlinear fashion [17], which may be due to nonlinear summation of motor units [18]. We have clearly documented that longer pulse duration, but not stimulation duration, resulted in a greater evoked and normalized torque compared with the shorter pulse duration, even after controlling for the activated muscular cross-sectional areas (CSAs) [14]. Others have clearly

documented that increasing pulse duration to 600 μ s results in increasing the evoked torque of the knee extensor [9,15–16]. It is still unclear whether skeletal muscle atrophy and infiltration of intramuscular fat interfere with the capacity of adjusting pulse durations to increase the evoked torque following SCI [19].

Despite the aforementioned evidence, there is still a scarcity of knowledge about the translation of this evidence to persons with SCI or to applications of FES cycling. Several studies have also reported applications of short pulse durations less than 350 μ s during FES cycling [2,6]; conversely, others have utilized pulse durations greater than 350 μ s during training of individuals with SCI [1,3,6,20–22]. The scientific basis on which these pulse durations were selected during FES cycling is unclear. Adjusting the stimulation parameters during FES cycling to optimize power output to subsequently improve card-metabolic benefits has been previously investigated [23]. Janssen and Pringle increased the current stimulation from 140 to 300 mA, widened the FES firing angles by 55°, stimulated the shank muscles, and increased the maximal resistance from 8.6 to 19.6 N [23]. It is possible that increasing pulse duration can enhance FES cycling performance and increase power output without evoking additional muscle fatigue [10]. This potentially could increase neuromuscular recruitment, evoking more torque and increased energy expenditure (EE) during cycling. Increasing EE may assist in the fight against obesity and other noncommunicable diseases after SCI [1,24].

The major purpose of the current work was to investigate the effects of administering different pulse durations (200, 350, and 500 μ s [P200, P350, and P500, respectively]) on EE and muscle fatigue during FES cycling in person with SCI. Our hypothesis was that increasing pulse duration length may enhance cycling performance without resulting in additional muscle fatigue during FES after SCI. The significance of this work is to provide a clinical basis for how to appropriately adjust and manipulate FES parameters to enhance performance and EE and minimize fatigue.

METHODS

Study Design

Participants were recruited from the inpatient and outpatient population of the Hunter Holmes McGuire Department of Veterans Affairs (VA) Medical Center (Richmond,

Virginia) via therapists, word of mouth, and flyers. All potential participants received a detailed explanation of the study interventions and procedures. After meeting the inclusion criteria, all participants were asked to read and sign a consent form that was approved by the local ethics committee. All study procedures were conducted according to the Declaration of Helsinki.

All participants reported to the SCI Exercise Laboratory (Hunter Holmes McGuire VA Medical Center) for a screening and familiarization session to orient them to the actual procedures and equipment. Three study visits were performed on the RT300 FES bike (Restorative Therapies Inc; Baltimore, Maryland). Each study visit lasted about 2 to 3 h. **Figure 1** presents the general design of the study.

Subjects

Ten individuals with motor complete SCI (cervical [C]5–thoracic [T]10, American Spinal Injury Association Impairment Scale [AIS] A and B) who were greater than 1 yr postinjury were recruited to ensure stability in body composition and no further transformation in fiber types from slow to fast twitch skeletal muscle fibers [25]. Fast twitch fibers are highly fatigable and the response of adjusting FES parameters may be different than in persons with acute SCI. For female participants, a pregnancy test was performed as part of routine standard of care.

Inclusion and Exclusion Criteria

Participants were included if they had motor complete SCI (C5–T10, AIS A and B), were more than 1 yr

postinjury to ensure a homogenous sample, were between 18 and 65 yr old, and were responsive to electrical stimulation. Participants with preexisting medical conditions (uncontrolled hypertension, uncontrolled hyperglycemia or a hemoglobin A₁C level greater than 7.0, chronic arterial diseases, recent deep vein thrombosis, uncontrolled autonomic dysreflexia, severe spasticity, fractures or history of fractures, pressure ulcers greater than grade II, documented osteoporosis, uncompensated hypothyroidism, renal disease, or pregnancy) were excluded from the study. Additionally, those who were unresponsive to surface electrical stimulation or who were involved in any training program, including lower-limb FES bike or surface electrical stimulation training, were not considered eligible to participate in the study. None of our participants were trained or had previous experience utilizing the FES bike.

Intervention

First Visit (Screening Visit)

Participants were medically screened to determine eligibility and safety to participate. A medical history including body weight and height were measured as part of the medical screening. A 12-lead electrocardiogram was performed and reviewed by a physician. Two adhesive electrodes were then placed on the right knee extensor muscle group and a portable electrical stimulation unit was used to determine the response of the knee extensors. The current was gradually increased until tetanic muscle contraction was visualized by the researcher.

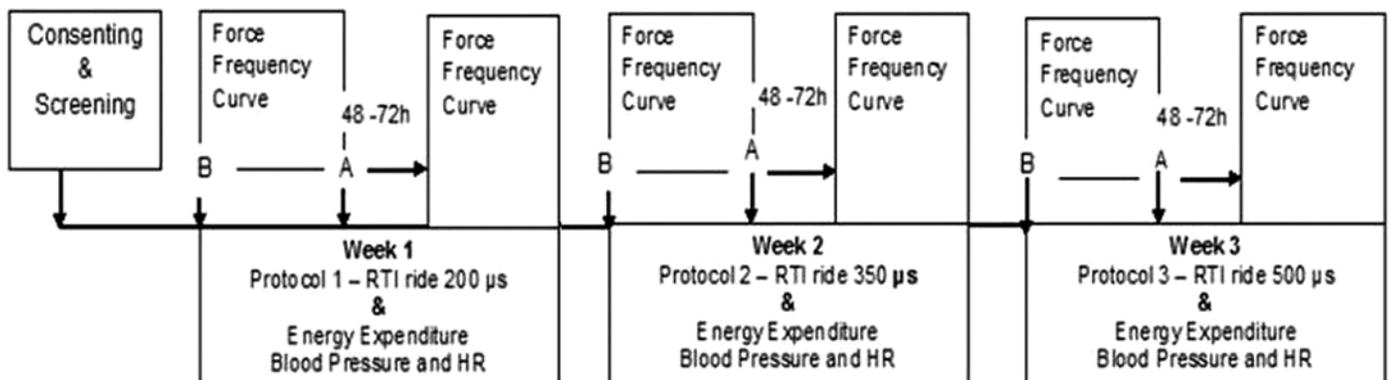


Figure 1.

Schematic diagram illustrates study design. Three functional electrical stimulation (FES) protocols were randomly assigned, and force frequency curves were performed before (B) and after (A) FES rides. HR = heart rate, RTI = Restorative Technologies Inc (FES bike).

Visits 2, 3, and 4 (Spaced 1 Week Apart)

Following the screening visit, all participants were invited to visit the SCI Exercise Laboratory three times separated by 1 wk. A 1 wk interval was provided to minimize possible occurrence of low-frequency fatigue and the accumulated effects of fatigue from the first bout of exercise. The order of FES cycling protocols was assigned in a random fashion to all participants. Simple randomization was performed by blindly selecting a folded paper with the protocol number (P200, P350, or P500) out of a bag. The amplitude of the current remained constant across the three riding protocols. The maximum amplitude of the current was set at 140, 140, and 100 mA for knee extensor, knee flexor, and gluteus maximus muscle groups, respectively [21–22]. Resting, exercise, and recovery heart rate (HR), blood pressure (BP), and EE were monitored across the three visits [21].

The three rides were performed with each subject sitting in his or her own wheelchair (power or manual). Conductive adhesive gel electrodes were used to evoke skeletal muscle stimulation [4,10,21]. Before removing the electrodes, the location of electrodes were marked to ensure exact positioning on subsequent trials. Subjects were given a marker to mark the location on a daily basis or to ask a member of their family to do so. After securing both legs to the bike, knee flexion angles were measured before starting to ensure that each participant attained a similar position across the three FES cycling rides. Rectangular adhesive electrodes were placed on the skin of the following corresponding skeletal muscle groups:

1. Knee extensor (7.5×10 cm): One electrode was placed on the skin 2 to 3 cm above the superior aspect of the patella over the vastus medialis muscle, and the other electrode was placed lateral to and 30 cm above the patella over the vastus lateralis muscle.
2. Hamstrings (7.5×10 cm): One electrode was placed 2 to 3 cm above the popliteal fossa and the other electrode was placed 20 cm above the popliteal fossa.
3. Gluteus maximus (5×9 cm): Two electrodes were placed parallel and on the bulk of muscle belly with two fingers-width separation between the electrodes.

In the three protocols, the servo-motor of the bike was permitted to assist during the warm-up period and initial phase of cycling for 5 min, then the motor was automatically discontinued during the actual cycling phase. We allowed the motor to function during the warm-up period to reduce spasms and the extensor spasticity that commonly interferes with the cycling pattern.

The bike is designed to stop when spasms are detected during cycling pattern as a method to protect the patient. The cycle motor was deactivated during the actual testing to examine the capacity of the stimulated muscles for maintaining cycling performance before fatigue. Time to maximum (100%) current amplitude was calculated to determine whether adjusting the pulse duration could delay maximum stimulation of the activated muscle groups. Fatiguing threshold was set at 18 rotations per minute (RPM) and once the RPM fell below this threshold, the bike automatically shifted from active to passive cycling (cool down). During the 3 min cool-down period, the patients passively cycled with no electrical stimulation. The cool-down period was then followed by 5 min of recovery, during which the participant was still connected to the bike but in a complete resting position. Participants were allowed to exercise until fatigue, and the time of active FES-leg cycle ergometry (LCE) until fatigue was calculated for each participant.

Protocol 1 (P200). Frequency was set to 33.3 Hz, resistance to 1 Nm, speed to 40 to 45 RPM, and pulse duration to 200 μ s.

Protocol 2 (P350). Frequency was set to 33.3 Hz, resistance to 1 Nm, speed to 40 to 45 RPM, and pulse duration to 350 μ s.

Protocol 3 (P500). Frequency was set to 33.3 Hz, resistance to 1 Nm, speed to 40 to 45 RPM, and pulse duration to 500 μ s.

Fatigue Test Using Force-Frequency Curve

The fatigue tests were done before, immediately after, and 48 to 72 h after each cycling session by establishing force-frequency curves [15–16]. A lift (ArjoHuntleigh; Malmö, Sweden) mounted in the ceiling was available to transfer subjects onto the isokinetic dynamometer chair (Biodex Medical Systems Inc; Shirley, New York) in order to measure torque generated by the knee extensor muscle group. Two adhesive electrodes were placed on one leg. A small amount of electrical current was generated to see whether the muscle group in that leg would contract to measure the force of muscle contraction. The duration of the test was 30 to 45 min. Participants were asked to return to the SCI Exercise Laboratory 48 to 72 h after visits 2, 3, and 4 for one isokinetic dynamometer chair test to re-measure low-frequency fatigue. Persistence of decline in torque longer than 48 h was defined as low-frequency fatigue.

Fatigue was assessed by measuring the torque elicited at 10, 20, 30, 40, 50, 60, 80, and 100 Hz before and immediately after cycling. The frequency order was randomly assigned to each participant [15–16]. Subjects were seated with the hip flexed at 90° and the knee flexed at 90° (where 0 corresponds with full knee extension). Each subject was securely strapped to the isokinetic dynamometer chair by two-crossover shoulder harnesses and a belt across the hip joint. The axis of rotation of the dynamometer was aligned to the anatomical knee axis. The lever arm was placed 2 to 3 in. above the lateral malleolus. Two electrodes were placed on the skin 2 to 3 cm above the superior aspect of the patella over the vastus medialis muscle and 30 cm above the patella over the vastus lateralis muscle. A battery-operated electrical stimulation unit (Empi; Vista, California) was used to establish the force-frequency curves. The current was gradually increased until evoking peak torque (i.e., maximal force generated by knee extensor muscle group). After determining the peak torque, the current (milliamperes) that could elicit 50 percent of the peak torque was recorded for all subjects, and this was referred to as mA_{50%}. The mA_{50%} was used to establish the force-frequency curve prior to, immediately after, and 48 to 72 h after cycling for the three pulse duration protocols. Pulse duration was set at 400 μs with a contraction time of 3 s. All frequencies were delivered in duplicate with 10 to 15 s intervals between two contractions and a 1 min interval between two consecutive frequencies. Force-frequency curves were constructed from the pre- and postcycling values. Percentage of fatigue was determined using **Equation 1** [10,14]—

% Fatigue 48–72 h or Immediately Postcycling =

$$\frac{\text{Force Frequency Curve Postcycling} - \text{Force Frequency Precycling}}{\text{Force Frequency Precycling}} \quad (1)$$

It should be noted that the first frequency assigned to the patient was then repeated at the end of the series of frequencies to account for the potentiation effects (i.e., abrupt increase in the evoked torque). For example, if 40 Hz was the first frequency to be delivered, then 40 Hz on torque was repeated again after delivering the remaining frequencies to account for the effects of potentiation on the tested muscle. The frequency order was randomized to the knee extensor muscle group. The order of randomization varied among the 10 participants.

Energy Expenditure

During each FES cycling protocol, EE was monitored using a K4 b2 portable metabolic unit (COSMED; Rome, Italy) portable metabolic unit [19]. After calibration, subjects were asked to place the mask on their face in order to monitor oxygen uptake (VO₂) and carbon dioxide production (VCO₂). A 3 min resting period allowed the subject to get used to breathing with the mask on while he or she was attached to the FES bike. VO₂ and VCO₂ were monitored throughout the exercise in order to determine total EE. After the resting period, EE was measured during the 3 min warm-up period, exercise period (until fatigue), and cool-down period. Additionally, the first 5 min of recovery was recorded to determine the efficacy of each protocol on EE and substrate utilization. Delta EE for each of the three FES protocols was calculated as the difference in EE between the exercise period and the resting period.

HR (via HR monitor [Polar Electro Inc; Lake Success, New York]) and BP (via 740 vital signs monitor [CAS Medical Systems Inc; Branford, Connecticut]) were measured during the resting, exercise, and recovery periods. The HR monitor was placed on the subject's chest below the xyphoid process and remained throughout the study. HR was recorded throughout the whole protocol every minute. Resting BP was recorded before, every 2 min during, and for another 5 min after cycling to ensure full recovery to baseline. The duration of active cycling was recorded at the end of each protocol.

The percentage of amplitude increase was recorded during cycling at the end of each minute, because it has been shown that to maintain cycling performance during fatigue, the amplitude of the current should increase. We also determined the duration before the amplitude of the current achieved 100 percent (140 mA for the knee extensor and flexor muscle groups and 100 mA for the gluteus maximus).

Statistical Analyses

All values are presented as mean ± standard deviation. A repeated-measures analysis of variance (ANOVA) using multivariate approach was used within intervention of VO₂ and EE (rest, warm-up, exercise, and recovery) and between protocols (P200, P350, and P500). A repeated-measures ANOVA was also used to determine the difference in delta EE among the three protocols (P200, P350, and P500). For torque, repeated-measures ANOVA was used for within (precycling, postcycling,

and 48–72 h postcycling) each of the three FES rides and between frequencies (0, 10, 20, 30, 40, 50, 60, 80, and 100 Hz). Statistical analyses were performed using SPSS version 19.0 (IBM Corporation; Armonk, New York), and statistical significance was set at $p < 0.05$.

RESULTS

Table 1 presents the physical characteristics of all participants. Each participant completed three rides as well as the follow-up visits 48 to 72 h postcycling. Time to maximum current intensity (100% of the delivered amplitude of the current) was 4 ± 3 , 4 ± 3 , and 6 ± 8 min in P200, P350, and P500, respectively. FES cycling HRs were not different from resting HR during the three protocols (**Table 2**). Systolic and diastolic BP increased significantly compared with resting BP during P200, P350, and P500 (**Table 2**). During FES cycling, dysreflexia responses ($>140/90$ mm Hg) were noted in 30, 30, and 60 percent in P200, P350, and P500, respectively. During the recovery period, BP dropped but was still elevated compared with the resting period.

Absolute and Relative Oxygen Uptake

Resting VO_2 was not different (0.22 ± 0.075 L/min) across the three FES protocols. Delta VO_2 between exercise and rest after P200, P350, and P500 were 0.13 ± 0.095 , 0.19 ± 0.097 , and 0.18 ± 0.095 L/min, respectively ($p = 0.30$).

There was no difference in absolute VO_2 or relative VO_2 among the three FES protocols ($p = 0.70$). Relative

VO_2 across through the three FES protocols increased from 2.7 ± 0.85 to 4.9 ± 1.8 mL/kg/min, resting to exercise, respectively ($p = 0.001$). Relative VO_2 during recovery (3.77 ± 1.12 mL/kg/min) was greater ($p = 0.001$) than both resting (2.7 ± 0.85 mL/kg/min) and warm-up (3.16 ± 0.95 mL/kg/min) periods. However, it remained lower ($p = 0.001$) than the exercise period (4.9 ± 1.8 mL/kg/min).

Cycling Energy Expenditure

Cycling EE (kilocalories per minute) collapsed across the three FES protocols increased significantly compared with the resting and warm-up periods. Delta EE between exercise and rest was 42 percent greater ($p = 0.07$) in P350 (1.05 ± 0.5 Kcal/min) and P500 (0.99 ± 0.5 Kcal/min) than in P200 (0.73 ± 0.47 Kcal/min) (**Figure 2(a)**). Recovery VO_2 was 23 percent greater in P350 than in P200 ($p = 0.03$) (**Figure 2(b)**). Cycling time to fatigue was not different among the three FES cycling protocols. Respiratory exchange ratio (VCO_2/VO_2) increased significantly ($p < 0.001$) during exercise (1.05 ± 0.18) compared with both the resting (0.85 ± 0.15) and warm-up (0.85 ± 0.15) periods and remained unchanged during the recovery period (1.05 ± 0.18).

Time to Fatigue

Time to fatigue was 11 ± 8 , 10 ± 8 , and 10 ± 8 min in P200, P350, and P500, respectively ($p = 0.70$). The active exercise time in P200, P350, and P500 ranged from 3 to 27, 3 to 26, and 5 to 29 min in P200, P350, and P500, respectively.

Table 1.

Physical characteristics of individuals with motor complete spinal cord injury (SCI).

Person with SCI	Age (yr)	Sex	Weight (kg)	BMI	Level of Injury	AIS (Score)	FM (kg)	FFM (kg)	%FM
1	45	M	61.7	21.3	C6	B	12.7	49.0	21.0
2	47	M	125.2	37.4	C5	A	34.2	91.0	27.0
3	49	M	71.2	21.3	T2	A	16.7	54.5	23.5
4	29	M	89.0	29.0	C5/C6	B	25.6	63.3	23.0
5	43	F	61.7	22.7	C6/C7	A	25.2	37.4	40.0
6	28	M	76.3	24.9	T6	A	12.1	64.2	16.0
7	42	M	61.6	18.4	C7	A	15.1	44.8	25.0
8	47	M	71.8	22.9	C5/C6	A	18.5	53.3	25.0
9	56	M	73.2	25.3	C5/C6	A	18.3	54.9	25.0
10	56	M	77.6	22.7	C6/C7	B	16.1	61.4	21.0
Mean \pm SD	44 \pm 10	—	77 \pm 19	25 \pm 4	—	—	19.5 \pm 7.0	57.4 \pm 15.0	24.7 \pm 7.0

%FM = percentage of fat mass, AIS = American Spinal Injury Association Impairment Scale, BMI = body mass index, C = cervical, F = female, FFM = fat-free mass, FM = fat mass, M = male, SD = standard deviation, T = thoracic.

Table 2.

Blood pressure (BP) and mean \pm standard deviation heart rate (HR) during rest, exercise, and recovery periods of three functional electrical stimulation cycling protocols in persons with spinal cord injury.

Protocol	Rest	Exercise	Recovery	<i>p</i> -Value*
P200				
BP (mm Hg)	103/68	128/81	121/75	0.01
HR (beats/min)	79 \pm 13	78 \pm 14	79 \pm 14	0.90
P350				
BP (mm Hg)	98/62	138/85	125/73	0.001
HR (beats/min)	75 \pm 16	77 \pm 13	83 \pm 14	0.80
P500				
BP (mm Hg)	102/65	144/91	119/73	0.005
HR (beats/min)	78 \pm 14	76 \pm 19	79 \pm 18	0.60

*Statistical outcomes of repeated-measure analysis of variance of systolic BP, diastolic BP, and HR.

P200 = pulse duration of 200 μ s, P350 = pulse duration of 350 μ s, P500 = pulse duration of 500 μ s.

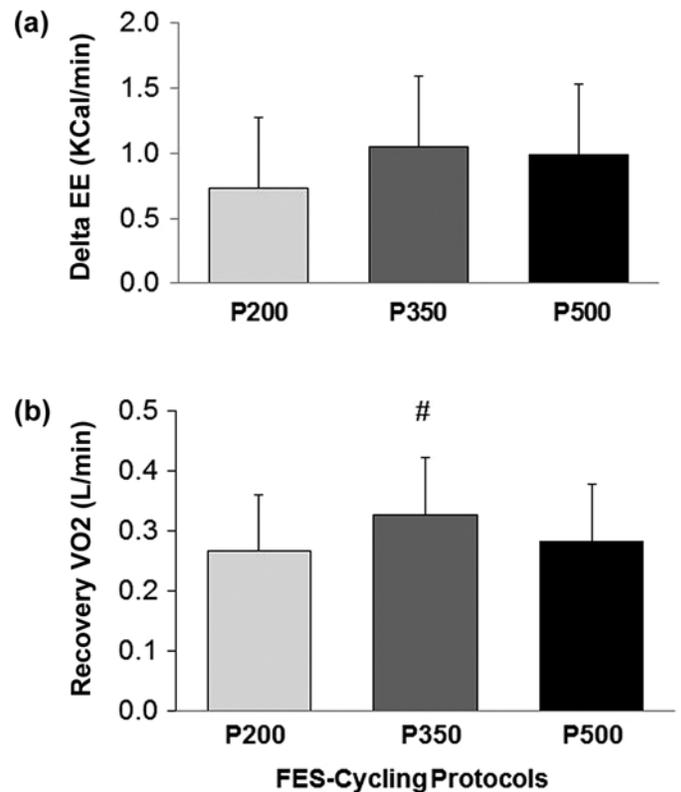
Force-Frequency Curves and Muscle Fatigue After Administering Three Pulse Durations

The pattern of reduction in knee extensor torque was similar across the three (P200, P350, and P500) protocols (**Figure 3**). There were no differences in the outcomes of the three pulse durations on muscle fatigue. Percentage of fatigue calculated immediately after cycling using P200, P350, and P500 was not different ($p = 0.50$) among the three protocols; however, there was a trend toward greater reduction in the knee extensor torque following P200 compared with P350 and P500 that ranged from 47 to 65 percent immediately after FES cycling. Percentage of fatigue 48 to 72 h after FES cycling was not different among the three protocols ($p = 0.90$) (**Table 2**).

Pattern of Force-Frequency Curves Immediately After and 48 to 72 Hours After Functional Electrical Stimulation Leg Cycling

Force-frequency curves were significantly decreased immediately after ($p < 0.001$) and 48 to 72 h after ($p < 0.001$) FES cycling compared with precycling in the three protocols (**Figure 3**). A frequency of 20 Hz showed the greatest reduction in knee extensor torque compared with the other frequencies. Following 48 to 72 h, knee extensor torque following P200 continued to show a decrement compared with the precycling isometric torque (2%–33%). The differences in percentage of fatigue between immediately after and 48 to 72 h after cycling were not different ($p = 0.20$) following P200 (**Table 3**).

In P350, percent reduction of knee extensor torque ranged from 33 to 59 percent immediately postcycling,

**Figure 2.**

(a) Delta energy expenditure (EE) between exercise and resting periods following three functional electrical stimulation (FES) protocols. Delta EE is 42 percent greater in P500 and P350 compared with P200 ($p = 0.07$). Delta EE was 3.07, 4.40, and 4.15 KCal/min for P200, P350, and P500, respectively. (b) Recovery oxygen uptake (VO₂) is 23 percent greater in P350 than P200 ($p = 0.03$). #Significant difference between P200 and P350. P200 = pulse duration of 200 μ s, P350 = pulse duration of 350 μ s, P500 = pulse duration of 500 μ s.

whereas the reduction in torque ranged from 6 to 19 percent following 48 to 72 h. After P350, 20 Hz showed the greatest reduction in torque compared with the other frequencies immediately after and 48 to 72 h after cycling. A similar pattern was noted in P500 immediately after (30%–58%) and 48 to 72 h after cycling (7%–23%).

DISCUSSION

Administering different pulse durations during FES cycling did not appear to affect cycling performance, EE, or muscle fatigue. P350 appears to have a greater effect

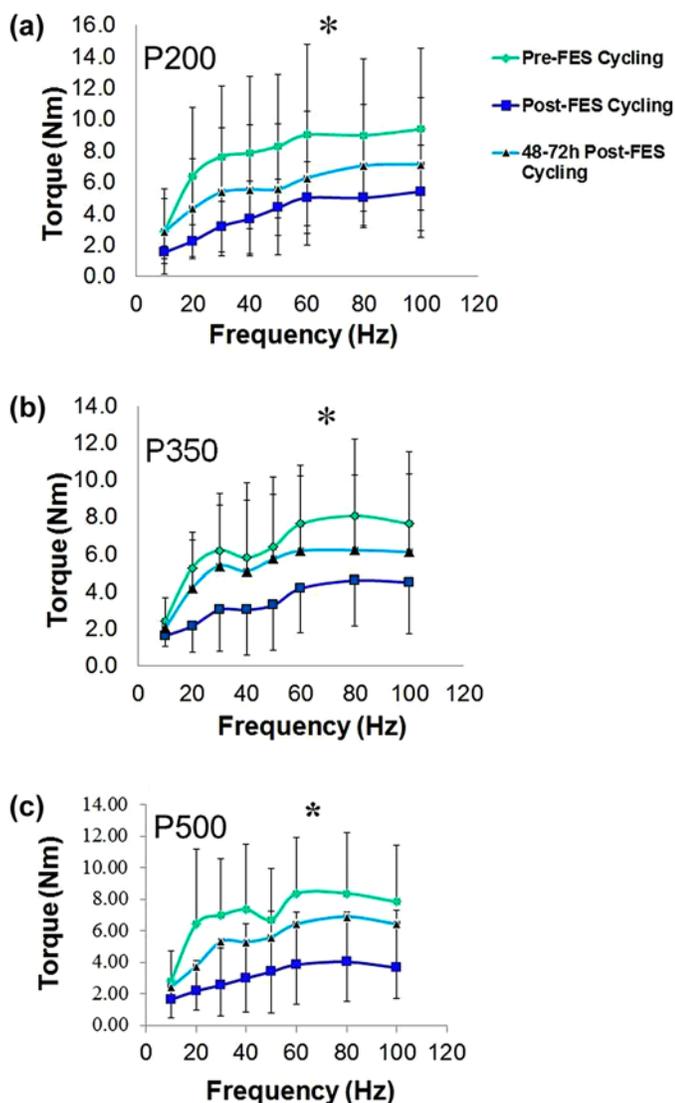


Figure 3. Force-frequency curve before functional electrical stimulation (FES) cycling, immediately after FES cycling, and 48 to 72 h after FES cycling in (a) P200, (b) P350, and (c) P500 protocols. *Pattern is significantly different from pre-FES cycling ($p < 0.05$). P200 = pulse duration of 200 μ s, P350 = pulse duration of 350 μ s, P500 = pulse duration of 500 μ s.

on delta EE as well as oxygen kinetics during recovery. A major finding of the current study is that an acute bout of FES cycling, despite the administered pulse duration, caused delayed-onset muscle fatigue that persisted 48 to 72 h postcycling. This finding has significant clinical implications on how to dose exercise frequency early in a rehabilitation program to train persons with SCI.

Table 3.

Percentage of fatigue as outcome of three functional electrical stimulation cycling protocols immediately postcycling and 48 to 72 h postcycling compared with precycling in men with spinal cord injury.

Protocol	Frequency (Hz)							
	10	20	30	40	50	60	80	100
P200								
Immediately Postcycling	28	45*	41*	36*	36*	22	33	34*
48–72 h Postcycling	6	3	1	4	24	5	10	15
P350								
Immediately Postcycling	18	55*	40*	42*	44*	39	35	39*
48–72 h Postcycling	9	18	6	10	11	15	19	18
P500								
Immediately Postcycling	30	55*	58*	52*	44*	52	46	52*
48–72 h Postcycling	7	23	23	16	14	25	13	13

*Percentage of fatigue is significantly different from that elicited at 10 Hz ($p = 0.001-0.047$) as determined by repeated-measures analysis of variance.

P200 = pulse duration of 200 μ s, P350 = pulse duration of 350 μ s, P500 = pulse duration of 500 μ s.

Administering pulse durations of different lengths did attenuate the time required to reach the maximum current amplitude, preadjusted for each muscle group. The maximum current amplitude is defined as 100 percent of 140, 140, and 100 mA adjusted for knee extensor, knee flexor, and gluteus maximus muscle groups, respectively. There is an inverse relationship between pulse duration and amplitude of the current [26]. Delaying the progress to maximum current amplitude may be a helpful strategy to delay the process of rapid muscle fatigue during FES cycling. The speed of cycling was set to maintain 45 RPM during the three bouts. When muscle fatigue ensued as determined by the drop in RPM, the amplitude of the current has increased to maintain the cycling speed. However, this strategy was no longer effective once the maximum amplitude of the current was reached. This was followed by a decline in the speed until it reached the fatigue threshold (18 RPM) and the FES bike would automatically switch to the cool-down mode.

One may assume that at a constant frequency, the current density that is the product of both the pulse duration and amplitude of the current may differ among the three FES protocols and influence the results of the study [14,26]. It appeared that the three FES protocols had a latency of 4 min (P200 or P350) or 6 min (P500) before reaching the maximum current amplitude. This indicated that all participants were exercising at the maximum current amplitude within 4 min from FES-LCE and the pulse duration at a given protocol had a greater influence on the

current density than on the amplitude of the current. This may also help to explain the greater percentage of autonomic dysreflexia in P500 compared with P350 or P200. Setting the pulse duration at 500 μ s exposed 6 of the 10 participants to an acute episode of autonomic dysreflexia (BP > 140/90 mm Hg) compared with only 3 when either 200 or 350 μ s were used. P500 may have resulted in a greater current density under the electrodes and trigger pain fibers below the level of SCI and cause an episode of autonomic dysreflexia, a phenomenon previously described during FES cycling [27].

Absolute and Relative Oxygen Uptake

Another problem in the current FES bike is a limited capacity to provide adequate exercise intensity to stress cardiovascular or bone systems. Cycling at a speed greater than 50 RPM may not be a feasible strategy to increase FES cycling exercise intensity, because the bike mechanics does not support such speed. The limited exercise intensity may require a high volume of FES cycling with duration up to 1 yr to partially reverse bone loss after SCI [28]. FES cycling was not sufficient to improve cardiovascular variables compared with hybrid exercise that utilized both upper limbs and FES cycling after SCI [29]. In the current study, a limited exercise EE and VO_2 during FES cycling was noted compared with the resting state accompanied with HR that barely exceeded 83 beats/min, which is not much above resting and lower than might be expected from full parasympathetic deactivation (**Table 2**). Muscle activation during FES cycling is limited because of the significant skeletal muscle atrophy, infiltration of intramuscular fat, and changes in skeletal muscle architecture after SCI [19]. Other factors similar to cardiac output, arteriovenous difference, and decrement in mitochondrial size and functions are responsible for limited VO_2 during submaximal FES-LCE [30–31]. A review that determines the peak and submaximal VO_2 during FES-LCE showed that range of the value is 0.47 to 2.5 and 0.51 to 1.36 L/min, respectively [30]. A recent study showed that submaximal FES-LCE elicited VO_2 about 0.41 and 0.48 L/min between 40 and 60 percent of peak VO_2 , respectively [29]. These values are comparable with the absolute values obtained in the current study, which is reasonable to suggest that our participants were exercising at an intensity lower or close to 40 percent of their VO_2 peak. Hunt et al. demonstrated that power output during FES is a function of both the mechanical power during active stimulation and negative power during passive cycling performed by

the motor during the desired cadence [32]. In the current study, the motor of the bike was turned off to determine the absolute energetic of the exercising skeletal muscle, which may have further limited oxygen utilization during FES-LCE. This could be further complicated by the fact that 8 of the 10 participants had tetraplegia, which is known to be on the lowest extreme level of physical activity [31]. The 23 percent greater VO_2 during recovery following P350 could be explained by the greater oxygen debt compared with P200 [30].

Previous work has indicated that adjusting pulse duration from 150 to 450 μ s increased the evoked torque and total activated muscle CSA as measured by T2-weighted magnetic resonance imaging [13]. The increase in motor unit recruitment has been previously suggested to help increase VO_2 [33–34]. Therefore, the increase in both the evoked torque and the activated CSA during surface electrical stimulation should result in increasing the actin-myosin cycling attachments and further increase VO_2 and adenosine triphosphate consumption. Elder et al. measured VO_2 relative to the recruited muscle mass during dynamic leg extension after using surface electrical stimulation (450 μ s and 30 Hz) of the knee extensor muscles [33]. Dynamic muscle action resulted in delta VO_2 increase by 0.24 L/min after an acute bout of exercise for 3 min [33]. FES cycling after P350 resulted in 0.19 L/min, which is comparable with previous findings. Despite the extensive skeletal muscle atrophy in persons with chronic SCI, individuals with SCI manage to evoke similar metabolic oxygen consumption to what has been reported in nondisabled controls utilizing dynamic electrical stimulation.

Cycling Energy Expenditure

Obesity, which is defined as excessive regional and whole body fatness, is a major problem after SCI that leads to several metabolic abnormalities [24,35]. Delta EE between exercise and rest was 42 percent greater in P350 than in P200 (+0.32 Kcal/min). The average active cycling time to fatigue was ~10 min. This means that P350 can consume an extra 3.2 Kcal/min at the end of active cycling compared with P200. We previously trained participants with SCI for about 1 h, 3 or 5 d/wk for 16 wk [6,19]. This means that adjusting to P350 during FES cycling could result in an extra 20 Kcal/session and 60 to 100 Kcal/wk. Therefore, a person with SCI may consume an additional 960 to 1,600 Kcal over 16 wk of training after using P350. Although this may appear

trivial, it is a step toward optimizing stimulation parameters to maximize EE and reduce the obesity problem after SCI. These findings may serve to explain previous research findings that showed no reduction in body weight following FES cycling [21]. EE was calculated using the Weir equation; it is possible that an increase in exercising VCO_2 is responsible for the greater delta EE during P350 compared with P200. Greater VCO_2 may have resulted from greater lactate concentration and dissociation of hydrogen ions that leads to bicarbonate formation and more VCO_2 release.

It is worth noting that our participants had restricted sympathetic outflow as a result of their above T6 SCI level. During exercise, VO_2 is partially increased as result of increase in the cardiac output, which is a function of both HR and stroke volume. The results in **Table 2** showed no cardio-acceleration response as reported by lack of significant changes in HR response. Previously, vagal withdrawal was noted to cause cardio-acceleration in individuals with SCI with restricted sympathetic outflow [36]; however, the current results did not support this observation. It is possible that limited increase in VO_2 is partially explained by improvement in venous return, which leads to partial increase in the stroke volume.

Fatigue

Stimulation parameters may be an essential component to modulate or control muscle fatigue during electrical stimulation [11–12]. Gorgey et al. previously studied the independent effects of the amplitude, frequency of pulses, and pulse duration on muscle fatigue [10]. The findings showed that compared with the frequency of the pulses neither the current amplitude nor the pulse duration significantly affected knee extensor muscle fatigue [10]. Increasing the frequency from 25 to 100 Hz increased the percentage of fatigue from 39 to 76 percent, whereas altering the pulse duration from 150 to 450 μ s did not influence percentage of fatigue [10]. The current study supports the previous findings, such that administering three different pulse durations at 30 Hz did not affect the percentage decline in the evoked torque or the pattern of the force-frequency curve.

Bickel et al. reported that acute bouts of electrical stimulation resulted in two times the percent decline in evoked torque in those with SCI compared with nondisabled controls [11]. They noticed that recovery of force was also challenged during repetitive isometric actions and that peak torque remained declined by 22 percent in

the group with SCI 3 d later [11]. They concluded that acute bouts of isometric actions evoked via electrical stimulation may induce delayed-onset muscle damage in individuals with SCI. Mahoney et al. studied the effects of an acute bout of isometric actions on the magnitude and recovery of low-frequency fatigue in individuals with SCI [12]. At 24 h poststimulation, the ratio of 20/100 Hz was significantly declined in the group with SCI [12]. They concluded that the delay in the recovery of force after SCI is confounded by muscle injury [12]. The pattern of the force-frequency curve was still challenged 48 to 72 h after FES cycling. The findings may suggest that twice-weekly FES training may be reasonable to minimize delayed onset fatigue or potential muscle damage that may frequently occur with repetitive bouts of stimulation. The absolute isometric torque values of knee extensors before cycling were smaller compared with previous reports [11–12]. A possible explanation is that we used a battery-operated unit to evoke knee extensor torques. However, a previous study showed no difference in the evoked isometric torques between battery-operated and plugged-in units [37].

Study Limitations

The current study recruited a convenience sample of individuals with chronic motor complete SCI. Despite our effort to ensure a homogenous sample, it is interesting to note the wide variability in response to FES cycling bouts among the participants. One participant could cycle for more than 25 min compared with others who were only able to cycle for 3 min. This can be partially explained by the wide range of percentage of body fat mass in the study participants, from 16 to 40 percent (**Table 1**), which may have negatively affected cycling performance and resulted in premature fatigue for those participants with a greater percentage of fat mass. Recently, greater intramuscular and subcutaneous adipose tissue accumulation has been shown to increase in the current intensity required to evoke leg extension [19]. The bike motor was turned off after the warm-up period to allow rapid muscle fatigue. Turning off the bike servo motor triggered muscle spasms and resulted in rapid termination of the FES cycling bout with only a few participants. We administered the three FES cycling protocols in a random fashion and allowed an interval of 1 wk to avoid possible contamination of the other two FES protocols. Possible contamination from previous FES bouts was minimized because we noted full recovery of the knee extensors evoking torque before

starting the next FES protocol. We have employed a 3×3 repeated-measures ANOVA design with only 10 subjects; this may have resulted in type II error and challenged the statistical significance of the pulse durations on VO_2 or EE. The available scientific evidence did not allow accurate power calculation before conducting the study, and the current work may serve as preliminary data to support future power calculation for similar research questions.

CONCLUSIONS

Administering different pulse durations during acute bouts of FES cycling did not affect cycling VO_2 or EE. However, exercising at P350 resulted in a greater delta EE between FES exercise and resting than at P200. This finding may encourage clinicians to select a higher pulse duration to optimize EE during FES cycling. Exercising at P500 elicited a dysreflexia response compared with both P200 and P350, which should be considered as a safety precaution for individuals with SCI who are hypertensive. Moreover, administering different pulse durations did not appear to influence percentage of decline in the evoked torque or the pattern of the force-frequency curve. However, it appears that an acute bout of FES cycling can lead to delayed-onset muscle fatigue. These findings may have important clinical implications in dosing and spacing FES exercise sessions early in rehabilitation of persons with SCI or those who have not previously trained using FES. Further studies are warranted to examine the effects of adjusting different stimulation parameters on FES cycling after SCI.

ACKNOWLEDGMENTS

Author Contributions:

Study concept and design: A. S. Gorgey, D. R. Gater.

Acquisition of data: A. S. Gorgey, H. J. Poarch, D. R. Dolbow.

Analysis and interpretation of data: A. S. Gorgey.

Drafting of manuscript: A. S. Gorgey.

Administrative, technical, or material support: A. S. Gorgey, D. R. Gater.

Study supervision: A. S. Gorgey, D. R. Gater.

Medical support to participants: T. Castillo, D. R. Gater.

Financial Disclosures: The authors have declared that no competing interests exist.

Funding/Support: This material was unfunded at the time of manuscript preparation.

Additional Contributions: We would like to thank the participants who volunteered to take part in this study. We would also like to thank Restorative Therapies Inc for supplying the electrodes that were used and the Hunter Holmes McGuire VA Medical Center for providing the space and the environment to conduct the current study. Dr. Gorgey is a Career Development Award-2 recipient (grant B7867-W) and is currently supported by the VA Rehabilitation Research & Development Service.

Institutional Review: The Hunter Holmes McGuire VA Medical Center Institutional Review Board approved the study.

Participant Follow-Up: The authors plan to inform participants of the publication of this study.

REFERENCES

- Griffin L, Decker MJ, Hwang JY, Wang B, Kitchen K, Ding Z, Ivy JL. Functional electrical stimulation cycling improves body composition, metabolic and neural factors in persons with spinal cord injury. *J Electromyogr Kinesiol.* 2009;19(4):614–22. [PMID:18440241] <http://dx.doi.org/10.1016/j.jelekin.2008.03.002>
- Johnston TE, Modlesky CM, Betz RR, Lauer RT. Muscle changes following cycling and/or electrical stimulation in pediatric spinal cord injury. *Arch Phys Med Rehabil.* 2011; 92(12):1937–43. [PMID:22133240] <http://dx.doi.org/10.1016/j.apmr.2011.06.031>
- Sköld C, Lönn L, Harms-Ringdahl K, Hultling C, Levi R, Nash M, Seiger A. Effects of functional electrical stimulation training for six months on body composition and spasticity in motor complete tetraplegic spinal cord-injured individuals. *J Rehabil Med.* 2002;34(1):25–32. [PMID:11900259] <http://dx.doi.org/10.1080/165019702317242677>
- Gorgey AS, Mather KJ, Cupp HR, Gater DR. Effects of resistance training on adiposity and metabolism after spinal cord injury. *Med Sci Sports Exerc.* 2012;44(1):165–74. [PMID:21659900] <http://dx.doi.org/10.1249/MSS.0b013e31822672aa>
- Dudley-Javoroski S, Saha PK, Liang G, Li C, Gao Z, Shields RK. High dose compressive loads attenuate bone mineral loss in humans with spinal cord injury. *Osteoporos Int.* 2012;23(9):2335–46. [PMID:22187008] <http://dx.doi.org/10.1007/s00198-011-1879-4>
- Dolbow DR, Gorgey AS, Ketchum JM, Moore JR, Hackett LA, Gater DR. Exercise adherence during home-based functional electrical stimulation cycling by individuals with spinal cord injury. *Am J Phys Med Rehabil.* 2012; 91(11):922–30. [PMID:23085704] <http://dx.doi.org/10.1097/PHM.0b013e318269d89f>
- Binder-Macleod SA, Guerin T. Preservation of force output through progressive reduction of stimulation frequency in

- human quadriceps femoris muscle. *Phys Ther.* 1990;70(10): 619–25. [PMID:2217541]
8. Binder-MacLeod SA, Halden EE, Jungles KA. Effects of stimulation intensity on the physiological responses of human motor units. *Med Sci Sports Exerc.* 1995; 27(4):556–65. [PMID:7791587]
<http://dx.doi.org/10.1249/00005768-199504000-00014>
9. Gregory CM, Dixon W, Bickel CS. Impact of varying pulse frequency and duration on muscle torque production and fatigue. *Muscle Nerve.* 2007;35(4):504–9. [PMID:17230536]
<http://dx.doi.org/10.1002/mus.20710>
10. Gorgey AS, Black CD, Elder CP, Dudley GA. Effects of electrical stimulation parameters on fatigue in skeletal muscle. *J Orthop Sports Phys Ther.* 2009;39(9):684–92. [PMID:19721215]
<http://dx.doi.org/10.2519/jospt.2009.3045>
11. Bickel CS, Slade JM, Dudley GA. Long-term spinal cord injury increases susceptibility to isometric contraction-induced muscle injury. *Eur J Appl Physiol.* 2004;91(2–3): 308–13. [PMID:14586584]
<http://dx.doi.org/10.1007/s00421-003-0973-5>
12. Mahoney E, Puetz TW, Dudley GA, McCully KK. Low-frequency fatigue in individuals with spinal cord injury. *J Spinal Cord Med.* 2007;30(5):458–66. [PMID:18092561]
13. Gorgey AS, Mahoney E, Kendall T, Dudley GA. Effects of neuromuscular electrical stimulation parameters on specific tension. *Eur J Appl Physiol.* 2006;97(6):737–44. [PMID:16821023]
<http://dx.doi.org/10.1007/s00421-006-0232-7>
14. Gorgey AS, Dudley GA. The role of pulse duration and stimulation duration in maximizing the normalized torque during neuromuscular electrical stimulation. *J Orthop Sports Phys Ther.* 2008;38(8):508–16. [PMID:18678958]
<http://dx.doi.org/10.2519/jospt.2008.2734>
15. Kesar T, Chou LW, Binder-MacLeod SA. Effects of stimulation frequency versus pulse duration modulation on muscle fatigue. *J Electromyogr Kinesiol.* 2008;18(4):662–71. [PMID:17317219]
<http://dx.doi.org/10.1016/j.jelekin.2007.01.001>
16. Chou LW, Binder-MacLeod SA. The effects of stimulation frequency and fatigue on the force-intensity relationship for human skeletal muscle. *Clin Neurophysiol.* 2007; 118(6):1387–96. [PMID:17466581]
<http://dx.doi.org/10.1016/j.clinph.2007.02.028>
17. Hultman E, Sjöholm H, Jäderholm-Ek I, Krynicky J. Evaluation of methods for electrical stimulation of human skeletal muscle in situ. *Pflugers Arch.* 1983;398(2):139–41. [PMID:6622220]
<http://dx.doi.org/10.1007/BF00581062>
18. Troiani D, Filippi GM, Bassi FA. Nonlinear tension summation of different combinations of motor units in the anesthetized cat peroneus longus muscle. *J Neurophysiol.* 1999;81(2):771–80. [PMID:10036276]
19. Gorgey AS, Cho GM, Dolbow DR, Gater DR. Differences in current amplitude evoking leg extension in individuals with spinal cord injury. *NeuroRehabilitation.* 2013;33(1): 161–70. [PMID:23949041]
20. Baldi JC, Jackson RD, Moraille R, Mysiw WJ. Muscle atrophy is prevented in patients with acute spinal cord injury using functional electrical stimulation. *Spinal Cord.* 1998;36(7):463–69. [PMID:9670381]
<http://dx.doi.org/10.1038/sj.sc.3100679>
21. Gorgey AS, Harnish CR, Daniels JA, Dolbow DR, Keeley A, Moore J, Gater DR. A report of anticipated benefits of functional electrical stimulation after spinal cord injury. *J Spinal Cord Med.* 2012;35(2):107–12. [PMID:22525324]
<http://dx.doi.org/10.1179/204577212X13309481546619>
22. Gorgey AS, Dolbow DR, Dolbow JD, Khalil RK, Gater DR. The effects of electrical stimulation on body composition and metabolic profile after spinal cord injury - Part II. *J Spinal Cord Med.* 2014 Jul 8. Epub ahead of print. [PMID:25001669]
23. Janssen TW, Pringle DD. Effects of modified electrical stimulation-induced leg cycle ergometer training for individuals with spinal cord injury. *J Rehabil Res Dev.* 2008; 45(6):819–30. [PMID:19009468]
<http://dx.doi.org/10.1682/JRRD.2007.09.0153>
24. Gorgey AS, Dolbow DR, Dolbow JD, Khalil RK, Castillo C, Gater DR. Effects of spinal cord injury on body composition and metabolic profile - Part I. *J Spinal Cord Med.* 2014;37(6):693–702. [PMID:25001559]
25. Burnham R, Martin T, Stein R, Bell G, MacLean I, Steadward R. Skeletal muscle fibre type transformation following spinal cord injury. *Spinal Cord.* 1997;35(2):86–91. [PMID:9044514]
<http://dx.doi.org/10.1038/sj.sc.3100364>
26. Robinson AJ. Basic concepts in electricity and contemporary terminology in electrotherapy. In: Robinson AJ, Snyder-Mackler L, editors. *Clinical electrophysiology.* 3rd ed. Philadelphia (PA): Wolters Kluwer Health/Lippincott Williams & Wilkins; 2008. p. 1–26.
27. Ashley EA, Laskin JJ, Olenik LM, Burnham R, Steadward RD, Cumming DC, Wheeler GD. Evidence of autonomic dysreflexia during functional electrical stimulation in individuals with spinal cord injuries. *Paraplegia.* 1993;31(9): 593–605. [PMID:8247602]
<http://dx.doi.org/10.1038/sc.1993.95>
28. Frotzler A, Coupaud S, Perret C, Kakebeeke TH, Hunt KJ, Donaldson NN, Eser P. High-volume FES-cycling partially reverses bone loss in people with chronic spinal cord injury. *Bone.* 2008;43(1):169–76. [PMID:18440891]
<http://dx.doi.org/10.1016/j.bone.2008.03.004>

29. Hasnan N, Ektas N, Tanhoffer AI, Tanhoffer R, Fornusek C, Middleton JW, Husain R, Davis GM. Exercise responses during functional electrical stimulation cycling in individuals with spinal cord injury. *Med Sci Sports Exerc.* 2013;45(6):1131–38. [PMID:23685444] <http://dx.doi.org/10.1249/MSS.0b013e3182805d5a>
30. Hettinga DM, Andrews BJ. Oxygen consumption during functional electrical stimulation-assisted exercise in persons with spinal cord injury: Implications for fitness and health. *Sports Med.* 2008;38(10):825–38. [PMID:18803435] <http://dx.doi.org/10.2165/00007256-200838100-00003>
31. Hagerman F, Jacobs P, Backus D, Dudley GA. Exercise responses and adaptations in rowers and spinal cord injury individuals. *Med Sci Sports Exerc.* 2006;38(5):958–62. [PMID:16672851] <http://dx.doi.org/10.1249/01.mss.0000218131.32162.ce>
32. Hunt KJ, Hosmann D, Grob M, Saengsuwan J. Metabolic efficiency of volitional and electrically stimulated cycling in able-bodied subjects. *Med Eng Phys.* 2013;35(7):919–25. [PMID:23253953] <http://dx.doi.org/10.1016/j.medengphy.2012.08.023>
33. Elder CP, Mahoney ET, Black CD, Slade JM, Dudley GA. Oxygen cost of dynamic or isometric exercise relative to recruited muscle mass. *Dyn Med.* 2006;5(1):9. [PMID:16965630] <http://dx.doi.org/10.1186/1476-5918-5-9>
34. Kushmerick MJ, Meyer RA, Brown TR. Regulation of oxygen consumption in fast- and slow-twitch muscle. *Am J Physiol.* 1992;263(3 Pt 1):C598–606. [PMID:1415510]
35. Gater DR Jr. Obesity after spinal cord injury. *Phys Med Rehabil Clin N Am.* 2007;18(2):333–51, vii. [PMID:17543776] <http://dx.doi.org/10.1016/j.pmr.2007.03.004>
36. Jacobs PL, Nash MS. Exercise recommendations for individuals with spinal cord injury. *Sports Med.* 2004;34(11):727–51. [PMID:15456347] <http://dx.doi.org/10.2165/00007256-200434110-00003>
37. Lyons CL, Robb JB, Irrgang JJ, Fitzgerald GK. Differences in quadriceps femoris muscle torque when using a clinical electrical stimulator versus a portable electrical stimulator. *Phys Ther.* 2005;85(1):44–51. [PMID:15623361]

Submitted for publication February 22, 2014. Accepted in revised form July 29, 2014.

This article and any supplementary material should be cited as follows:

Gorgey AS, Poarch HJ, Dolbow DR, Castillo T, Gater DR. Effect of adjusting pulse durations of functional electrical stimulation cycling on energy expenditure and fatigue after spinal cord injury. *J Rehabil Res Dev.* 2014; 51(9):1455–68.

<http://dx.doi.org/10.1682/JRRD.2014.02.0054>



