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Original Article

Resistance training and locomotor recovery after incomplete spinal cord injury: a case series

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Study design: Longitudinal intervention case series.
Objective: To determine if a 12-week resistance and plyometric training program results in improved muscle function and locomotor speed after incomplete spinal cord injury (SCI).
Setting: University research setting.
Methods: Three ambulatory individuals with chronic (18.7 ± 2.2 months post injury) motor incomplete SCI completed 12 weeks of lower extremity resistance training combined with plyometric training (RPT). Muscle maximum cross-sectional area (max-CSA) of the knee extensor (KE) and plantar flexor (PF) muscle groups was determined using magnetic resonance imaging (MRI). In addition, peak isometric torque, time to peak torque (T_{20-80}), torque developed within the initial 220 ms of contraction ($torque_{220}$) and average rate of torque development (ARTD) were calculated as indices of muscle function. Maximal as well as self-selected gait speeds were determined pre- and post-RPT during which the spatio-temporal characteristics, kinematics and kinetics of gait were measured.
Results: RPT resulted in improved peak torque production in the KE ($28.9 \pm 4.4\%$) and PF ($35.0 \pm 9.1\%$) muscle groups, as well as a decrease in T_{20-80}, an increased $torque_{220}$ and an increase ARTD in both muscle groups. In addition, an increase in self-selected (pre-RPT = 0.77 m/s; post-RPT = 1.03 m/s) and maximum (pre-RPT = 1.08 m/s; post-RPT = 1.47 m/s) gait speed was realized. Increased gait speeds were accompanied by bilateral increases in propulsion and hip excursion as well as increased lower extremity joint powers.
Conclusions: The combination of lower extremity RPT can attenuate existing neuromuscular impairments and improve gait speed in persons after incomplete SCI.
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Introduction

The proportion of persons that suffer a spinal cord injury (SCI) resulting in an incomplete lesion has risen dramatically over the past 20 years. As a result ~55% of the new injuries sustained in the United States are now classified as incomplete. In addition, the life expectancy for persons with an incomplete injury is higher than after a complete SCI, and is approaching that of noninjured persons, regardless of age at injury.[1] As such, the increased incidence and prevalence of persons with this type of injury necessitates a comprehensive understanding of the adaptations that occur of and the potential for rehabilitative interventions to impact persons with incomplete SCI. Unfortunately, despite the proportion of persons sustaining and subsequently living with incomplete SCI, the preponderance of scientific literature describing the physiological and functional adaptations to SCI involves persons with complete injuries. Accordingly, limited data are available that describe motor function and its impact on functional ability in this large subject cohort.

The ability to independently ambulate is a primary goal of many persons after SCI. However, even though a large number of individuals with incomplete SCI regain some ability to walk, limitations in gait speed may make this method of mobility impractical for activities of daily living. Slow speed combined with other mobility deficits (eg difficulty climbing stairs, curbs, etc.) could negate the ability to safely ambulate in the community, resulting in a perceived disability. Interestingly, rehabilitation practice focusing on compensatory approaches to locomotion has largely been based on the prevailing assumption that neural as well as functional recovery is limited in persons with chronic SCI. However, recent evidence from both animal and human studies indicates that with the appropriate training stimuli, neural as well as muscular

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plasticity can be induced even years after injury.[2,3] Improvements in functional ability, however, vary greatly and the incidence of disability remains high.[4,5]

Previous data suggest that persons after incomplete SCI produce less voluntary torque about the knee and ankle than non injured controls. Perhaps more importantly, impairments in the ability to produce torque in a timely manner as well as a reduced walking velocity are also common to these persons.[6] It is our belief that reduced muscle power generation significantly impacts locomotor function and that functional recovery can be facilitated with rehabilitation interventions that attenuate this impairment. Specifically, the ankle plantar flexor (PF) and knee extensor (KE) muscle groups are of interest primarily because of their purported roles during bipedal locomotion, with torque demands at these joints during walking representing the two highest in the lower extremity. As such, the potential for impaired torque production about these joints to be a limiting factor in locomotor performance, seems high.

The common goal of resistance training programs is to increase maximal strength in the trained musculature. In addition, the focus of plyometric training, which incorporates high-velocity stretch-shortening-type contractions, has been to improve performance in activities requiring fast contractions (eg jumping or sprinting).[7,8] The combination of these two types of training has been shown to be effective in improving both maximal strength as well as muscle power production, and results in improved jump height and sprint speed in neurologically healthy individuals.[7,9] Interestingly, the potential for rehabilitative training-induced changes in muscle strength and power to affect functional ability after incomplete SCI is largely unstudied. In addition, it is unknown and of obvious value whether potential increases in muscle function in these persons identified during strength testing are reflective of improved muscle power output during functional tasks. Accordingly, the challenge is to now develop, evaluate and implement strategies that maximize neuromuscular plasticity in individuals after incomplete SCI with the hopes of resultant improvements in functional capacity and a subsequent decreased disability. As such, the purpose of this study was threefold: (1) to determine if improvements in muscle function can be realized following a combined resistance and plyometric jump-training program in persons with chronic incomplete SCI; (2) to determine if bilateral enhancement of lower extremity strength and power are accompanied by improvements in locomotor ability; and (3) to determine if potential improvements in gait speed are explained by improved task-specific muscle function after training, as indicated by changes in kinetic and kinematic measures during gait.

Methods

Subjects

Three independently ambulatory male subjects with chronic motor-incomplete SCI participated in this study. Criteria for inclusion included: (1) age 18–70 years; (2) first time SCI (C5–T10); (3) medically stable and asymptomatic for bladder infection, decubitis, cardiopulmonary disease or other significant medical complications prohibiting testing and/or training; (4) if using anti-spasticity medication, agreement to maintain current levels throughout study; (5) ability to walk without the use of an assistive device for a minimum of 50 ft. Of note, the decision to limit the scope of our investigation to persons who could walk without the use of an assistive device was deemed necessary so as to increase the likelihood that improvements in ambulatory ability would be reflected by increases in gait speed rather than changes in assistive device. Exclusion criteria were: (1) participation in a rehabilitation or research protocol that could influence outcomes of this study; (2) history of congenital SCI or other disorders that may confound treatment, study and/or evaluation procedures; (3) non-MR-compatible implants or devices, pregnancy or severe claustrophobia. Before participation, written informed consent was obtained from all subjects, as approved by the Institutional Review Board at the University of Florida.

Subject 1, a 22-year-old man (69 kg, 185 cm), suffered a traumatic SCI (T4, 17 months post-injury) and was classified as American Spinal Injury Association (ASIA) impairment level D, with a lower extremity motor score (LEMS) of 44/50. Before resistance and plyometric training (RPT), this subject had a self-selected gait speed of 0.71 m/s and a maximal gait speed of 1.01 m/s. This subject completed 29 sessions of RPT over the 12-week study period.

Subject 2, a 61-year-old man (93 kg, 189 cm), suffered a traumatic SCI (C5, 27 months post-injury) and was classified as ASIA D with a LEMS of 48/50 before RPT. Subject 2 had a self-selected gait speed of 0.82 m/s and a maximal gait speed of 1.18 m/s. Subject 2 completed 30 sessions of RPT over the 12-week study period.

Subject 3, a 58-year-old man (88 kg, 178 cm), suffered a traumatic SCI (C5, 24 months post-injury) and was classified as ASIA D with a LEMS of 35/50. Before RPT this subject had a self-selected gait speed of 0.78 m/s and a maximal gait speed of 1.06 m/s. This subject completed 30 sessions of RPT over the 12-week study period.

Resistance training program

Lower extremity progressive resistance training was 12 weeks in duration and subjects completed 2–3 sessions/week for a total of 30 sessions. Resistance exercises included unilateral leg press, knee extension/flexion, hip extension/flexion and ankle plantar flexion exercises performed on adjustable load weight machines. During the initial training session, a predicted 1-repetition maximum (1-RM) was calculated for each subject and for each exercise. 1-RM was determined using a prediction table based on a single set to volitional failure with load that allowed between 6 and 12 repetitions.[10] During subsequent training sessions, subjects performed 2–3 sets of 6–12 repetitions at a relative

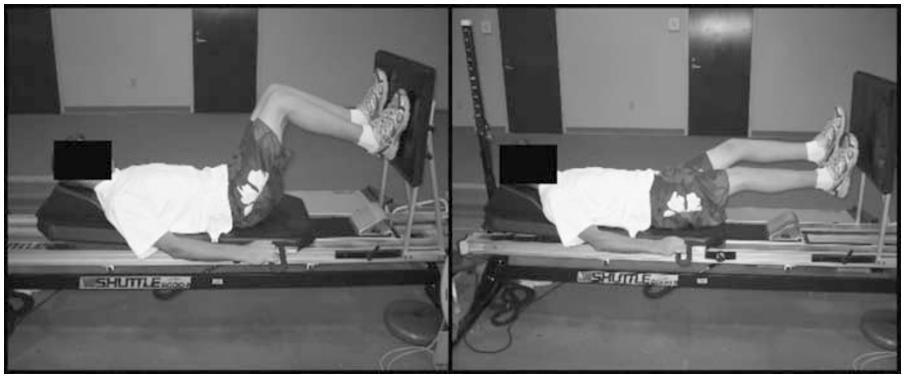

Figure 1 Example of ballistic jump-training device

intensity of ~70–85% of predicted 1-RM. Maximal strength was evaluated weekly to assess for training-related improvements, and exercise loads were adjusted accordingly. Specifically, if the subject achieved the target number of repetitions for all prescribed sets of a given exercise, a new predicted 1-RM was prescribed, and resistance was increased for subsequent training sessions.

Plyometric training
Unilateral plyometric jump-training exercises were performed in both limbs in a supine position on a ballistic jump–training device (ShuttlePro MVP®, Contemporary Design Group, Figure 1). Session intensity for this exercise was modified by changing either the resistance or the number of ground contacts and progressed over the training period, accordingly. Briefly, after familiarization with the training device, subjects completed a total of 20 unilateral ground contacts (eg jumps) with each limb at a resistance of ~25% of body mass. Thereafter, upon successful completion of at least 20 ground contacts per limb (eg complete clearance from foot plate), resistance was increased in increments of 10 lbs. When a new resistance was set, repetition goal was set at 10 ground contacts per limb for the initial session. Subsequent sessions allowed for up to 20 contacts per limb. Thus, a minimum of two sessions at a given resistance was required before load was increased. Resistance was held consistent between limbs throughout the training program.

Magnetic resonance imaging
Magnetic resonance imaging (MRI) was used to determine the maximal cross-sectional area (max-CSA) of muscle groups in the anterior and posterior compartments of the thigh (KE and knee flexors (KF)) and posterior compartment of the leg (PF). 3-D data were collected in a clinical 1.5 T magnet (General Electric) using a fast gradient-echo sequence, with repetition time = 100 ms, echo time = 10 ms, flip angle = 30°, encoding matrix = 256 × 256, field of view = 16–24 cm and a 7 mm slice thickness. Chemically selective fat suppression was employed to enhance the definition between muscles. A standard quadrature extremity coil was utilized in the collection of images of the leg to enhance spatial resolution, whereas a body coil was utilized for thigh imaging. The fat-free max-CSA was determined pre- and post-RPT using a custom-designed interactive computer program that allows for correction of partial volume filling effects, as described previously.[11,12] In addition, we previously showed that the max-CSA of individual PF muscles can be measured reliably, with test–retest correlation coefficients of 0.978–0.999.[11]

Dynamometry
Strength measurements were performed in the PF and KE muscle groups using a Biodex isokinetic dynamometer (Biodex Corp Shirley, NY, USA). PF strength was assessed with subjects seated in a semi-reclined (~70° hip flexion) position, with the knee flexed ~15° and the ankle in an anatomical neutral position (0° of plantar flexion). The axis of the dynamometer was aligned with the lateral malleolus, and the foot was secured with straps placed at the forefoot and ankle. Proximal stabilization was achieved with straps across the chest, hips and knee. KE strength assessments were performed with subjects seated in the same position used for PF testing, with the exception that the knee was flexed to 90°. The axis of the dynamometer was aligned with the knee joint line, and the leg was secured to the lever arm.

Peak torque (Nm) was defined as the highest isometric torque achieved during three maximal contractions (~3 s contractions separated by a minimum of 60 s rest). In the event that the peak torque values during the three trials differed by more than 5%, additional contractions were performed. In addition to peak torque, values for $T_{20–80}$, $torque_{220}$ and average rate of torque development (ARTD) were also determined both pre- and post-RPT. These measures were used as indices of a subject's ability to produce torque in an explosive manner and account for potential differences in both the timing and magnitude of torque production. $T_{20–80}$, used to represent the time to peak tension, was defined as the amount of time to generate from 20 to 80% of peak

isometric torque. This time interval was chosen to minimize potential errors in the determination of the precise onset and nadir of torque development while still representing a majority of the time interval for achieving maximal torque production. ARTD was defined as the average increase in torque generated in unit time (Nm/s), and was calculated over the same interval as $T_{20\text{-}80}$. Hence, ARTD was calculated through numerical differentiation as:

$$ARTD = \frac{1}{N}\sum_{i=1}^{N}\frac{\delta f_i}{\delta t}$$

where, N is the total number of time slots for numerical differentiation, δf_i is the change in torque in the time slot i and δt is the unit time duration for an individual slot. $Torque_{220}$ was defined as the absolute amount of torque generated during the initial 220 ms during a maximal voluntary contraction, and, it is based on the calculated time that is available for concentric torque generation during a typical gait cycle at a speed designated necessary for community ambulation.[13] For example, the speed commonly deemed necessary for persons to safely ambulate in the community is 1.2 m/s.[14] At this speed, the time taken to complete one gait cycle (ie right heel strike to right heel strike) is $\sim$1.1 s. Given that the PF muscles are reported to be active for $\sim$40% of the gait cycle and that approximately 1/2 of this active time is spent generating concentric torque, roughly 220 ms is available for force generation (eg propulsion) by this muscle group.

Voluntary activation deficits
Voluntary activation deficits were determined using the twitch interpolation method.[15,16] Briefly, a single biphasic, supramaximal pulse (600 μs pulse duration) was delivered at rest and during maximal voluntary isometric contraction. Voluntary activation deficit was calculated using the ratio between the torques produced by the superimposition of a supra maximal twitch on a peak isometric contraction (a) and the torque produced by the same stimulus in the potentiated resting muscle (b). Voluntary activation deficits were expressed as: voluntary activation deficit (%) = (a/b)*100.

Locomotor data collection
Subjects performed repeated 10 m walks over a 14 ft long mat (GaitRite) that measures the geometry and the applied pressure of each footfall as a function of time in order to determine both self-selected and maximal overground walking speed (three trials each). In addition to gait speed, the GaitRite system calculates all of the spatio-temporal characteristics of the gait pattern. Gait analyses were performed 3 months before training as well as at both pre- and post-RPT time points. Multiple baseline tests were conducted to control for improvements resulting from natural recovery.

Following pre- and post-RPT overground testing, subjects were placed on a split-belt treadmill (Tecmachine) with custom instrumentation to allow for evaluation of 3-D ground reaction forces. Treadmill speeds for each testing session were set to match those determined overground, and subjects completed three 20-s trials during which kinematic and kinetic data were captured. Post-RPT testing included analysis at velocities matched to pre-RPT self-selected and maximal speeds as well as each subject's new self-selected and maximal speed. Retro-reflective markers were placed at 32 landmarks according to the PlugInGait marker set (Vicon), based on a modified Helen Hayes marker set. 3-D kinematics were captured using a commercially available motion analysis system (Vicon Motion Systems) with eight 1000 Hz cameras. Joint powers were calculated using standard inverse dynamics analyses, as described previously,[13,18] and the time integral of the positive anterior ground reaction force was determined and referred to as propulsive impulse.[17]

Results

Dynamometry
All subjects demonstrated improvements in peak torque production, $T_{20\text{–}80}$, $torque_{220}$ and ARTD during post-*versus* pre-RPT dynamometric testing. On average, RPT resulted in a 35.0 ± 9.1 and 28.9 ± 4.4% improvement in peak isometric torque production in the PF and KE muscle groups, respectively. Individual gains ranged from 17 to 76% in the PF and from 22 to 45% in the KE. Time to peak tension, represented by $T_{20\text{–}80}$, decreased from 470.8 ± 82.2 to 312.0 ± 65.7 ms in the PF and from 324.5 ± 35.4 to 254.2 ± 34.5 ms in the KE muscle groups following training. In addition, both indices of muscle power generation, ARTD and $torque_{220}$, were noticeably improved following training. It is interesting to note that both $torque_{220}$ and ARTD showed more pronounced improvements in the PF compared with the KE muscles with training. Specifically, a 62.1 and 122.2% improvement in $torque_{220}$ and ARTD were seen in the PF muscles, with only a 33.4% improvement in $torque_{220}$ and a 66.4% improvement in ARTD in the KE muscle group. In addition, the largest relative gains in indices of explosive muscle strength ($T_{20\text{–}80}$, $torque_{220}$ and ARTD) occurred in the PF muscle group of the more-involved limb. Peak torque, $torque_{220}$, $T_{20\text{–}80}$ and ARTD data are summarized in Table 1.

Magnetic resonance imaging
On average training resulted in a 14.2 ± 3.8 and 8.3 ± 1.9% increase in max-CSA for the PF and KE muscle groups, respectively. Individual gains in max-CSA are presented in Figure 2. Of note, although we did not perform dynamometric evaluation of the KF strength, a 9.8% increase in max-CSA of the hamstring muscles was realized after training (pre = 42.1 ± 2.1 cm^2; post = 46.1 ± 1.3 cm^2).

Table 1 Pre- and post-RPT isometric torque data for the PF and KE muscle groups

	Pre-RPT					*Post-RPT*				
	Peak torque	*ARTD*	*Torque$_{220}$*	*T_{20-80}*	*Activation deficit (%)*	*Peak torque*	*ARTD*	*Torque$_{220}$*	*T_{20-80}*	*Activation deficit (%)*
Knee extensors										
More-involved										
S1	99.8	282.4	67.5	283.0	39.0	125.7	478.6	88.9	241.6	31.0
S2	100.3	204.6	44.1	440.8	34.0	123.8	497.4	71.0	210.6	25.0
S3	65.1	196.1	28.1	370.4	50.0	81.6	244.5	30.5	280.4	35.0
Less-involved										
S1	136.4	482.1	78.2	254.1	32.0	177.6	706.2	108.5	250.3	29.0
S2	143.9	501.8	69.7	240.7	20.0	176.1	827.3	102.4	300.9	14.0
S3	112.5	330.9	53.8	360.2	19.0	162.7	570.5	54.2	215.5	18.0
	Pre-RPT					*Post-RPT*				
Plantar flexors										
More-involved										
S1	45.4	59.1	13.7	807.3	36.0	56.1	95.7	22.8	587.7	28.0
S2	27.3	50.4	12.1	430.1	42.0	36.1	119.5	27.5	240.1	31.0
S3	17.0	28.6	5.6	490.9	41.0	26.8	102.9	9.5	280.6	28.0
Less-involved										
S1	56.7	105.2	14.4	403.2	18.0	66.4	259.8	26.4	252.2	16.0
S2	32.7	95.0	26.7	380.6	34.0	42.8	164.9	34.4	300.4	15.0
S3	33.2	84.5	12.5	315.9	47.0	58.6	256.1	16.9	215.4	41.0

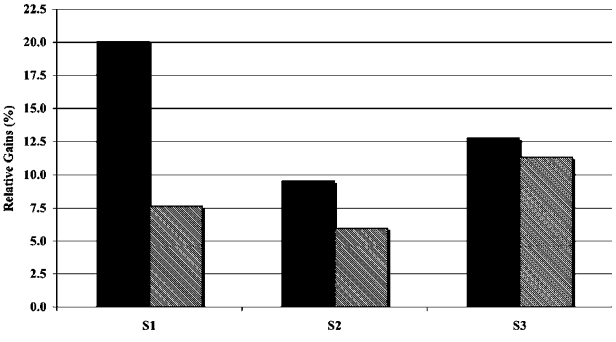

Figure 2 Relative gains in muscle max-CSA in the PF (black bars) and KE (gray bars) muscle groups

Voluntary activation deficits

Significant voluntary activation deficits were noted in both the PF and KE muscle groups before training. RPT resulted in reductions in activation deficits in both the PF and KE muscle groups in each subject. Individual data for activation deficits are presented in Table 1. Although significant bilateral asymmetries existed before and after the intervention, these differences were seemingly attenuated in both muscle groups following RPT.

Locomotor analyses

Values for maximum and self-selected gait speeds did not differ by more than 0.04 and 0.02 m/s, respectively, for any of the subjects in this study when comparing tests carried out 3 months before the onset of training and immediately before training. Following RPT, a 36.1% average increase in maximum gait speed and a 34.7% average improvement in self-selected gait speed were realized. Training-related improvements in maximal and self-selected gait speed were accompanied by increases in step length in both the more-involved and less-involved limbs with little difference in step cadence. Accordingly, little change in percentage time spent in single or double support was noted following RPT. Spatio-temporal characteristics of gait are presented in Table 2. Increases in step length following RPT were accompanied by bilateral increases in both A/P and vertical ground reaction forces (Figure 3) as well as greater bilateral hip excursion. In addition, an improved symmetry in propulsion, represented by % total propulsion, was primarily explained by increased propulsion in the more-involved limb following RPT. Interestingly, increases in peak power at the hip and ankle accompanied increases in gait velocity (Figure 4), with the greatest improvement noted in the more-involved ankle joint. Kinematic and kinetic data are presented in Table 3.

Discussion

The results of this study suggest that a combination of resistance and plyometric training in persons with motor incomplete SCI results in bilateral improvements in (1) peak torque production, (2) time to peak torque and (3) rate of torque production in the PF and KE muscle groups. These improvements in muscle function can be

Table 2 Pre- and post-RPT spatio-temporal characteristics of gait for maximal and self-selected gait speeds

	Self-selected		*Maximum*	
	Pre	*Post*	*Pre*	*Post*
Speed (m/s)	0.77±0.04	1.03±0.14	1.08±0.09	1.47±0.18
Step length (cm)				
More-involved	46.7±6.1	61.8±4.2	52.8±6.7	72.6±6.7
Less-involved	38.8±6.1	57.8±4.2	46.3±6.7	72.1±6.7
% Single support	35.5±3.9	33.7±4.1	40.1±3.2	38.4±3.1
% Double support	28.3±3.6	32.1±2.9	20.5±1.1	23.0±2.5
Cadence (steps/min)	89.0±1.9	95.5±2.6	120.2±3.4	124.3±6.1

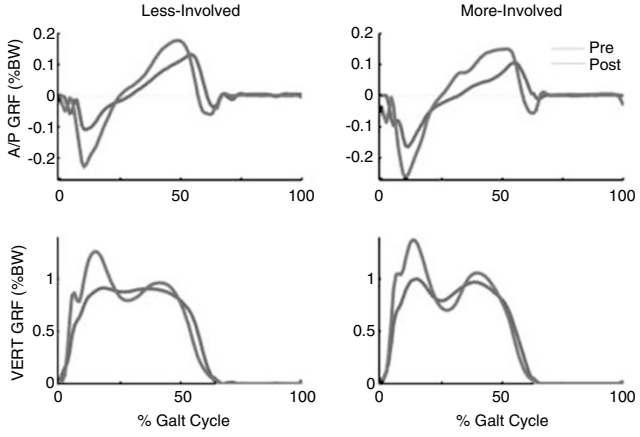

Figure 3 Representative plot of A-P (top) and vertical (bottom) GRF plotted over relative gait cycle (%) for both pre-RPT (blue) and post-RPT (red) time points in the less-involved limbs (left) and more-involved (right) at self-selected gait speed

attributed to both an increase in muscle cross-sectional area and an increased ability to voluntarily activate affected skeletal muscles. Interestingly, the magnitude of improvement in these outcomes was most pronounced in the more- involved *versus* the less-involved limb and in the PF *versus* the KE muscle group. In addition, improvements in both self-selected and maximum gait speeds were realized and were explained by increased propulsion in the more-involved limb as well as increased lower extremity joint powers, suggestive of improved task-specific muscle function (ie during walking).

Injury to both descending spinal pathways as well as decreased activation history have the physiological consequence of reducing the ability to voluntarily activate affected skeletal muscles. Although restoration or repair of the injured spinal cord is not a reasonable expectation with training, the potential to improve deficits resulting from disuse seems likely, and has been demonstrated after periods of inactivity in other populations.[19,20,21] In this study, significant activation deficits that existed before RPT are comparable to other models of disuse (ie cast immobilization, limb suspension).[22] Interestingly, these deficits were partially attenuated with training, and this enhancement of neural function could serve to explain a portion of the strength gains realized post-RPT. In addition to enhanced neural transmission, muscle hypertrophy post-RPT cannot be ignored as a mechanism for improved muscle torque production during both dynamometric testing as well as during walking. However, although significant skeletal muscle hypertrophy (eg larger effector) might suggest improved torque generation independent of the activation pattern, the magnitude of strength gains would suggest that the majority of these gains were accounted for by means other than muscle hypertrophy.

In this study, we chose to examine the morphological and contractile characteristics of the ankle PF and KE muscle groups primarily because of their purported roles during bipedal locomotion. Torque demands at these joints during walking are the two highest in the lower extremity. In addition, we have previously shown that torque generation about these joints is limited in persons after incomplete SCI.[6] Similarly, subjects in the present study presented with reduced PF and KE peak torque values before RPT as well as reduced gait speeds. Interestingly, marked improvements in PF and KE isometric torque generation and gait speed were realized following RPT. However, post-RPT measures of peak torque about these joints as well as maximum gait speeds are still reduced relative to control values,[6] thereby suggesting the potential for further functional improvements if additional increases in torque production can be realized by these muscle groups.

In addition to absolute torque production, a likely mechanism explaining impaired muscle function during locomotor tasks may be an inability to produce properly graded and timed muscle output. This impairment has been identified in this and other populations with central nervous system (CNS) dysfunction[6,23–25] and shown to relate to reduced gait speed.[25] The combination of a prolonged time to peak torque and a decreased ability to generate maximal torque in these persons suggests that at least some of the limitations in gait speed in persons with incomplete SCI might result from impaired muscle function. However, the dramatic improvements in muscle function demonstrated in the present study highlight the potential for this type of training to attenuate existing deficits in neuromuscular function and facilitate functional improvements.

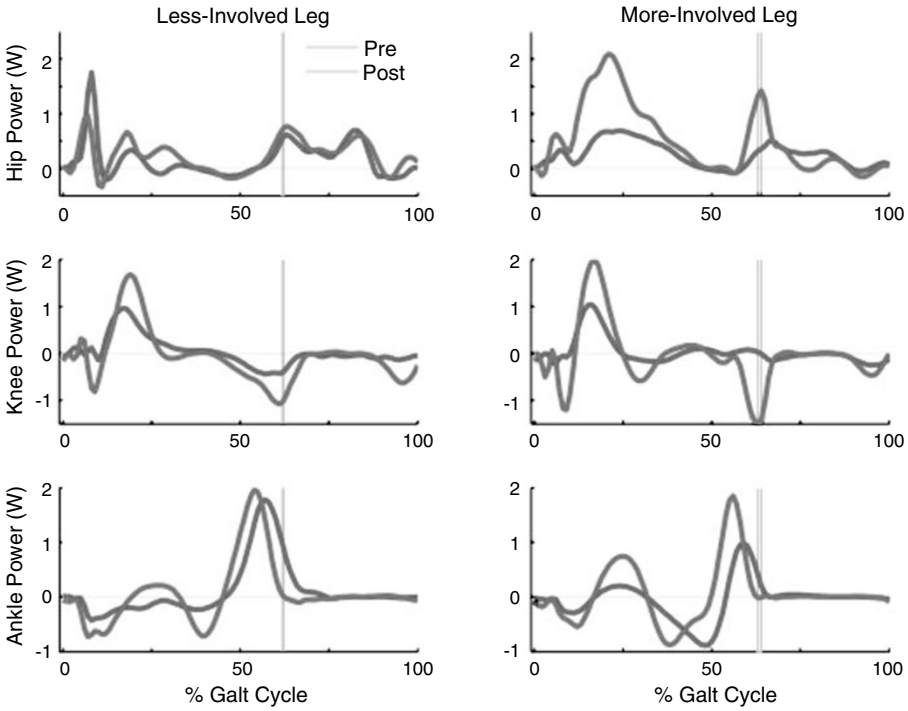

Figure 4 Representative plot of hip (top), knee (middle) and ankle (lower) joint powers for the more-involved and less-involved limbs plotted over relative gait cycle (%) for both pre-RPT (blue) and post-RPT (red) time points at self-selected gait speed

Table 3 Pre- and post-RPT kinetic and kinematic characteristics of gait for a single subject during maximal and self-selected gait speeds

	Pre		*Post*	
	More-involved	*Less-involved*	*More-involved*	*Less-involved*
Hip power (W)	0.51	0.68	0.85	0.97
Ankle power (W)	0.99	1.78	1.85	1.96
Hip excursion (°)	33.72	35.6	57.82	61.85
Peak hip extension (°)	−0.86	0.63	7.7	8.1
Propulsion (Ns/Kg)	150.3	261.5	300.7	293.3
% Total propulsion	36.5	63.5	50.7	49.4

Recent therapeutic interventions examining gait in persons after CNS injury have largely focused on the task specificity of training with little focus on impairment level deficits.[26–28] Although the rationale for task-specific training interventions to result in improvements in motor function is quite strong and shown to be effective in producing cortical reorganization,[29,30] we feel that *in vivo* muscle function is also a limiting factor in these persons, and appropriate training can also induce neuroplastic changes in these tissues, facilitating locomotor improvements by improving the element of muscle function dictated by locomotor task performance. Accordingly, given that few studies have attempted to examine the relationship between lower extremity strength and gait in persons after incomplete SCI, comparisons to other populations with CNS involvement yield valuable information. For example, data examining the relative importance of lower extremity strength in persons after stroke demonstrate significant correlations between the strength of the paretic hip flexors ($r = 0.57$), KE ($r = 0.41$) and primarily the ankle PF ($r = 0.85$), with maximal gait speed.[31,32] In addition, previous simulation work suggests that force production by the soleus and gastrocnemius is critical to trunk forward progression, swing initiation and power generation during gait.[13,18] Thus, one might predict slower gait speeds if force production by these muscles is abnormal during locomotion. Indeed, the negative impact of reduced PF function is supported by experimental data. For example, Lamontagne *et al*[34] suggested that more than 50% of the variance in gait speed in persons post-stroke was explained by the peak activation of the medial gastrocnemius. In addition, Mulroy *et al*[33] demonstrated that ankle moments were substan-

tially reduced in two groups of hemi-paretic persons compared with slow walking controls, with household walkers having reduced moments relative to limited community walkers.[32] These same investigators also found that at two different time points, walking speed was strongly associated with PF voluntary strength. Specifically, deficits in PF strength were pronounced, with the slow subject group (~10% of normal age-matched speed) demonstrating strength equal to ~18% of normal age-matched strength upon admission to rehabilitation. Interestingly, at 6 months post stroke, PF strength increased to 22% of control value, an increase of ~20%, and was associated with increased walking speed (~20%). Thus, these data provide support to suggest a relationship may exist between changes in PF strength and gait speed, at least at slow velocities. Interestingly, the relative gains in PF strength in the present study (35.0%) are almost identical to the increases in fastest (36.1%) and self-selected (34.7%) gait speeds post-RPT.

Improvements in gait speed following RPT reflect a greater capacity for locomotor function. However, the changes in the spatio-temporal and the kinetic measures that accompanied these increases in speed likely illustrate the mechanisms by which gait speed improved following training and suggest means other than compensatory strategies. For example, pre-RPT values for % propulsion demonstrate dramatic bilateral differences that most likely serve to explain the asymmetries present in step length before training. Specifically, propulsion in the less-involved limb was ~75% greater than in the more-involved limb before RPT, thereby contributing to the reduced contra-lateral step length. However, following RPT, propulsion in the more-involved limb improved, resulting in essentially identical values for % propulsion between limbs, and bilateral asymmetries in step length being ameliorated. It is these changes that we suggest are likely to reflect restorative rather than compensatory mechanisms to explain functional improvements.

In conclusion, the importance of the proposed work revolves around the fact that little is known about the extent to which skeletal muscle plasticity may impact functional outcomes after incomplete SCI. The desire 'to be more normal' with respect to locomotor ability is one that many persons after this type of injury possess. Accordingly, the development of appropriate rehabilitation strategies that target improvements in locomotor ability with the goal of increasing functional independence could have a tremendous impact on this population. The data in the present study provide support for the use of physical rehabilitation interventions aimed at attenuating neuromuscular impairments as a means of improving not only gait speed but also the strategies utilized by these persons to ambulate. As such, we suggest that the benefits reported following a combination of resistance and plyometric training represent a first step in the use of these modalities to facilitate the recovery of motor function and functional ability in this population. Although we report significant gains in strength and gait speed following 12 weeks of RPT, at this point we do not know if the subjects in this study reached a plateau in any of the outcomes measured. Therefore, future studies examining the impact of physical rehabilitation training programs after incomplete SCI should focus on the optimal volume (eg duration and frequency) and intensity of training, as well as the potential of this type of training to serve as an adjunctive therapy in the overall treatment of these persons. In addition, these studies need to focus not only on gait but also on other functional outcomes (eg stair climbing, sit to stand) as well as the potential psychosocial benefits (ie community integration) that likely parallel increased functional capacity.

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