“Task-Oriented” Exercise Improves Hamstring Strength and Spastic Reflexes in Chronic Stroke Patients

Gerald V. Smith, PhD, PT; Kenneth H.C. Silver, MD; Andrew P. Goldberg, MD; Richard F. Macko, MD

Background and Purpose—Despite the belief that after cerebral infarction only limited functional gains are possible beyond the subacute period, we tested the hypothesis that a 12-week program of “task-oriented” treadmill exercise would increase muscle strength and decrease spastic reflexes in chronic hemiparetic patients.

Methods—Fourteen subjects, aged 66 ± 3 (mean ± SEM) years, with residual gait deviations due to remote stroke (>6 months), underwent repeated measures of reflexive and volitional (concentric and eccentric) torque with use of isokinetic dynamometry on the hamstring musculature bilaterally. Torque output was measured at 4 angular velocities (30°, 60°, 90°, and 120°/s).

Results—After 3 months of 3 times/wk low-intensity aerobic exercise, there were significant main effects (2 legs [P < 0.01] × 2 times [P < 0.01] × 4 angular velocities [P < 0.05]) for concentric torque production. Torque/time production in the concentric mode also improved significantly in the paretic (50%, P < 0.01) and nonparetic hamstrings (31%, P < 0.01). Eccentric torque/time production increased by 21% (P < 0.01) and 22% (P < 0.01) in the paretic and nonparetic hamstrings, respectively. Passive (reflexive) torque/time generation in the paretic hamstrings decreased by 11% (P < 0.027). Reflexive torque/time was unchanged in the nonparetic hamstrings (P = 0.45).

Conclusions—These findings provide evidence that progressive treadmill aerobic exercise training improves volitional torque and torque/time generation and reduces reflexive torque/time production in the hemiparetic limb. Strength changes associated with improved functional mobility in chronic hemiparetic stroke survivors after treadmill training will be reported in future articles. (Stroke. 1999;30:2112-2118.)

Key Words: exercise • muscles • stroke, chronic

Recent estimates suggest that nearly three quarters of a million Americans annually suffer a stroke, an incidence rate nearly 50% higher than previously calculated. Notwithstanding current rehabilitative efforts, outcomes data consistently indicate that approximately two thirds of stroke survivors have chronic neurological impairments that affect their activities of daily living (ADL), especially their mobility. Principal among these long-term impairments is motor weakness. Residual hemiparesis and spasticity enhance fall risk, increase energy expenditure during gait, and ultimately foster activity intolerance in these individuals. Collectively, the functional consequences associated with long-term motor deficits predispose the great majority of stroke survivors to a generally sedentary lifestyle with concomitant deconditioning, disuse atrophy, and sarcopenia, thereby contributing to further declines in ADL function and disability status.

Accordingly, effective and cost-efficient therapeutic interventions are needed to help promote cardiovascular fitness, improve endurance, and minimize chronic neuromuscular impairments due to disease or disuse in stroke survivors beyond the subacute poststroke rehabilitation period. Despite strong interest and considerable research, optimal modalities for promoting longitudinal fitness and functional mobility during the acute and subacute phases of stroke remain elusive. At this time, even less is known with respect to optimal exercise interventions in the chronic phase of stroke. The present investigation was designed as part of an ongoing series of studies to begin to address this gap in our knowledge.

Currently, most stroke survivors are thought to have achieved their maximum functional recovery by means of standard rehabilitative therapies within a 3-month epoch after the stroke onset. During this relatively brief period, the patient normally is offered intensive rehabilitative care, after which they typically are discharged to routine medical follow-up with few or no empirically based recommendations for additional therapeutic interventions to promote cardiovascular fitness and optimize neuromuscular function. Conse-
Exercise improves chronic stroke patients' strength and spasticity

Smith et al

Representative hamstring torque curves: reflexive (R; ), concentric (C; ), and eccentric (E; ) hamstring torque curves in a patient’s paretic limb at 60°/s angular velocity. The mean torque (in newton-meters) recorded between 35° to 45° of flexion (dashed lines) for each type of contraction was used for statistical analysis. Movement direction for each exercise mode is indicated by arrows. Each data point represents the mean of 3 trials recorded at 0.5° intervals.

Subjects and Methods

Subjects

Fourteen patients, 12 men and 2 women, aged 66±3 years (mean±SEM; range 58 to 85 years), with mild to moderate hemiparesis and persistent gait deviations due to remote stroke (>6 months; mean 19 months after infarction; range 7 months to 6.7 years) were recruited for this pilot/feasibility study from Veterans Affairs (VA)-Maryland Health Care System and the University of Maryland Medical System (UMMS) clinics. Informed consent approved by the joint VA/University of Maryland/UMMS Institutional Review Board (IRB) was obtained from all patients before their inclusion in the study. Eleven of the patients were black and 3 were white. Seven of the patients had a left hemispheric stroke, 6 had an infarction in the right cerebral hemisphere, and 1 had bilateral cerebral damage. The latter patient, however, manifested only unilateral gait deviations on neurological testing. Gait deviations were defined operationally as any readily observable asymmetries during ambulation, especially altered stance and swing cycles secondary to “hip-hiking,” hip circumbduction, genu recurvatum, and/or “foot drop.” All patients could ambulate independently in their home environment, but needed some form of assistance in the community.

At the time of recruitment, all patients had completed conventional rehabilitative training well before entry and were therefore considered to be “plateaued” in their neurological recovery. All patients received a complete baseline medical and neurological evaluation, which included a customized cardiac treadmill exercise test.11 Subject exclusion criteria included unstable angina pectoris, congestive heart failure, peripheral arterial disease (ankle/brachial index of <0.97)12; significant clinical depression (Center for Epidemiological Studies–Depression scale score of >16)13; moderate to severe dementia (Mini-Mental Status Examination score of <21)14; language deficits (ie, sensory aphasia) that precluded following simple commands consistently; painful orthopedic conditions involving the pelvis, hips, knees, or ankles (ie, severe osteoarthritis); or other preexisting disorders that could limit successful participation in the study. Patients already participating in regular exercise, regardless of type or intensity, were also excluded to rule out prior aerobic training effects.

Exercise Protocol

The exercise intervention consisted of a modified low-intensity cardiac rehabilitation program.10,11 Patients walked on a motorized treadmill 3 times/wk for 3 months using handrail support ad libitum. The training intensity was limited at the beginning of the study to 40% of the calculated heart rate reserve (HRR) and increased as tolerated to targeted levels. Patients were progressed over the course of the training program to 40 minutes of continuous exercise at 60% to 70% of their previously calculated HRR. Five-minute warm-up and cool-down periods at 30% of HRR were included in each training session.

Testing Protocol

Repeated measures of reflexive (passive) and volitional (concentric and eccentric) torque were obtained from the hamstring musculature bilaterally by use of isokinetic dynamometry (Kin-Com 125AP, Chattex Group, Inc). Torque generation was measured at 4 angular velocities (30°, 60°, 90°, and 120°) over 60° of knee flexion/extension. The testing protocol was modified from the earlier work of Knutsson and Martensson,15 which established a quantitative methodology to measure spasticity. The aforementioned angular velocities were chosen because they represent a range of movement speeds at which many common ADL occur. Additionally, using Lance’s widely accepted definition of spasticity as being velocity-dependent reflexive phenomena,16 the passive torque produced at these velocities was deemed likely to produce a characteristic upwardly sloped trendline in the presence of heightened spinal reflex activity. The dynamometer was calibrated according to the manufacturer’s guidelines. The same investigator (G.V.S.) conducted all testing.

Patients were positioned by means of a standard seating protocol that was manually modified to ensure that the dynamometer’s lever arm axis of rotation and the knee joint’s axis were aligned and that the force transducer was positioned appropriately on the distal tibia.17 Once optimal positioning and alignment of the subject had been achieved, data reflective of these parameters were stored in the computer for replication before subsequent testing. As part of the set-up procedures, a gravity compensation algorithm was calculated for each patient before each testing session.18 Data recording was made at half-degree intervals between 70° of knee flexion and –10° of knee extension. The latter parameters were chosen because the beginning and ending ranges were achievable by all patients, they minimized the potential for knee injury during terminal extension, and they included acceleration and deceleration phases sufficient to allow the Kin-Com to achieve and maintain the preset steady-state angular velocity in the midrange of the movement. During testing, patients were given verbal instructions before each change in procedure, encouraged verbally during each trial, and allowed to observe in real-time the force curves produced during each test replicate. These procedures were followed to ensure high levels of patient understanding and cooperation. Three trials at each angular velocity were recorded passively, followed by 3 trials of concentric contractions and then 3 trials of eccentric contractions. The order of testing was the same for all patients, but the testing order for the limbs was randomized.

Data Reduction and Analysis

Mean passive (reflexive), concentric and eccentric torque was determined between 35° to 45° of knee flexion from the dynamometer’s computer records in the paretic and nonparetic limbs, respec-
tively (Figure). This interval, roughly midway in the tested range of motion, was chosen on the basis of a preliminary analysis that demonstrated consistently high levels of reliability in maintaining the desired angular velocities within this range (data not shown). Moreover, we felt a determination of the mean torque and the mean torque per unit of time (T/T) generated within a fixed locus in the range of motion that more or less corresponds to the muscle’s optimal force generating length19 rather than the peak torque would permit a higher degree of comparability in the data analysis within each exercise mode over time. Nevertheless, as illustrated in the Figure, the reported measures do not necessarily reflect or include the peak torque achieved under any of the test conditions.

Data analysis includes descriptive statistics expressed as the mean±SEM at each angular velocity. These values were calculated from the means of the 3 trials recorded over the 10° arc for each patient under each test condition. Thus, the data reported represent the mean of the 14 subjects, not the mean of the 42 individual test replicates recorded for each test condition. To ascertain the main effects of the exercise intervention on the torque generation capacity within the specified range of motion, the data for each type of muscle contraction were subjected to a ANOVA test—ie, 2 (limbs)×3 (times)×4 (angular velocities) design—with use of a commercially available statistical software package (BMDP version 7.0, BMDP Statistical Software, Inc).

A secondary statistical analysis designed to begin to tease apart the underlying neurophysiological effects of the exercise intervention was also performed. To do so, we examined the rate of torque production in each limb as an indirect indicator of spinal motor unit recruitment and the sustainability of the force output. T/T was calculated as the ratio of the mean torque (in Newton meters) divided by the time (in seconds) required for the limb to traverse the 10° arc in the range of motion at each angular velocity. The new variable, T/T (nm/s), created by this operation was subjected to a univariate analysis (paired t test). As a follow-up, we also assessed the clinical impact (or percent change [∆]) in the T/T output as a consequence of the intervention. The overall ∆ value was calculated with the following equation using mean T/T data:

\[(T/T_{pre} - T/T_{post})/(T/T_{pre}) \times 100\]

where T/T_pre=baseline T/T value and T/T_post=follow-up T/T value.

To assess differences in baseline and follow-up T/T values between limbs, an indicator of the interlimb symmetry in torque generating capacity, the mean T/T data for all subjects under each exercise condition was converted using the following formula to give a percent:

\[(A/NA) \times 100\]

where A=affected limb T/T value and NA=Nonaffected limb T/T value.

Statistical significance was accepted for all tests at an alpha level of P<0.05.20 The results are presented below.

**Results**

**Graphic Representation**

The Figure displays the mean torque curves produced by 1 patient at 60°/s angular velocity before the exercise intervention. Arrows beside each torque curve indicate the direction of movement. The dashed lines delineate that portion of the joint range of motion from which the mean of 3 test replicates for each exercise mode was calculated for each patient. As indicated above, this region was selected because it represents a theoretically optimum region along the muscle’s length-tension curve. However, as illustrated in the figure, this region does not necessarily match the peak torque output measured for any exercise mode.

**Major Findings**

With the exception of the reflexive torque values, the absolute decline in torque values expected as the angular velocity increased was observed in both limbs at baseline and after the exercise intervention. Mean baseline and follow-up hamstring torque measures for each angular velocity under each exercise condition are presented in Table 1. The multifactorial ANOVA revealed significant improvements in concentric hamstring torque generation as a function of limbs (ie, affected<nonaffected; P<0.01), times (ie, pretest<posttest; P<0.01), and angular velocities (ie, 30°>60°>90°>120°/s; P<0.05). Eccentric torque production was significantly different as a function of the limb tested (affected<nonaffected; P<0.01) and angular velocity (30°>60°>90°>120°/s; P<0.01), but it did not reach significance as a function of time (affected=nonaffected; P=0.28). Reflexive torque generation was significantly different as a function of the limb tested (affected > nonaffected; P<0.05). Changes in the reflexive torque measures did not reach significance as a function of angular velocity (P=0.15) or time (P=0.64) in this small sample when the 2×2×4 statistical design was used. However, univariate test results with the transformed T/T data rather than the raw torque output measures indicated a significant decline over time in passive T/T generation in the affected limb (P<0.05) but not the nonaffected limb (P=0.45). Calculations of the change in interlimb symmetry (Δs) after exercise with the T/T data demonstrated a 12% decline overall in the reflexive T/T measures, a 15% increase in the concentric T/T values, and a 10% increase in eccentric T/T aggregate (Table 2).

**T/T Measures in the Affected Hamstrings**

Univariate tests indicated the within limb changes in the paretic limb in T/T production for each exercise mode was significant as a result of the intervention. For example, there was an 11% decline (P<0.05) in the reflexive T/T measure. The within-limb concentric T/T measure increased significantly (50%, P<0.01) in the more affected limb after the 3-month treadmill exercise intervention. Likewise, the within-limb eccentric T/T measure increased substantially (21% overall) on the more affected side (P<0.01; Table 2).

**T/T Measures in the Nonaffected Hamstrings**

Concentric and eccentric T/T production increased significantly in the nonaffected limb, by 31% (P<0.01) and 22% (P<0.01), respectively. Interestingly, while the relative gains in eccentric T/T production were roughly equivalent for each limb (21% versus 22%), the concentric T/T measures were very different (50% versus 32%) for the paretic and nonparetic limbs, respectively. The reflexive T/T measure did not vary over time in the nonaffected limb as a result of the exercise intervention (P=0.45; Table 2).

**Between-Limb Normalization of the T/T Values**

Relative to the nonaffected limb, the hamstring muscle group on the affected side showed substantial improvement in both volitional and reflexive T/T production as a result of the training protocol. The reflexive T/T symmetry measure declined by 12% after 3 months of progressive treadmill...
exercise. The concentric torque measures showed an improvement of 15% overall. The normalized eccentric T/T measure was essentially unchanged (↓ 0.6%; Table 2).

**Discussion**

We have reported previously the parameters by which treadmill exercise could be prescribed safely for the poststroke population.11 We have also demonstrated heretofore that the energy costs and cardiovascular demands of hemiparetic gait decline as a consequence of our progressive treadmill training protocol.10 The present investigation describes the effects of 3 months of “task-oriented” treadmill exercise on reflexive (passive) and volitional (concentric and eccentric) torque and T/T production, basic indices of strength and spasticity in the hamstrings muscle groups bilaterally. We show herein that 12 weeks of regular treadmill training increases volitional torque production and T/T output bilaterally and decreases reflexive T/T generation in the affected hamstring muscles of chronic hemiparetic stroke patients. Additionally, in preliminary analyses, the measured improvements in strength correlate in our test population with enhanced mobility and functional activity on conventional clinical measures of physical performance (ie, velocity-matched overground and treadmill walks, a modified “get-up and go” test, and in recovery from dynamic postural perturbation).21–23 (The abstracts cited have been presented at national or international conferences and will be described more fully in future publications.)

The strength changes noted in this study are remarkable for several reasons. First, significant muscle strengthening is not normally expected as an outcome of AEX training,24,25 or at best is relatively modest in severely deconditioned older patients.24,26 Second, and perhaps more importantly, longitudinal stroke studies have indicated that the recovery of motor function, particularly ambulatory capacity, “plateaus” roughly 90 days after cerebral infarction.9 In the present investigation, the participants were on average nearly 2 years poststroke, a time frame well beyond the currently accepted window of spontaneous neural recovery. Moreover, they had been discharged from all conventional rehabilitation therapies well before their participation in this study, which indicates from a standard clinical perspective that they had already achieved their maximum motor recovery by means of established therapeutic approaches. Furthermore, there is no evidence in the rehabilitation literature, to our knowledge, that spastic reflexes are modifiable by exercise in the chronic phase of stroke or can be altered to a meaningful degree at any time after stroke as a consequence of ambulatory exercise alone. Thus, contrary to prevailing dogma, the present data constitute a clear-cut finding that survivors of ischemic stroke can achieve significant improvements in both volitional torque and T/T production and show similar reductions in spastic reflexes with use of AEX training modalities.

It is quite possible that the substantial gains in volitional strength observed in this study merely reflect contumaciously debilitated patients overcoming the physical deconditioning

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**TABLE 1. Mean Hamstring Torque in the Affected and Nonaffected Limbs by Exercise Mode**

<table>
<thead>
<tr>
<th>Exercise Mode</th>
<th>Mean Torque Output, Newton meters</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30°/s</td>
</tr>
<tr>
<td>Reflexive contraction</td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td></td>
</tr>
<tr>
<td>Affected</td>
<td>28.9 ± 2.9</td>
</tr>
<tr>
<td>Nonaffected</td>
<td>28.5 ± 2.5</td>
</tr>
<tr>
<td>3-month follow-up</td>
<td></td>
</tr>
<tr>
<td>Affected</td>
<td>25.8 ± 2.3</td>
</tr>
<tr>
<td>Nonaffected</td>
<td>27.7 ± 2.8</td>
</tr>
<tr>
<td>Concentric contraction</td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td></td>
</tr>
<tr>
<td>Affected</td>
<td>61.6 ± 11.9</td>
</tr>
<tr>
<td>Nonaffected</td>
<td>109.9 ± 17.1</td>
</tr>
<tr>
<td>3-month follow-up</td>
<td></td>
</tr>
<tr>
<td>Affected</td>
<td>98.7 ± 22.9</td>
</tr>
<tr>
<td>Nonaffected</td>
<td>109.6 ± 17.2</td>
</tr>
<tr>
<td>Eccentric contraction</td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td></td>
</tr>
<tr>
<td>Affected</td>
<td>72.7 ± 10.1</td>
</tr>
<tr>
<td>Nonaffected</td>
<td>133.4 ± 20.6</td>
</tr>
<tr>
<td>3-month follow-up</td>
<td></td>
</tr>
<tr>
<td>Affected</td>
<td>97.8 ± 14.1</td>
</tr>
<tr>
<td>Nonaffected</td>
<td>155.4 ± 16.4</td>
</tr>
</tbody>
</table>

Data are mean±SEM for all patients at each angular velocity for each exercise condition.
associated with a sedentary habit. Assuming this intuitively sensible explanation were correct, the present results, viewed in isolation, would represent an important clinical finding, because they indicate that the deleterious effects of long-standing physical deconditioning can be reversed or modified considerably by regular AEX even in patients with chronic hemiparesis. Indeed, on the basis of these results, our previous findings, and the cardiac rehabilitation literature taken as a whole, it may be reasonable to hypothesize that it might even be possible to alter the stroke survivor’s long-term risks for future brain or heart attacks by vigorously promoting the regular use of this training modality. However, while this latter supposition may essentially be correct, in our opinion, a rigorous interpretation of the data on which it is based does not account fully for the greater relative (but not absolute) magnitude of the improvements observed in the concentric mode in the more-affected limb compared with the less-affected limb. Nor does a reversal of physical deconditioning adequately reconcile the substantial reduction in the reflexive T/T, an indirect measure of spasticity, seen in the affected limb. To illustrate the first point, in comparison with the present findings, in patients diagnosed with another disease known to limit mobility and ADLs secondary to generalized debility and aerobic impairment, chronic obstructive pulmonary disease, 1 recent report revealed a far more modest, albeit significant \((P<0.05)\), improvement in lower-extremity muscle strength after individualized exercise training at the patient’s gas exchange threshold.\(^{27}\) In this study, a 3-week program of progressive resistive exercises, specifically designed to enhance muscle strength, endurance, and symptom-limited \(\dot{V}O_2\) consumption, produced more limited strength gains (8%) in the patients with chronic obstructive pulmonary disease than those seen in our stroke patients (50% and 31% for the impaired and nonimpaired limbs, respectively). Thus, while both of these populations were functionally impaired aerobically and presumably weaker than a nonsedentary age-matched cohort, the stroke patients in the current study made relatively greater gains overall as a result of an exercise program not intended (or expected) to produce strengthening. Although there were substantial differences in the length (3 versus 12 weeks) and focus of the training (aerobic versus progressive resistive exercises) programs that may account for some of the disparity in the training effects, it could also be hypothesized that some mechanism(s) other than a simple reversal of deconditioning might account for at least part of the strength changes observed in the present study.

Although several alternative explanations may be offered to account for the exaggerated motor gains noted on the stroke-affected side, we are of the opinion, based on a rapidly growing basic science literature,\(^{28,29}\) that all or a significant part of these anomalous findings may be due to cortical sensorimotor plasticity in response to the highly repetitive nature of the training paradigm. Although still somewhat speculative, a case could be made that treadmill walking “forces the use” of the impaired limb in a biomechanically more normal way,\(^{30}\) thereby providing undamaged cortical circuits at or near the site of the lesion with sufficient somatosensory stimuli to promote local terminal sprouting and/or functional synaptic reorganization.\(^{31,32}\) Assuming this conceptual framework is substantively correct, it should be possible to demonstrate by means of modern neuroimaging\(^{33}\) or brain-mapping\(^{34}\) techniques whether and to what extent neocortical changes are induced through “task-oriented” exercise. Ongoing collaborative studies involving members of our research team are addressing this exciting and important prospect.

Although the suggested “neuroplasticity” model may be debatable in its details or appropriateness, there should be no serious disagreement that the ability of cortical motor areas to recruit spinal motor units in a well-coordinated and timely

### TABLE 2. Changes in Hamstring Torque/Time Measures by Limb Side After Regular Aerobic Exercise

<table>
<thead>
<tr>
<th>Limb Side</th>
<th>Mean T/T, Newton meters/s</th>
<th>Baseline</th>
<th>Follow-Up</th>
<th>Within-Limb</th>
<th>(\Delta)</th>
<th>(\alpha) Value</th>
<th>Between-Limb</th>
<th>(\Delta)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reflexive T/T production</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affected</td>
<td>38.6±6.7</td>
<td>34.3±5.5</td>
<td>-11%</td>
<td>0.027*</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonaffected</td>
<td>37.7±5.2</td>
<td>37.9±5.8</td>
<td>+0.7%</td>
<td>0.45</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normalized value†</td>
<td>102.4%</td>
<td>90.5%</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>-12%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Concentric T/T production</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Affected</td>
<td>34.7±7.8</td>
<td>52.2±10.7</td>
<td>+50%</td>
<td>0.002*</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonaffected</td>
<td>88.3±19.3</td>
<td>115.5±20.5</td>
<td>+31%</td>
<td>0.001*</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normalized value†</td>
<td>39.3%</td>
<td>45.2%</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>+15%</td>
<td></td>
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<tr>
<td>Eccentric T/T production</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Affected</td>
<td>80.1±12.6</td>
<td>97±15.5</td>
<td>+21%</td>
<td>0.002*</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonaffected</td>
<td>121.9±23.2</td>
<td>148.5±26.2</td>
<td>+22%</td>
<td>0.001*</td>
<td>...</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normalized value†</td>
<td>65.7%</td>
<td>65.3%</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>-0.6%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Significant improvements (indicated by asterisk) were found in both limbs under each exercise mode, with the exception of the reflexive torque in the nonaffected limb. Within- and between-limb %\(\Delta\) were calculated for each condition.

†Normalized value = (affected/nonaffected)\times100.
manner underlies effective synergistic muscle contraction, concomitant force production, and ultimately purposeful movement. Accordingly, the posttraining trends toward greater torque and T/T output, as well as the normalization of the affected/nonaffected limb T/T output ratio, might be taken as an indirect marker of improved spinal motor unit recruitment that is consistent with the hypothesis of central neural reorganization. The improvements noted in the patient’s performance of functional activities would also attest to this construct. However, it also remains possible that exercise-mediated adaptations at the level of the muscle itself, such as a reversal of disuse atrophy in the affected limb, could potentially account for part of the more robust relative gains in motor output observed in the affected limb. Thus, the basic question of whether the improvements identified in this study are due to central or peripheral neuromuscular adaptations remains largely unanswered. Bearing in mind the invasiveness of the procedure, the latter possibility could (and probably should) be explored by serial muscle biopsies to determine what morphological changes, if any, occur in paretic and nonparetic muscles as a result of regular “task-oriented” AEX.

In summary, we report that 3 months of treadmill AEX produces significant gains in strength and reduced reflexive T/T production in the hamstring muscles of chronic hemiparetic stroke patients. These findings indicate that regular “task-oriented” treadmill training has beneficial effects on voluntary torque and T/T output and spasticity. These effects, moreover, appear to carry over to locomotor ability and other measures of functional performance. The present findings also suggest that conventional precepts regarding the windows of motor recovery following stroke may be too narrow in their scope. And, while we are persuaded that AEX training in a “task-oriented” treadmill paradigm can be a useful adjunct to traditional therapeutic interventions in stroke survivors, the timing, intensity, and duration of the training, as well as its long-term effects on other areas of physical performance, require further investigation in more rigorous randomized controlled studies.

Acknowledgments

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