Effects of 12-wk eccentric calf muscle training on muscle-tendon glucose uptake and SEMG in patients with chronic Achilles tendon pain

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Masood T, Kalliokoski K, Magnusson SP, Bojesen-Møller J, Finni T. Effects of 12-wk eccentric calf muscle training on muscle-tendon glucose uptake and SEMG in patients with chronic Achilles tendon pain. J Appl Physiol 117: 105–111, 2014. First published May 22, 2014; doi:10.1152/japplphysiol.00113.2014.—High-load eccentric exercises have been a key component in the conservative management of chronic Achilles tendinopathy. This study investigated the effects of a 12-wk progressive, home-based eccentric rehabilitation program on ankle plantar flexors’ glucose uptake (GU) and myoelectric activity and Achilles tendon GU. A longitudinal study design with control (n = 10) and patient (n = 10) groups was used. Surface electromyography (SEMG) from four ankle plantar flexors and GU from the same muscles and the Achilles tendon were measured during submaximal intermittent isometric plantar flexion task. The results indicated that the symptomatic leg was weaker (P < 0.05) than the asymptomatic leg at baseline, but improved (P < 0.001) with eccentric rehabilitation. Additionally, the rehabilitation resulted in greater GU in both soleus (P < 0.01) and lateral gastrocnemius (P < 0.001) in the symptomatic leg, while the asymptomatic leg displayed higher uptake for medial gastrocnemius and flexor hallucis longus (P < 0.05). While both patient legs had higher tendon GU than the controls (P < 0.05), there was no rehabilitation effect on the tendon GU. Concerning SEMG, at baseline, soleus showed more relative activity in the symptomatic leg compared with both the asymptomatic and control legs (P < 0.05), probably reflecting an effort to compensate for the decreased force potential. The rehabilitation resulted in greater SEMG activity in the lateral gastrocnemius (P < 0.01) of the symptomatic leg with no other within- or between-group differences. Eccentric rehabilitation was effective in decreasing subjective severity of Achilles tendinopathy. It also resulted in redistribution of relative electrical activity, but not metabolic activity, within the triceps surae muscle.

postron emission tomography; eccentric exercise; biomechanics; Achilles tendon; tendinopathy

ACHILLES TENDINOPATHY IMPAIRS calf myotendinous unit function and affects muscle activation strategies within the triceps surae (TS) muscle (10, 22, 33, 38). Generally, chronic Achilles tendinopathy is not accompanied by tendon inflammation (2, 24). Since Achilles tendinopathy leads to higher cell count (28), theoretically it can result in higher metabolic activity in the tendinous tissue. Tendinosis is also shown to be associated with neovascularization of the Achilles tendon (AT), which might be the potential source of tendon pain (25, 31, 26). Victorian Institute of Sports Assessment-Achilles (VISA-A) questionnaire is widely used to assess the severity of Achilles tendinopathy, with a maximum score of 100 signifying a normal tendon (29).

High-load eccentric exercise has been an integral part of conservative management of Achilles tendinopathy over the past few decades, although the precise rehabilitating mechanism remains unclear. Proposed mechanisms, however, are tendon hypertrophy, alterations in tendon mechanical properties (1, 21), lengthening of calf-muscle-tendon unit (1), reversal of the neovascularization (25a), and distinctive neural commands (5). Eccentric training has been shown to alter the AT’s biomechanical properties in both animals (19) and humans (8, 27, 32, 35, 39). Furthermore, there is evidence to suggest that eccentric exercise is more effective in treating the midportion tendinopathy than the insertional tendinopathy (6), thus necessitating protocol modification in the insertional-type cases (16).

Previous studies have evaluated the effects of eccentric training on Achilles tendinopathy using methods such as ultrasonography and power Doppler (25a), magnetic resonance imaging (MRI) (32), and isokinetic muscle testing (39). Although conventional bipolar surface electromyography (SEMG) has been widely used to measure the electrical activity of skeletal muscles during various physical activities (9, 12), the information it provides may not reflect activation of the whole muscle (14, 20). To noninvasively study whole muscle activation, the use of positron emission tomography (PET) is one alternative (11, 18, 36) that may reveal new aspects related to functional restoration of muscles.

Therefore, the purpose of this study was to investigate the effects of eccentric calf muscle-tendon rehabilitation on skeletal muscle-tendon glucose uptake (GU) and myoelectric activity patterns of various plantar flexors in unilateral chronic Achilles tendinopathy patients. It was hypothesized that eccentric rehabilitation will improve plantar flexion strength and, consequently, GU during submaximal isometric exercise in the TS muscle of the symptomatic (Pain) leg. We further hypothesized that the relative contributions of ankle plantar flexors will change as a result of eccentric rehabilitation in the Pain leg.

MATERIALS AND METHODS

Study Design

A longitudinal study design with a patient group and a control group (CTRL) was employed. The patient group went through a 12-wk eccentric rehabilitation program and was tested both before and after the intervention. However, the CTRL group was tested only once, to limit their radiation exposure, and did not undertake the exercise program.
Subjects

Achilles tendinopathy group. The inclusion criteria comprised ages ranging from 18 to 35 yr, unilateral AT pain for at least the previous 6 wk, and absence of any other major musculoskeletal or systemic disorder. All patients had been diagnosed by a clinician with a history of treatment with drugs, including anti-inflammatory agents. Most of the patients had also tried other physical rehabilitation regimes. However, none of the patients was taking any medications or trying other rehabilitation programs during his or her participation in the study. The mean age (±SD) of the 10 patients (7 men and 3 women), recruited through public advertisements, was 27.9 ± 4 y, height 175.4 ± 5 cm, and body mass 66.5 ± 5 kg. Five of the patients had symptoms in the right AT, whereas the other five had them in the left leg. Average duration of the symptoms at baseline measurements was 10.4 ± 8 mo (range: 2–25 mo).

Control group. Ten healthy volunteers (7 men and 3 women) with no history of recent musculoskeletal or systemic disorder with matched (±5%) demographics were recruited: 27.6 ± 4.1 yr, 174.3 ± 3.1 cm, and 68.7 ± 6.6 kg.

Approval for the study protocol was issued by the Ethics Committee of the Hospital District of South-Western Finland. All subjects gave informed, written consent before the study, which was carried out in accordance with the Declaration of Helsinki.

Study Protocol

The measurements were carried out at the Turku PET Centre, University of Turku, Finland, and followed the same protocol as used earlier (23). Each subject completed all measurements on a single day and was required to fast for at least 8 h before the PET scans. The measurements were repeated for the patient group after the 12-wk intervention period.

Subject preparation started with shaving, abrading, and cleaning of the skin for SEMG, placement of electrodes on both legs, and securing of an electronic goniometer to the ankle. Additionally, catheters were introduced into the antecubital veins of both arms: one each for [18F]fluorodeoxyglucose tracer injection and venous blood sampling. Afterward, the subjects were positioned in the exercise apparatus for carrying out the isometric exercise protocol. Subjects were allowed to familiarize themselves with the equipment and the plantar flexion task through a set of submaximal contractions from each leg. Maximal voluntary contraction (MVC) force of ankle plantar flexors was recorded for both legs alternately. The highest of the three MVC trials (second-order Butterworth filter, 12 dB/octave, cutoff frequency: 10 Hz) to correct DC offset and minimize noise signal. Root mean square (RMS) amplitude for each muscle was obtained from the middle 3-s epoch of a submaximal isometric contraction and was normalized to the MVC RMS amplitude.

Plantar flexion force. An in-house custom-built portable force transducer (University of Jyväskylä, Finland) was used to register isometric ankle plantar flexion force under maximal and submaximal conditions. The transducer plate was secured to the seat-back with steel chains to create a rigid frame. Force data was recorded with Signal 4.0 (Cambridge Electronic Design) in sync with SEMG. Data were analyzed to obtain average maximal and submaximal ankle plantar flexion force.

Musculoskeletal Imaging and Analyses

PET. A CTI-Siemens ECAT EXACT HR+ (Siemens, Knoxville, TN) PET scanner was used to image the legs in four adjacent regions from toes to the upper thigh with the subject lying supine. Radioactive markers were taped on the lateral malleoli and medial femoral condyles of both legs to enable later merger of MRI and PET images. The emission scans to measure the amount of the tracer taken up and transmission scans for the correction of tissue attenuation, along with the transition time between the regions, lasted ~32 min.

After correcting the PET images for decay and tissue attenuation, parameter fractional uptake rate (FUR) images were computed using the corrected PET image data and the individual input function (plasma radioactivity data). Transverse plane FUR images were used for regions of interest (ROIs) drawings encompassing whole individual muscle using Carimas 2.0 software (Turku PET Centre, University of Turku, Finland). ROIs were marked, at every centimeter of muscle and AT thickness, by the same investigator (TM) to avoid interrater differences. Subsequently, the FUR values for SOL, MG, LG, FHL, and the AT were further converted to GU rate values with the following formula:

$$\text{Glucose uptake (µmol·100 g−1·min−1)} = \frac{\text{FUR} \times \text{Plasma glucose}}{\text{Lumped constant} \times \text{Tissue density}}$$

where plasma glucose level came from the plasma sampling, lumped constant (1.2) based on an earlier study (26a), and tissue density from Report of the Task Group on Reference Man (34).

MRI. Bilateral MRI scanning was carried out with 1.5 T Philips Intera MRI (Philips Healthcare, Eindhoven, The Netherlands). Lipid pills were secured, for anatomical reference, to the same anatomical landmarks as used in the PET scanning. MRI images only served as anatomical references to delineate the investigated muscles during the drawing of ROIs on the PET images.

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SEMG and Force Data

Electromyographic data were recorded using conventional bipolar SEMG technique from both legs. Silver-silver chloride electrodes (Ambu BlueSensor N, Ambu A/S, Ballerup, Denmark) were placed on the skin over soleus (SOL), medial gastrocnemius (MG), and lateral gastrocnemius (LG) muscles according to the SENIAM (Surface Electromyography for the Non-Invasive Assessment of Muscles) recommendations (13) (interelectrode distance: 22 mm). For flexor hallucis longus (FHL), 16-mm apart electrodes were placed after locating the muscle behind the medial malleolus by manual palpation (4, 23). Furthermore, an indifferent electrode was positioned on the right medial malleolus. SEMG data were recorded online via EISA (bandwidth 10 Hz to 1 kHz per 3 dB), a 10-channel EMG detection system (model: 16-2, University of Freiburg, Germany) at a measurement frequency of 1,000 Hz. The signal was preamplified with a factor of 200 via an integrated cable preamplifier. Digital conversion of analog SEMG and force data was done with Power 1401 (Cambridge Electronic Design, Cambridge, UK). Compatible Signal 4.0 (Cambridge Electronic Design) software was used to record, reduce, and analyze the myoelectric signals from the beginning, middle, and end of the isometric exercise.

SEMG data differentiation was accomplished by high-pass filtering (second-order Butterworth filter, 12 dB/octave, cutoff frequency: 10 Hz) to correct DC offset and minimize noise signal. Root mean square (RMS) amplitude for each muscle was obtained from the middle 3-s epoch of a submaximal isometric contraction and was normalized to the MVC RMS amplitude.

Isometric exercise protocol. The task was performed while sitting, with ankle in neutral position, knee in full extension, and hip in 90° of flexion, on a seat placed on the floor. Each set of the exercise protocol included five unilateral, voluntary isometric plantar flexion contractions at 30% MVC. Each contraction lasted for 5 s followed by a 5-s rest. Real-time visual feedback, regarding both the target force level and subject’s attempt, was provided on a monitor display. After two sets of warm-up contractions for both legs, 150 MBq of [18F]fluorodeoxyglucose tracer were infused. The infusion was followed by the performance of further 10 sets of the plantar flexion task while alternating between legs after each set. Thus the total exercise and rest time, including warm ups, was ~25 min. Blood sampling for plasma radioactivity data was conducted repeatedly from the tracer injection to the end of PET scan.

At the cessation of the exercise protocol, the subjects were taken to the PET scanner with a minimal leg muscle usage. PET scanning was followed by MRI.
Eccentric Rehabilitation Intervention

The rehabilitation program comprised eccentric training for calf muscles of the Pain leg done twice a day, every day, for 12 successive wk (1). Each session consisted of 3 sets of 15 eccentric contractions each, which amounted to a total of 90 contractions per day. Slight modification in the training technique was made for oneinsertion-type tendinopathy patient, as proposed by Jonsson and colleagues (16). Each subject was instructed about the proper exercise technique through practical demonstrations and written instructions. During the rehabilitation period, all subjects were asked to maintain a training diary. They documented the time of the day, pain level, and the intensity of the exercise, as well as other daily physical training and activities performed during the 12 wk. Each subject started the training by using only body weight, and additional weights (2.5 kg/wk on average), in the form of backpack loads, were added as the training progressed. Reevaluation of the exercise intensity was made after every 2 wk, and necessary modification, in terms of adding or removing weight, was made accordingly. Virtual analog scale (VAS) pain level was documented at the end of each eccentric training session by the subjects.

Statistical Analysis

Paired-samples T-test was used to compare the legs with the two study groups across all GU and SEMG variables before (Pre) and after the rehabilitation (Post). For between-subject analyses, unpaired-sample T-test was chosen. A paired-samples T-test was utilized to conduct the longitudinal analysis of VISA-A scores. For comparing various muscles within a leg, repeated-measures analysis of variance, with Bonferroni confidence interval adjustment, was used. Additionally, statistical correlations (Pearson’s r) were computed between the changes in submaximal exercise target force level and the changes in GU rate and normalized SEMG of individual muscle/tendon due to rehabilitation. All statistical analyses were done with IBM SPSS 20.0 (IBM, New York, NY) software. Alpha (α) level of significance was set at a P value of 0.05. The results are reported as means ± SD.

RESULTS

Plantarflexion Force

Maximal contraction plantar flexion force from the asymptomatic (No-Pain) leg was significantly greater than that of the Pain leg (1,262 ± 198 vs. 1,104 ± 185 N, P < 0.05) during Pre measurements. Significant improvement (P < 0.001) in the plantar flexion force production in the Pain leg (1,104 ± 185 vs. 1,298 ± 253 N) was seen Post, while the increase in No-Pain was also significant (P < 0.05). During the Post measurements, the No-Pain leg was not significantly stronger than the Pain leg (1,343 ± 209 vs. 1,262 ± 198 N). No significant differences between either Pain or No-Pain and CTRL legs were seen.

Submaximal force. The 30% MVC target plantar flexion force used in the isometric exercise protocol for Pain, in absolute terms, was significantly greater during Post vs. Pre (P < 0.001, 392 ± 83 vs. 328 ± 48 N). No-Pain also exercised at higher force level during Post (P < 0.05) than Pre. CTRL group legs did not show within- or between-group differences. Similarly, no difference between the submaximal target force levels of Pain and No-Pain during the Post session was present.

Muscle GU

GU results are shown in Table 1. At baseline, no significant differences in the GU rate of SOL, MG, LG, FHL, and AT were present between the two legs of either group. However, compared with CTRL, FHL took up more glucose in No-Pain (P < 0.05). In terms of intermuscle comparisons, no significant differences among muscles were present within either Pain or No-Pain. Nevertheless, in the Right leg, MG was greater than both LG (P = 0.013) and FHL (P = 0.009), while in the left leg only MG was greater than FHL (P = 0.043). As a result of 12-wk eccentric training, GU rate of SOL (P = 0.003) and LG (P = 0.000) rose significantly in Pain. In No-Pain, there was an increase in MG and FHL uptake (P < 0.05). During Post measurements, all muscle-tendon GU rates were significantly different between the groups for both legs (Table 1). Concerning intermuscle differences, GU rates of both SOL and MG were greater than FHL (P = 0.019; 0.022) in Pain. There were no intermuscle differences (SOL > FHL: P = 0.054) in No-Pain.

No significant correlation was present between post-rehabilitation changes in GU of any muscle and changes in submaximal target force levels in either leg of the patient group.

Tendon GU

Both at baseline and Post, both patient legs had higher tendon GU than CTRL (Pain: P < 0.05, No-Pain: P < 0.01), while there was no effect of rehabilitation (Table 1). All three TS components (SOL, MG, LG) have significantly higher GU than AT (P < 0.002, 0.023, and 0.009, respectively) in Pain. In No-Pain, only LG and FHL showed greater GU than AT (P = 0.022; 0.008). In CTRL, all four muscles took up GU at greater rate than AT in both Right (P < 0.02) and Left (P < 0.02) legs.

At Post, all muscles had higher GU than AT (P < 0.025) in Pain. As for No-Pain, all TS components had greater GU than AT (P < 0.05). Additionally, significant positive correlation was found between the changes in AT GU and changes in submaximal target force levels over 12 wk of eccentric training in Pain (r = 0.70, P < 0.05).

Table 1. Muscle-tendon glucose uptake values for both study groups from both study sessions

<table>
<thead>
<tr>
<th></th>
<th>Pain</th>
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<th></th>
<th>Controls</th>
<th></th>
<th></th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Pre Post</td>
<td>%ΔGU</td>
<td></td>
<td></td>
<td>Right</td>
<td></td>
<td>Left</td>
</tr>
<tr>
<td>Solus</td>
<td>2.64 ± 0.74a</td>
<td>3.47 ± 0.98a</td>
<td>39.65 ± 52.07</td>
<td></td>
<td>3.03 ± 1.85</td>
<td>3.50 ± 1.07a</td>
<td>31.04 ± 43.90</td>
</tr>
<tr>
<td>Medial gastrocnemius</td>
<td>3.08 ± 1.54c</td>
<td>3.56 ± 0.80a</td>
<td>32.82 ± 54.72</td>
<td></td>
<td>3.53 ± 2.94a</td>
<td>4.13 ± 2.27a</td>
<td>31.91 ± 35.79</td>
</tr>
<tr>
<td>Lateral gastrocnemius</td>
<td>2.43 ± 0.76c</td>
<td>3.20 ± 0.74a</td>
<td>38.65 ± 37.58</td>
<td></td>
<td>2.78 ± 1.39c</td>
<td>3.06 ± 0.91c</td>
<td>20.16 ± 34.94</td>
</tr>
<tr>
<td>Flexor hallucis longus</td>
<td>2.22 ± 1.0c</td>
<td>2.55 ± 0.66c</td>
<td>24.59 ± 40.35</td>
<td></td>
<td>2.08 ± 0.41c</td>
<td>2.40 ± 0.55cd</td>
<td>18.02 ± 31.55</td>
</tr>
<tr>
<td>Achilles tendon</td>
<td>1.50 ± 0.51c</td>
<td>1.73 ± 0.64c</td>
<td>22.81 ± 48.39</td>
<td></td>
<td>1.39 ± 0.45c</td>
<td>1.63 ± 1.00c</td>
<td>15.74 ± 41.54</td>
</tr>
</tbody>
</table>

Values are means ± SD of glucose uptake (GU) in μmol/100 g tissue min⁻¹. Pain, symptomatic leg; No-Pain, asymptomatic leg; Pre, before rehabilitation; Post, after rehabilitation; %Δ, percent change from Pre to Post. Significant difference between Pre and Post: *P < 0.05, **P < 0.01, ***P < 0.001. Significant difference between Pain and No-Pain, P < 0.05. Significantly different from control: 1P < 0.05, 2P < 0.01, 3P < 0.001.
Table 2. Submaximal normalized surface electromyography root mean square values for both study groups during both study sessions

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Pain Pre</th>
<th>Pain Post</th>
<th>%ΔEMG</th>
<th>No-Pain Pre</th>
<th>No-Pain Post</th>
<th>%ΔEMG</th>
<th>Controls Right</th>
<th>Controls Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial gastrocnemius</td>
<td>30.74 ± 10.05</td>
<td>34.64 ± 14.57</td>
<td>18.71 ± 43.46</td>
<td>29.10 ± 8.21</td>
<td>28.7 ± 8.14</td>
<td>09.21 ± 44.72</td>
<td>29.82 ± 10.06</td>
<td>33.37 ± 9.71</td>
</tr>
<tr>
<td>Lateral gastrocnemius</td>
<td>19.17 ± 4.95 *</td>
<td>26.47 ± 10.52</td>
<td>38.38 ± 39.32</td>
<td>16.5 ± 8.95</td>
<td>18.43 ± 6.15</td>
<td>64.26 ± 137.54</td>
<td>22.41 ± 11.65</td>
<td>22.41 ± 11.65</td>
</tr>
<tr>
<td>Flexor hallucis Longus</td>
<td>25.58 ± 12.81</td>
<td>22.67 ± 19.03</td>
<td>8.86 ± 49.19</td>
<td>21.48 ± 20.74</td>
<td>17.42 ± 11.59</td>
<td>22.69 ± 92.00</td>
<td>36.60 ± 18.01</td>
<td>36.12 ± 34.68</td>
</tr>
</tbody>
</table>

Values are means ± SD of electromyography (EMG) in %maximal voluntary contraction EMG. Due to missing values, n is different in the statistical analyses from that used to calculate mean values here. *Significantly different between Pre and Post: P < 0.01. Significantly different between Pain and No-Pain: ‡P < 0.05. §Significantly different from control: P < 0.05.

Electromyography

At baseline, SOL showed more relative SEMG activity in Pain compared with both No-Pain and CTRL (P < 0.05). No significant within- or between-group differences were evident for other muscles (Table 2). As for intermuscle comparison, there were no significant differences among the muscles in any leg in both groups.

At Post, in the Pain leg, normalized SEMG in LG was significantly higher (P < 0.01) compared with baseline. In the case of No-Pain, there was no major change in the SEMG of any of the muscles as a result of rehabilitation. Consequently, during the Post session, LG displayed greater activity in Pain than No-Pain (P < 0.001), while SEMG of other muscles was not significantly different within or between groups (Table 2). Regarding intermuscle differences, only MG displayed greater SEMG than FHL (P = 0.034) in Pain. No such differences in No-Pain and CTRL legs were evident. Muscle SEMG in neither Pain nor No-Pain was different from CTRL at Post. The relative contribution of SOL, as a percentage of cumulative SEMG of all four muscles, was reduced, while that of MG increased after the training (P < 0.05) (Fig. 1 A and B).

No significant correlations were present between changes in the TS SEMG and changes in plantar flexion target force for either leg of the patient group.

Questionnaires

The average VISA-A questionnaire score at Pre was 63.8 ± 19 (range 27–86). As a result of 12-wk eccentric calf muscle training, the score rose significantly (P < 0.05) to 79.6 ± 17 (range 52–100). However, individual VISA-A questionnaire scores displayed a heterogeneous trend (Fig. 2).

The VAS pain score, registered after each eccentric exercise session, showed a nonsignificant decline due to rehabilitation. Training compliance, based on the self-reported training diaries, was 90% in terms of days and 81% in terms of overall training sessions, with an average of ~6,050 eccentric actions.

DISCUSSION

Eccentric exercise training improved maximal plantar flexion force of the Pain leg by 18%. The consequent increase in force during the submaximal exercise was accompanied by a significant rise in the muscle GU of SOL and LG. While the AT GU in both patient legs was greater than in CTRL, rehabilitation did not have an effect on tendon GU. In terms of SEMG, rehabilitation-induced increase in LG myoelectric activity in the Pain leg meant that LG had significantly higher activity, compared with the No-Pain leg, during Post testing. Self-reported qualitative assessment (VISA-A questionnaire) suggested significant symptom relief in this study, where the training compliance was 90%.

As hypothesized, the maximal plantar flexion force production improved significantly in the Pain leg as a result of eccentric rehabilitation (P < 0.05). Due to this strength gain, mean maximal force level at Post was similar between legs. Due to the study protocol, the target force level of submaximal isometric exercise was also higher in the Pain leg at Post (P < 0.01). Our findings confirm those of Alfredson et al. (1), who reported significant weakness of the injured leg at the baseline, followed by significant improvement as a result of eccentric rehabilitation.

It was expected that greater plantar flexion force at Post would lead to greater GU in the TS muscle. In Pain, the isometric exercise-induced GU rate of all TS components during Post rose compared with the baseline values. Both SOL and LG had increments of nearly 40%, while MG showed...
considerable increase as well (~30%). This finding confirms our hypothesis that GU rate of TS would increase at Post. While this increase in GU rate was concomitant with increased force level, lack of correlation between the changes in muscle GU rate and force leave some open questions.

It is known that Achilles tendinopathy-induced tendon pain decreases muscle torque production (3, 37). Therefore, the enhanced muscle GU seen here may be indicative of restoration of normal contribution of TS to force production, as the tendon pain subsided and normal force transmission through the tendon reestablished. FHL manifested the smallest rise in muscle GU, signifying that most of the improved plantar flexion strength was accomplished by the superficial plantar flexors. Similar observation was reported by Finni et al. (7), showing that the initial restoration of plantar flexion strength after physical rehabilitation of ruptured AT may be due to increased contribution from FHL rather than restoration of TS muscle function.

Although intermuscle differences were not significant at baseline, both SOL and MG of TS significantly higher GU than FHL at Post. Subsequently, the Post GU of all four muscles was significantly higher than CTRL, even though both groups were comparable at baseline. In the No-Pain leg, GU rate increased significantly in both MG and FHL due to eccentric training, with no intermuscle differences at either baseline or after training.

Not surprisingly, the GU uptake rate in the Achilles tendinous tissues was significantly less than that of muscle tissues in both study groups. Surprisingly, however, AT took up more glucose in both Pain and No-Pain legs of the patient group, compared with the healthy CTRL legs at baseline, and stayed elevated at Post. Since Achilles tendinopathy could lead to a higher cell count (28), it may explain an increased metabolic demand in the Pain tendon. Alternatively, the presence of mild tendon inflammation/neovascularization at Pre could be responsible for higher GU. Higher GU in the Pain AT could, however, be explained by transmission of greater muscle force through the tendon. Previously, eccentric exercise has been shown to reverse the neovascularization in Achilles tendinopathy (25a), thereby likely reducing tendon GU, since glucose delivery rate is negatively affected by reduced local blood flow, at least in muscle tissues (30). The unexpected finding that tendon GU did not fall due to eccentric training in our study could be the result of an increased GU due to higher plantar flexion torque production during Post testing. This assertion is supported by the existence of significant correlation between rises in tendon GU and target plantar flexion force levels during submaximal isometric exercise.

Overall, there is a dearth of information concerning GU behavior of ankle plantar flexors in submaximal isometric conditions among patients with lower leg musculoskeletal problems. However, exercise in general has been shown to increase plantar flexor myotenon GU. For instance, a case study involving a 30-yr-old man reported a threefold increase in MG GU, while AT uptake increased two times compared with the resting contralateral leg (17). Although theoretically muscle GU may be influenced by muscle fiber type, it will be merely speculative to discuss our findings on these lines in the absence of relevant histological data. Only one past study has reported an abnormal, enhanced AT GU in the case of Achilles tendinitis (15).

Similar to the clear and marked pattern revealed by GU findings, SEMG results were quite comparable except for SOL in Pain. Increased electrical activity of FHL in the Pain leg, at the baseline, was expected as a compensatory mechanism to augment the reduction in the input of the TS due to AT pain. Heightened myoelectric activity of SOL, on the other hand, was not expected in the Pain leg, and the underlying mechanism is open to interpretation. It must be noted, though, that the myoelectric activity of SOL, relative to that of FHL, was similar in the Pain and No-Pain legs, thanks to a simultaneous increase in the activity of both muscles on the Pain side. While the eccentric rehabilitation diminished the relative myoelectric activity of SOL, it increased the activity of both gastrocnemii in the Pain leg. LG was shown to be significantly more active (P < 0.05) during the Post session compared with the baseline activity. Thus the most comparable muscle between the two techniques was LG, which displayed significantly higher metabolic and myoelectric activities at Post. The myoelectric activity data of FHL displayed large standard variation, which denotes substantial individual variation as to the extent of FHL’s contribution to isometric plantar flexion task. This finding confirms previous reports of large interindividual variation in FHL contribution (4, 7). The SEMG of FHL remained largely unchanged, which is in line with the PET findings. Similar to PET, there were no significant SEMG differences among muscles at baseline. However, during Post, MG showed significantly more activity than FHL. It can be stated that eccentric training leads to an apparent redistribution of myoelectric activity of TS components in Pain, with SOL reducing its role only to be compensated by an increased input by the gastrocnemii, as demonstrated in Fig. 2. Alternatively, the tendinopathy might have negatively affected the myoelectric activity of gastrocnemii, which resulted in a compensatory increase in SOL activity only to be ultimately normalized by the eccentric exercise.

It might be possible that, in the TS muscle with a painful tendon, some components contribute relatively more under diminished strength during submaximal exercise. In our case,
SOL showed considerably greater activity than LG before eccentric training. As the tendon healed, contributions of all muscles became more or less homogenous, with no remaining major TS intermuscle differences. As indicated by both PET and SEMG, the least active TS component at baseline was LG and showed greatest increments after 12 wk.

In contrast to Pain, the No-Pain leg SEMG of both superficial and deep plantar flexors did not change at Post. Since the SEMG of LG increased significantly at Post in Pain, while staying unchanged in No-Pain, LG on the Pain side had significantly higher SEMG than No-Pain subsequent to training. In fact, all other muscles in No-Pain tended to have a slightly lower SEMG compared with Pain. No significant differences among muscles were evident in either study group.

To the best of the authors’ knowledge, there are no published reports addressing the effects of eccentric training on calf muscle activation behavior in chronic AT pain. The difference in the electrical and metabolic behaviors of the Pain and No-Pain legs can possibly be explained by the difference in the nature of muscle actions performed during the rehabilitation. As originally devised, the Pain legs performed only the eccentric contractions, while the concentric muscle action was done by the No-Pain legs (1). Finally, the possibility of altered muscle activity being a cause, rather than a consequence, of AT pain cannot be ruled out.

Regarding SEMG results, one fundamental assumption made in this study should be noted as a limitation. It was assumed that all compartments of TS muscle were fully activated during the MVC recordings. Additionally, the SEMG results of this study are normalized to the RMS during the MVC and, hence, only represent the activity of the muscles relative to the maximal plantar flexion effort.

In terms of the questionnaire-based (VISA-A) assessment of the AT pain, most patients displayed improvement, although the VISA-A score dropped at Post in two subjects. Overall, the score increased by 25% due to the rehabilitation. VAS pain score was expected to fall, but the recorded decline was nonsignificant. The lack of correlation between VISA-A and pain VAS scores could stem from the fact that pain scores were recorded immediately after the eccentric exercise bouts only and thus may not represent pain associated with general physical activity. Although lack of training compliance has been an issue with many training studies, our subjects demonstrated a high compliance, with training performed on 90% of days (range: 74–100%).

In conclusion, eccentric rehabilitation significantly improved plantar flexion strength in the Pain leg, which was accompanied by enhanced TS GU, increased electrical activity of the gastrocnemii, and improved VISA-A scores. Although there was a rehabilitation-induced redistribution of myoelectric activation within the TS muscle, no such effect was seen in GU patterns.

GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


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