Electromyographic Evidence of Excessive Achilles Tendon Elongation During Isometric Contractions After Achilles Tendon Repair

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Background: Weakness in end-range plantarflexion has been demonstrated after Achilles tendon repair and may be because of excessive tendon elongation. The mean frequency (MNF) of surface electromyogram (EMG) data during isometric maximum voluntary contraction (MVC) increases with muscle fiber shortening.

Hypothesis: During isometric plantarflexion, MNF during MVCs will be higher on the involved side compared with the uninvolved side after Achilles tendon repair because of excessive tendon elongation and greater muscle fiber shortening.

Study Design: Case series; Level of evidence, 4.

Methods: Isometric plantarflexion MVC torque was measured at 20° and 10° dorsiflexion, neutral, and 10° and 20° plantarflexion in 17 patients (15 men, 2 women; mean age, 39 ± 9 years) at a mean 43 ± 26 months after surgery. Surface EMG signals were recorded during strength tests. MNF was calculated from fast Fourier transforms of medial gastrocnemius (MG), lateral gastrocnemius (LG), and soleus (SOL) EMG signals.

Results: Patients had marked weakness on the involved side versus the uninvolved side in 20° plantarflexion (deficit, 28% ± 18%; $P < .001$) but no significant weakness in 20° dorsiflexion (deficit, 8% ± 15%; $P = .195$). MNF increased when moving from dorsiflexion to plantarflexion ($P < .001$), but overall, it was not different between the involved and uninvolved sides ($P = .195$). However, differences in MNF between the involved and uninvolved sides were apparent in patients with marked weakness. At 10° plantarflexion, 8 of 17 patients had marked weakness (>20% deficit). MNF at 10° plantarflexion was significantly higher on the involved side versus the uninvolved side in patients with weakness, but this was not apparent in patients with no weakness (side by group, $P = .012$). Mean MNF at 10° plantarflexion across the 3 muscles was 13% higher on the involved side versus the uninvolved side in patients with weakness ($P = .012$) versus 3% lower in patients with no weakness ($P = .522$).

Conclusion: Higher MNF on the involved side versus the uninvolved side in patients with significant plantarflexion weakness is consistent with greater muscle fiber shortening. This indicates that weakness was primarily because of excessive lengthening of the repaired Achilles tendon. If weakness was simply because of atrophy, a lower MNF would have been expected and patients would have had weakness throughout the range of motion. Surgical and rehabilitative strategies are needed to prevent excessive tendon elongation and weakness in end-range plantarflexion after Achilles repair.

Keywords: weakness; EMG; mean frequency; plantarflexion; MVC

Several studies have demonstrated the separation of Achilles tendon repairs in the weeks after surgery.[9,10,12,15] These studies indicate that approximately 1 cm of separation occurs in the first 10 to 12 weeks after surgery, with no subsequent further separation. More recently, Eliasson et al[2] demonstrated Achilles tendon elongation up to 6 months after Achilles repair. Mullaney et al[11] demonstrated a marked weakness in end-range plantarflexion in patients at a minimum of 6 months after Achilles repair with no weakness in dorsiflexion. These patients were also unable to perform a heel raise when initiated from a position of plantarflexion (standing on a decline), but they were less impaired when standing on a flat surface and not impaired when standing on an incline. This disproportionate weakness in plantarflexion was attributed to excessive tendon elongation during plantarflexion contractions creating active insufficiency at very short muscle lengths. More recently, in a follow-up study,[14] weakness in end-range plantarflexion was still evident despite the use of stronger repairs (epitendinous augmentation) and avoidance of stretching into dorsiflexion for the first 12 weeks after surgery. Furthermore, the amplitude of the surface electromyogram (EMG) signal was not different

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between the involved and noninvolved sides during plantarflexion contractions, indicating that the observed disproportionate weakness in plantarflexion was not because of inhibition. However, the logical assumption that end-range plantarflexion weakness after Achilles repair is due to excessive tendon elongation has yet to be confirmed by ultrasound imaging or other techniques.

Analysis of the frequency content of the surface EMG signal during isometric contractions at different joint angles on the involved and noninvolved sides of patients after Achilles repair may yield information on excessive tendon elongation. The frequency content of the surface EMG signal is typically quantified using mean frequency (MNF) or median frequency (MDF) computations. MNF and MDF are indirect measures of muscle fiber conduction velocity, and fibers with a smaller diameter have a slower conduction velocity. Thus, weakness of the quadriceps muscles after anterior cruciate ligament injury has been associated with lower MDF.[7,8] Similarly, patients with severe ankle osteoarthritis had plantarflexion weakness (38% deficit) and lower soleus (SOL) (17%) and medial gastroc nemius (15%) MNF on the involved side versus the noninvolved side.[18]

The frequency content of the surface EMG signal is also sensitive to changes in muscle length, with higher frequencies at shorter muscle lengths.[3,17] This effect is because of higher conduction velocities with larger muscle fiber diameters at short versus long muscle lengths. During isometric contractions, muscle fibers shorten as tendons elongate.[6]

If disproportionate weakness at short muscle lengths after Achilles tendon repair is because of excessive elongation of the tendon during isometric contractions, then at any given joint angle, muscle fibers would be shorter on the involved side versus the noninvolved side. Therefore, MNF would be expected to be higher on the involved side versus the noninvolved side. However, if weakness is because of atrophy, a lower MNF would be expected on the involved side versus the noninvolved side during isometric maximum voluntary contractions (MVCs) and the weakness would not be disproportionate at shorter muscle lengths. The purpose of this study was to compare MNF between the involved and noninvolved sides during isometric plantarflexion MVCs performed at multiple joint angles from dorsiflexion to plantarflexion. It was hypothesized that MNF would be higher on the involved side versus the noninvolved side, indicating excessive tendon elongation and greater muscle fiber shortening.

METHODS

Surface EMG signals from 17 patients were recorded during isometric plantarflexion MVCs at 20° and 10° dorsiflexion, neutral, and 10° and 20° plantarflexion. The functional results for these patients have already been published.[14] The previously published data included strength, EMG amplitude, range of motion, girth, functional tests, and passive ankle stiffness. These data were from 18 patients, but the EMG data for 1 of those patients were unavailable for frequency analysis in the present study. This study consists of the previously reported strength data with the addition of EMG frequency data. The 17 patients in the present study were a mean 43 ± 26 months post–Achilles tendon repair (range, 9 mo to 8 years). There were 15 men and 2 women, with a mean age of 39 ± 9 years at the time of follow-up. All patients gave informed consent before participation, and the study protocol was approved by an institutional review board.

The details of the surgery and subsequent rehabilitation were described in detail in the original study.[14] Briefly, 4-strand Krackow technique suture repairs were performed with an epitendinous cross-stitch weave augmentation. All patients were nonweightbearing for 6 weeks, weightbearing in a controlled ankle motion walker boot for 3 weeks, and unassisted walking at 9 weeks postsurgically. Importantly, neither active nor passive stretching of the Achilles or gastrocnemius-SOL complex past the initial tendon tension was permitted for the first 12 weeks after surgery. Jogging and return to sports were milestone-dependent and typically started around 13 to 20 weeks and 20 weeks to 1 year, respectively. To be included in this study, patients had to have completed the rehabilitation protocol with no postoperative complications and to have returned to their desired activities.

Strength Testing

The full procedures for strength testing were described previously[14] and are described in brief here. Maximum isometric plantarflexion torque was measured at 20° and 10° of dorsiflexion, neutral position, and 10° and 20° of plantarflexion (Biodex System 2; Biodex Medical Systems). The percentage strength deficit at each test angle between the involved and noninvolved limbs is reported.

Electromyography

Surface EMG data were acquired from each leg during isometric contractions. After the skin of each patient was shaved, cleaned, and lightly abraded, disposable silver/silver chloride passive dual electrodes (2.0-cm interelectrode distance) (Noraxon) were applied. Electrodes were placed over the muscle bellies of the medial gastrocnemius (MG), lateral gastrocnemius (LG), and SOL muscles in

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accordance with the *Anatomical Guide for the Electromyographer*.[16] Muscle activity was sampled at 1000 Hz (common mode rejection ratio >110 dB at 50-60 Hz; input impedance >10 GO) (BTS FREEEMG 1000; BTS Bioengineering).

Frequency analysis of the EMG data was performed using custom scripts written in MATLAB R2017b (The MathWorks). For each muscle, 1 s of data around the peak amplitude was selected for analysis. Power spectral density (PSD) of these segments was estimated using the Welch modified periodogram method, with a 256-point Hanning window with an overlap of 128 points. The Welch method was used as it reduces the variability of the PSD estimate.[13] MNF was then calculated for the PSD of each muscle. The mean MNF for each of the 3 contractions at each angle was calculated for each muscle.

Statistical Analysis

Single-factor (angle), repeated-measures analysis of variance (ANOVA) was used to assess isometric plantarflexion strength deficits across the 5 test angles. The effects of muscle length (joint angle) and Achilles repair (involved versus noninvolved) on MNF across the 3 plantarflexors were assessed using muscle by angle by side repeated-measures ANOVA. Bonferroni correction was used for planned pairwise comparisons.

RESULTS

Strength

As previously reported,[14] patients had disproportionate weakness with decreasing muscle length (when moving from dorsiflexion to plantarflexion; angle effect, $P < .001$). Strength deficits were 28% ± 18% at 20° plantarflexion ($P < .001$), 18% ± 16% at 10° plantarflexion ($P < .001$), 14% ± 14% at neutral angle ($P = .005$), 9% ± 14% at 10° dorsiflexion ($P = .07$), and 8% ± 15% at 20° dorsiflexion ($P = .195$). Clinically significant strength deficits (>20%) were seen in 14 of 17 patients at 20° plantarflexion, 8 of 17 patients at 10° plantarflexion, 9 of 17 patients at neutral angle, 4 of 17 patients at 10° dorsiflexion, and 4 of 17 patients at 20° plantarflexion.

EMG Mean Frequency

MNF during isometric contractions increased from dorsiflexion to plantarflexion (angle effect, $P < .001$), consistent with the known effect of muscle length on conduction velocity. However, this effect varied between muscles (angle by muscle, $P < .002$) with greater increases in MNF from dorsiflexion to plantarflexion for the MG and LG compared with the SOL (Figure 1). Contrary to the hypothesis, there was no significant difference in MNF between the involved and noninvolved sides (side effect, $P = .195$) nor was there a difference between sides in the increase in MNF with decreasing muscle length (side by angle, $P = .473$) (Figure 1).

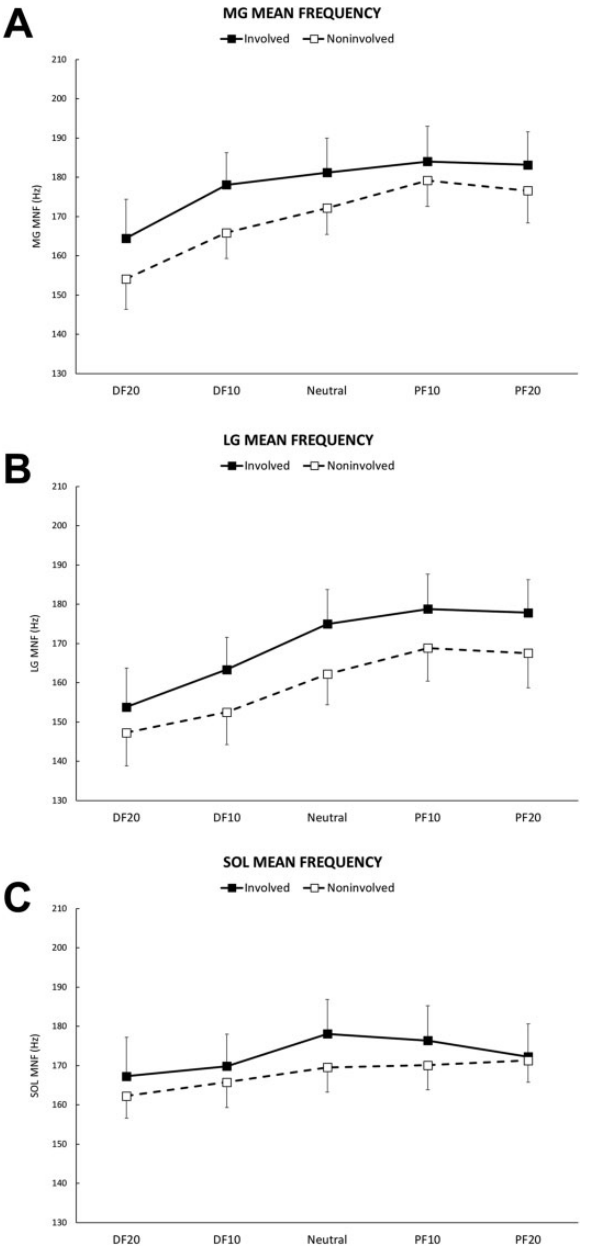

Figure 1. Effect of joint angle on MNF for the involved (closed squares) and noninvolved (open squares) (A) MG, (B) LG, and (C) SOL. Muscle by angle, $P = .002$; angle effect for MG and LG, $P < .001$; and for SOL, $P = .02$. There was a greater angle effect for MG versus SOL ($P = .014$) and LG versus SOL ($P = .001$), and no difference in angle effect between MG and LG ($P = .45$). Side effect (involved vs noninvolved), $P = .195$; side by angle, $P = .473$. DF, dorsiflexion; LG, lateral gastrocnemius; MG, medial gastrocnemius; MNF, mean frequency; PF, plantarflexion; SOL, soleus.

Since it was hypothesized that weakness in plantarflexion was because of excessive tendon elongation on the involved side, MNF was compared between patients with and without marked weakness in plantarflexion (>20% deficit). Eight patients had a marked weakness in

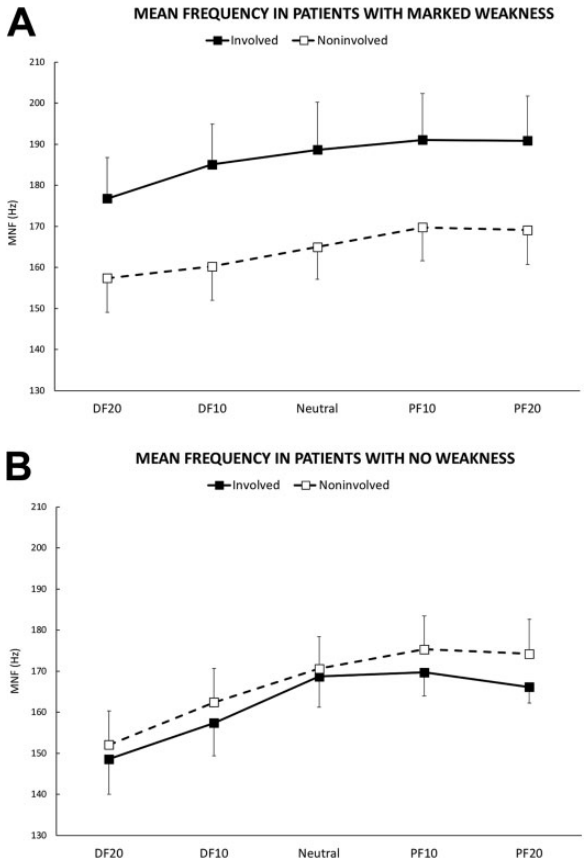

Figure 2. Comparison of MNF between patients with (A) marked weakness and (B) patients with no weakness. MNF values for the MG, LG, and SOL are averaged. Side (involved vs noninvolved) by group (marked weakness vs no weakness), $P = .015$. Higher MNF was found on the involved side versus the noninvolved side for patients with marked weakness ($P = .012$) and there was no difference in MNF between the involved and noninvolved side for patients with no weakness ($P = .522$). DF, dorsiflexion; LG, lateral gastrocnemius; MG, medial gastrocnemius; MNF, mean frequency; PF, plantarflexion; SOL, soleus.

plantarflexion (deficit >20% at both 10° and 20° plantarflexion).

MNF was significantly higher on the involved side (186 ± 25 Hz) compared with the noninvolved side (164 ± 23 Hz) for patients with marked weakness ($P = .012$), but it was not different between the involved (162 ± 20 Hz) and noninvolved sides (167 ± 22 Hz) for patients without marked weakness ($P = .522$) (Figure 2).

DISCUSSION

The hypothesis that MNF of the plantarflexors would be higher on the involved side versus the noninvolved side during isometric MVCs was not directly supported by the data (Figure 1). However, when the patients were separated into those with marked weakness in plantarflexion (>20% deficit at 10° and 20° plantarflexion) versus no weakness (deficit ≤ 20% at either angle), it was apparent that weakness was associated with higher MNF. The clinical interpretation is that the marked weakness in plantarflexion was because of excessive elongation of the previously repaired Achilles tendon such that the muscle fibers shortened into a position of active insufficiency (on the ascending limb of the length-tension curve). The basis of this interpretation is that indices of EMG frequency, such as MNF, are sensitive to muscle fiber lengths, with higher frequencies at shorter muscle lengths. Thus, it is important that MNF was significantly higher during MVCs in plantarflexion (short muscle length) versus dorsiflexion (long muscle length). This was clearly the case for the MG and LG but less so for the SOL (Figure 1).

The fact that SOL MNF during isometric MVCs was less affected by joint angles compared with MG and LG implies that SOL muscle fibers shortened less than MG and LG fibers during isometric MVCs when moving from dorsiflexion to plantarflexion. This interpretation is supported by measurements of fascicle length changes during isometric plantarflexion MVCs in healthy men.[6] Kawakami et al[6] measured MG, LG, and SOL fascicle lengths during isometric contractions at 15° dorsiflexion, neutral, 15° plantarflexion, and 30° plantarflexion. Fascicle shortening during MVCs was significantly less in the SOL versus the MG and LG. Specifically, during MVCs at 15° plantarflexion versus 15° dorsiflexion (30° difference), MG fascicles were 10 mm shorter and LG fascicles were 13 mm shorter, while SOL fascicles were only 7 mm shorter. In the present study, during MVCs on the noninvolved side at 20° dorsiflexion versus 10° plantarflexion (30° difference), MG MNF increased by 22 Hz, LG MNF increased by 23 Hz, and SOL MNF only increased by 8 Hz.

While these EMG frequency analyses provide indirect evidence of excessive tendon lengthening during isometric contractions, this technique would not be sufficiently sensitive to use on a case by case basis. Thus, if one wanted to confirm, in a particular patient, that disproportionate weakness in plantarflexion after Achilles repair was because of excessive tendon lengthening, ultrasound imaging of fascicle length changes would be needed. However, this ultrasound technique is used primarily by physiologists for research purposes and not by radiologists or ultrasonographers for clinical purposes.

For the majority of patients, weakness in end-range plantarflexion after Achilles repair will not have a marked effect on function. Most patients who rupture their Achilles are in their 30s or older and are therefore unlikely to be involved in explosive running and jumping sports that place a high demand on plantarflexion strength in a plantarflexed position. However, for dancers, basketball players, and gymnasts, load absorption and propulsion at the Achilles is critical to executing routine functions in those sports. Weakness in end-range plantarflexion would likely impair performance. Of note, only 11 of 18 professional basketball players in the National Basketball Association (NBA) returned to play in the NBA after Achilles rupture; they had lower performance ratings and shorter careers than a control group.[1]

During normal gait, the Achilles serves to store and release elastic energy.[5] Peak EMG amplitude of the plantarflexors during walking occurs while the ankle is dorsiflexing.[5] This serves to elongate the Achilles tendon while the muscle fascicles remain isometric. During the late push-off phase, the ankle rapidly plantarflexes until toe-off. Importantly, there is very low EMG activity in the plantarflexors during this phase because rapid shortening of the plantarflexor muscle-tendon units is primarily due to elastic recoil of the tendinous tissues. This has been referred to as a catapult action.[5] It is likely that after Achilles tendon repair this catapult action is impaired, but this may be difficult to detect experimentally. Additionally, there may be an increased metabolic cost for walking and running after Achilles tendon repair because of decreased use of elastic energy.

The clinically relevant question is whether weakness in end-range plantarflexion can be avoided or reversed. Performing stronger repairs and avoiding stretching for 12 weeks did not prevent this weakness.[14] A successful intervention would need to increase tendon stiffness so that the fascicles do not shorten excessively in plantarflexion and so active insufficiency can be avoided. In healthy male participants, 12 weeks of eccentric training of the plantarflexors did not increase Achilles stiffness.[4] However, 10 weeks of isometric training increased Achilles stiffness by 18%, and during drop jump landings, gastrocnemius fascicle lengths were longer posttraining.[19] Interestingly, the SOL fascicle lengths were unchanged. This very much fits with the EMG observations in the present study and the fascicle length changes demonstrated by Kawakami et al,[6] indicating that SOL muscle fibers have smaller length changes during muscle contractions than the gastrocnemii. Isometric plantarflexor training in late rehabilitation ($\geq$4 months postoperative) after Achilles repair is a viable option for trying to reverse end-range plantarflexion weakness.

It is important to acknowledge that MNF is also related to muscle fiber type, with fast-twitch fibers having a higher frequency because of their having a larger cross-sectional area and faster conduction velocity. In fact, lower quadriceps MDF in patients before and after anterior cruciate ligament reconstruction has been attributed to fast-twitch fiber atrophy.[7] Higher plantarflexor MNF in patients with marked weakness in this study could be because of preferential activation of fast-twitch fibers or selective slow-twitch fiber atrophy. However, there is no rationale for why such an effect would only be apparent at short muscle lengths or how it could explain the selective weakness in plantarflexion. An additional limitation in this study is that anterior tibialis EMG activity was not recorded. The lower plantarflexion torque on the involved side could, in part, be because of anterior tibialis cocontraction.

In conclusion, higher MNF on the involved side versus the noninvolved side in patients with significant plantarflexion weakness is consistent with greater muscle fiber shortening. This indicates that weakness was primarily because of excessive lengthening of the repaired Achilles tendon. If weakness was simply because of atrophy, a lower MNF would have been expected and patients would have had weakness throughout the range of motion.

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