Weakness in End-Range Plantar Flexion After Achilles Tendon Repair

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Background: Separation of tendon ends after Achilles tendon repair may affect the tendon repair process and lead to postoperative end-range plantarflexion weakness.

Hypothesis: Patients will have disproportionate end-range plantarflexion weakness after Achilles tendon repair.

Study Design: Descriptive laboratory study.

Methods: Four-strand core suture repairs of Achilles tendon were performed on 1 female and 19 male patients. Postoperatively, patients were nonweightbearing with the ankle immobilized for 4 weeks. Plantarflexion torque, dorsiflexion range of motion, passive joint stiffness, toe walking, and standing single-legged heel rise (on an incline, decline, and level surface) were assessed after surgery (mean, 1.8 years postoperative; range, 6 months-9 years). Maximum isometric plantarflexion torque was measured at 20° and 10° of dorsiflexion, neutral, and 10° and 20° of plantar flexion. Percentage strength deficit (relative to noninvolved leg) was computed at each angle. Passive dorsiflexion range of motion was measured goniometrically. Passive joint stiffness was computed from increase in passive torque between 10° and 20° of dorsiflexion, before isometric contractions.

Results: Significant plantarflexion weakness was evident on the involved side at 20° and 10° of plantar flexion (34% and 20% deficits, respectively; $P < .001$), with no torque deficits evident at other angles (6% at neutral, 3% at 10° of dorsiflexion, 0% at 20° of dorsiflexion). Dorsiflexion range of motion was not different between involved and noninvolved sides ($P = .7$). Passive joint stiffness was 34% lower on the involved side ($P < .01$). All patients could perform an incline heel rise; 14 patients could not perform a decline heel rise ($P < .01$).

Conclusion: Disproportionate weakness in end-range plantar flexion, decreased passive stiffness in dorsiflexion, and inability to perform a decline heel rise are evident after Achilles tendon repair. Possible causes include anatomical lengthening, increased tendon compliance, and insufficient rehabilitation after Achilles tendon repair.

Clinical Relevance: Impairments will have functional implications for activities (eg, descending stairs and landing from a jump). Weakness in end-range plantar flexion may be an unrecognized problem after Achilles tendon repair.

Keywords: heel rise; isometric; elongation; length tension

Published reports on the strength of suture repairs of cadaveric Achilles tendons indicate that these repairs are extremely weak compared with the forces that the tendon is exposed to during walking. Commonly used repair techniques have been shown to fail at forces ranging from 45 to 250 N.[3,6,8,14,20] The strongest reported techniques are the triple-bundle technique failing at 453 N[6] and a 4-strand Krakow repair with epitendinous augmentation failing at 323 N,[8] but neither of these techniques appears to be in common use. Achilles tendon forces of approximately 1500 N have been recorded during walking in healthy subjects.[4] Achilles tendon forces of 370 N have been estimated for healthy subjects walking with the ankle immobilized in neutral.[1] The addition of a 1-in heel lift decreased the estimated force to 191 N. Considering the apparent weakness of Achilles tendon repairs, in comparison with forces during walking, it is surprising that the current practice of early postoperative weightbearing and early motion after Achilles tendon repair is not associated with an increased rerupture rate.[2,7,13,18,19] Although rerupture remains uncommon, excessive tendon lengthening may be a more likely occurrence with early weightbearing

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and aggressive rehabilitation. For example, separation of tendon ends has been clearly demonstrated after Achilles repair.[13,15,16] Of note, the degree of separation was not affected by early motion.[13]

Clinically excessive tendon lengthening after Achilles tendon repair is typically assessed by examining for increased passive or active dorsiflexion range of motion relative to the noninvolved side.[9,13] Increased active dorsiflexion motion (3°) and isokinetic plantarflexion weakness (13%) have been reported at a mean of 3.1 years after Achilles repair in patients with good overall functional outcome.[9]

Plantarflexion strength and the ability to perform a heel rise are commonly used methods to assess function after treatment for Achilles tendon rupture.[9,11,13] Strength is typically assessed isokinetically, with the peak values reported regardless of where in the range of motion they occur.[9,11,12,18] Heel rise performance is usually assessed in single-limb stance on a flat surface.[11,13] These tests (isokinetic strength and heel rise) may not be sensitive for detecting the functional impact of excessive tendon lengthening. Excessive tendon lengthening after Achilles tendon repair would be expected to decrease strength in end-range plantar flexion owing to insufficiency of the elongated tendon. However, peak plantarflexion torque occurs at or around 20° of dorsiflexion.[22] In addition, heel rise performance is dependent on the ability to initiate a forceful plantarflexion torque with the ankle in a neutral position. Assessment of strength and heel rise performance with the ankle in plantar flexion may provide a more sensitive indication of the functional impact of Achilles lengthening after surgical repair. For example, Mortensen et al[13] reported isometric plantarflexion strength to be 89% of the noninvolved side at 15° of dorsiflexion and 72% to 75% at 15° of plantar flexion more than 1 year after surgery. However, there was no discussion of the statistical or functional significance of this apparent difference. With respect to the effect of tendon lengthening on heel rise performance, testing patients on a declined surface may be a more sensitive test of impairment than is testing on a flat surface.

The purpose of this study was to determine if patients have disproportionate plantarflexion muscle weakness in end-range plantar flexion after Achilles tendon repair. To this end, plantarflexion torque and heel rise performance were assessed with the ankle in dorsiflexion, in neutral, and in plantar flexion.

METHODS

Nineteen male patients and 1 female patient (age, 40 ± 10 years) were examined at a mean of 1.8 years after surgical repair of the Achilles tendon (range, 6 months-9 years). All patients had acute midsubstance Achilles tendon ruptures. The surgical technique consisted of approximating the tendon ends with a 4-stranded modified Kessler core suture using No. 2 Ethibond (Ethicon Inc, Johnson & Johnson, Somerville, NJ). Postoperatively, patients were nonweightbearing for 4 weeks. Ankles were immobilized in a splint at 20° of plantar flexion immediately after surgery. At 1 week, the splint was removed, and the ankle was placed in a cam walker at 20° of plantar flexion. The cam walker was advanced to 15° of plantar flexion at 3 weeks. At 4 weeks postoperatively, patients began physical therapy. At this time, patients were allowed to bear weight as tolerated with the cam walker at 15° of plantar flexion. The cam walker was discontinued between 6 and 8 weeks postoperatively, and patients ambulated with a 1-in heel lift for 2 additional weeks. Patients began active nonweightbearing dorsiflexion motion from plantar flexion to neutral between the second and fourth postoperative week.

The investigating surgeons from our institute perform approximately 17 Achilles tendon repairs per year. Our testing population was selected from this patient pool once patients reached at least postoperative month 6 and returned to full activity. This patient sample was selected because the patients had no postoperative complications, had a normal progression in rehabilitation, had returned to their desired activities, and were satisfied with their treatment outcomes. Patients gave informed consent before participation, and the institutional review board approved the study.

Before testing, each patient performed a 5-minute warm-up on the treadmill at 3.0 to 3.5 mph with no elevation. This was followed by passive goniometric range of motion measurements for dorsiflexion and plantar flexion. Patients then performed the functional tests (toe walk, neutral heel rise, incline heel rise, and decline heel rise). The first 3 patients in this series did not perform the functional tests during the evaluation.

The toe walk consisted of walking in a plantarflexed position for 40 feet. If patients could maintain a plantarflexed foot, similar to the noninvolved side, the test had a negative result. If the heel was noticeably lower on the involved side or touched the floor, the test was graded as having a positive result. For the neutral single-legged heel rise, the patient was instructed to perform a heel rise while standing on the floor without using any manual support. If the patient was unable to elevate the involved heel from the ground to a similar height as the noninvolved side, then the test was graded as having a positive result. The same criteria were used for grading incline and decline single-legged heel rises. For the incline heel rise test, the patient stood on an incline board at an angle of 21° (of dorsiflexion) and was instructed to perform a heel rise with the noninvolved lower extremity, followed by the involved lower extremity. For the decline single-legged heel rise test, the patient stood on the same board facing the opposite direction (21° of plantar flexion). The patient was instructed to perform a heel rise from the plantarflexed position.

After a 5-minute rest period, plantarflexion torque was measured isometrically in newton-meters (Biodex System II, Biodex Medical Systems, Shirley, NY) at 20° of plantar flexion, 10° of plantar flexion, neutral (0°), 10° of dorsiflexion, and 20° of dorsiflexion. Patients were seated at 135° of trunk extension with the knee in full extension. Full knee extension was chosen, rather than a flexed knee, to enable an overall torque measure of the entire gastrocnemius-soleus complex. The foot was secured in the ankle testing

attachment with stabilization straps across the knee, upper thigh, and trunk. Order of angle testing was randomized to begin at either 20° of plantar flexion or 20° of dorsiflexion and progress toward the corresponding opposite angle. All patients began the testing with their noninvolved lower extremity and were instructed to perform maximal plantarflexion contractions at each angle. Three maximal contractions of 3 seconds' duration were performed at each angle. Patients were given a 10-second rest between contractions and a 1-minute rest between each set (test angle). The contraction yielding peak torque was recorded at each angle. The difference in plantarflexion torque between the involved and noninvolved sides at each test angle was expressed as a percentage strength deficit (noninvolved – involved/noninvolved).

Passive joint stiffness (N·m/deg) was computed from the measurement of passive tension measured at 10° and 20° of dorsiflexion. Before performing the maximal contractions at 10° and 20° of dorsiflexion, the patient was instructed to remain relaxed during a 3-second recording of the passive peak torque. Stiffness was computed as the increase in passive tension from 10° to 20° divided by the angular displacement (N/deg).

Data were analyzed with repeated-measures analysis of variance (strength and range of motion data) and Wilcoxon signed rank tests (functional tests). Bonferroni corrections were used for pairwise comparisons (involved vs noninvolved) when analysis of variance revealed significant main effects (side or angle) or interactions (side by angle). Mean ± SD data are reported in the text, and mean ± SE data are shown in the figures.

RESULTS

Plantarflexion Torque

Strength was not significantly different between the involved and noninvolved sides at 20° of dorsiflexion (0% ± 26% deficit), 10° of dorsiflexion (2% ± 31% deficit), and neutral (6% ± 37% deficit) for all 20 patients. In contrast, marked weakness was evident on the involved side at 10° of plantar flexion (20% ± 19% deficit, $P < .001$) and 20° of plantar flexion (34% ± 18% deficit, $P < .001$) (Figure 1). Ten patients had a clinically significant strength deficit of >20% at 10°, whereas 15 patients had a clinical strength deficit at 20°. Plantarflexion strength deficits were negatively correlated with time from surgery (less weakness with longer follow-up) at 20° of dorsiflexion ($r = -.54$, $P < .05$), 10° of dorsiflexion ($r = -.53$, $P < .05$), neutral ($r = -.7$, $P < .05$), and 10° of plantar flexion ($r = -.53$, $P < .05$). However, the strength deficit at 20° of plantar flexion was not significantly correlated with time from surgery ($r = -.17$, $P = .5$).

Angle-Torque Relationship

Peak plantarflexion torque occurred at 20° of dorsiflexion for 18 of 20 patients on the involved side and 17 of 20 patients on the noninvolved side. Peak torque occurred at 10° of dorsiflexion in the other patients. The decline in torque from 20° of dorsiflexion to 20° of plantar flexion was much greater on the involved side compared with the noninvolved side ($P < .001$) (Figure 2). At 10° of plantar flexion, isometric torque was 71% of peak torque on the noninvolved side but only 57% of peak torque on the involved side ($P < .01$). At 20° of plantar flexion, isometric torque was 56% of peak torque on the noninvolved side but only 37% of peak torque on the involved side ($P < .01$).

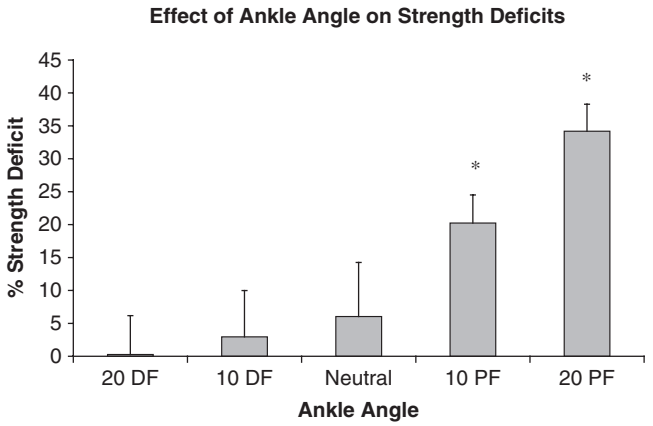

Figure 1. Isometric plantarflexion strength deficits throughout the ankle range of motion. Effect of ankle angle, $P < .0001$. *Significant strength deficits were apparent at 10° of plantar flexion ($P < .001$) and 20° of plantar flexion ($P < .001$). DF, dorsiflexion; PF, plantar flexion.

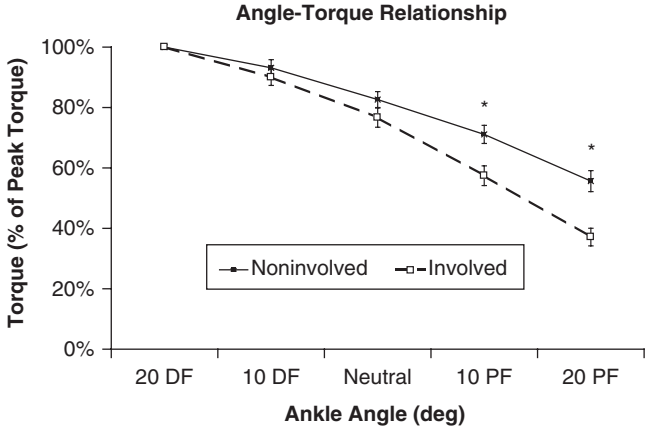

Figure 2. Angle-torque relationship for plantarflexion strength. Torque is expressed as a percentage of torque at 20° of dorsiflexion. Side (involved vs noninvolved) by ankle angle (20 DF, 10 DF, neutral, 10 PF, 20 PF) interaction, $P < .001$. Torque on the involved side was significantly lower than that on the noninvolved side at 10° of plantar flexion ($P < .01$) and 20° of plantar flexion ($P < .01$). DF, dorsiflexion; PF, plantar flexion.

Ankle Range of Motion

Passive dorsiflexion range of motion was not different between the involved and noninvolved sides (22° ± 3° vs

22° ± 2°, $P = .7$). Similarly, passive plantarflexion range of motion was not different between the involved and noninvolved sides (46° ± 6° vs 48° ± 6°, $P = .14$). The difference in passive dorsiflexion motion was 0.25° ± 2.9°. One patient had 4° greater dorsiflexion motion on the involved side, and 3 patients had 3° greater dorsiflexion motion on the involved side. One patient had 7° less dorsiflexion motion on the involved side, and 2 patients had 4° less dorsiflexion motion on the involved side.

Passive Joint Stiffness

Passive joint stiffness, measured as the increase in passive torque from 10° of dorsiflexion to 20° of dorsiflexion, was 34% lower on the involved side ($P < .01$). Stiffness was 0.7 ± 0.5 N/deg on the involved side versus 1.1 ± 0.7 N/deg on the noninvolved side.

Functional Strength Tests

Heel rise performance was markedly impaired when performed on a decline ($P < .001$): 14 of 17 patients could not perform a heel rise on a decline on the involved side, whereas only 1 patient could not perform a heel rise on a decline on the noninvolved side. Ability to toe walk and heel rise from a level surface was slightly impaired on the involved side ($P < .05$): 6 patients were unable to toe walk and 4 patients were unable to perform a heel rise on a level surface on the involved side. All patients could toe walk and perform a heel rise on a level surface on the noninvolved side. Heel rise performance was not impaired when performed on an incline ($P = .9$); all patients could perform a heel rise on an incline on both the involved and noninvolved sides.

DISCUSSION

The primary finding in this study was that patients had marked plantarflexor weakness in end-range plantar flexion after Achilles tendon repair. Of note, no weakness was apparent at the ankle angle at which peak plantarflexion torque occurred (20° or 10° of dorsiflexion). These findings were observed more than 6 months after surgery, and all patients had returned to full activities. Weakness in end-range plantar flexion was hypothesized based on previous observations that excessive tendon lengthening may occur during tendon remodeling after surgical repair.[13,15,16] Increased tendon length leads to greater muscle shortening during muscle contraction. With the ankle in plantar flexion, the muscle is already in a shortened position and below the angle for optimal force production. Therefore, based on the length-tension relationship, further muscle shortening due to tendon lengthening would decrease force production.

The results of the functional tests also indicated weakness in end-range plantar flexion. No impairments were evident with heel rise performance initiated from a dorsiflexed position (incline heel rise), and only 4 patients had impairments with a heel rise initiated from a neutral (standing heel rise) position. However, marked impairments were evident when patients attempted to initiate a heel rise from a plantarflexed position (decline heel rise). Performance tests involving multiple heel rises have previously been used to identify impairments after Achilles tendon repair[9,11,13]; however, a single heel rise initiated from a plantarflexed position was sufficient to identify impairments in 14 patients in this study. Although some patients had impairment in toe walking, this was a less sensitive test of functional strength impairment in plantar flexion.

An additional finding was decreased passive joint stiffness. Passive stiffness measured from 10° to 20° of dorsiflexion was 34% lower in the ankle that had an Achilles tendon repair. The measurement of passive stiffness using this method reflects the combination of resistance from the muscle tendon units crossing the ankle joint and the resistance from the joint and its ligamentous attachments. The muscle tendon units of the plantar flexors are presumed to primarily contribute to the measured resistance. Herbert et al[5] demonstrated that most of the strain imposed by passive dorsiflexion was absorbed by the tendon. Examination of their data on muscle tendon unit and medial gastrocnemius fascicle length changes[5] showed that the muscle tendon unit elongated by approximately 6 mm from 10° to 20° of dorsiflexion. Of this elongation, approximately 1.3 mm was absorbed by the fascicles and 4.3 mm by the tendon. Based on these findings,[5] the passive stiffness measurement in the present study likely involved significant tendon elongation; therefore, the results may reflect lower Achilles tendon stiffness after Achilles repair.

Increased passive or active dorsiflexion range of motion relative to the noninvolved side has been used as an indicator of excessive tendon lengthening after Achilles tendon repair.[9,13] Such an effect was not evident for passive dorsiflexion in this study. This is surprising given the lower passive joint stiffness on the involved side. However, determination of the end of range of motion with manual motion testing is subjective, and the stretching force applied by the tester is not standardized. It is possible that variation in the applied dorsiflexion force used by the tester to determine terminal dorsiflexion motion obscured a real difference. Furthermore, joint constraints may limit motion at terminal dorsiflexion sufficiently to mask a difference in the extensibility of the plantar flexors. Given these limitations, passive resistance to stretch may be a more sensitive measurement than is manual goniometric assessment of maximal range of motion. Alternatively, the fact that patients did not have increased passive dorsiflexion motion may indicate that the observed weakness in end-range plantar flexion was owing to increased tendon compliance during loading rather than a lengthened tendon at rest.

It was not possible to determine if the observed effects attributed to excessive tendon lengthening resulted from excessive tendon loading during rehabilitation. Of note, Mortensen et al[13] found that the separation of tendon ends during tendon healing was not affected by early postoperative motion. However, weightbearing would be more likely to have a detrimental effect on tendon healing. Rettig et al[17] recently reported reruptures in 4 of 24 patients (17%) 30 years or younger 7 to 10 weeks after Achilles tendon repair. All patients began partial weightbearing at 3 weeks

and were full weightbearing by a mean of 7 weeks after surgery. No reruptures occurred in 65 patients older than 30 years who followed the same rehabilitation program.[17] Considering that the healing Achilles tendons of 4 patients were exposed to forces sufficient to rupture the repairs, it is possible that, in general, repaired Achilles tendons may be exposed to forces sufficient to cause detrimental lengthening without actual rupture during aggressive rehabilitation.

Although weakness in end-range plantar flexion is attributed to excessive Achilles tendon lengthening, there may be other explanations for this effect. Other possible explanations include a disproportionate change in Achilles tendon moment arm in plantar flexion or selective plantar flexor inhibition in end-range plantar flexion. With respect to moment arm changes, the Achilles tendon moment arm increases by approximately 25% from 15° of dorsiflexion to 15° of plantar flexion.[10] If this increase in moment arm with plantar flexion were reduced after Achilles tendon repair, weakness relative to the normal contralateral side would be observed. However, it is difficult to conceive how the repair could have only altered the moment arm with the ankle in a plantarflexed positioned. All Achilles tendon repairs were midsubstance repairs, and because the tendon insertions were not involved, the line of pull of the Achilles should have been unaffected.

It is not possible to rule out selective inhibition of the plantar flexors in end-range plantar flexion as an explanation for the observed end-range plantarflexion weakness. A comparison of surface EMG activity between the involved and noninvolved limbs would have provided some clarification on this issue. However, there is no theoretical rationale for plantar flexor inhibition isolated to end-range plantar flexion after Achilles repair. The findings of end-range plantarflexion weakness, a decrease in passive dorsiflexion stiffness, and well-documented studies of tendon separation during healing[13,15,16] led us to believe that tendon lengthening during contraction may be contributing to end-range weakness. Future studies using ultrasonic imaging could examine Achilles tendon length changes during plantarflexion contractions.

The functional implications of weakness in end-range plantar flexion are evident in the inability of many patients to perform a heel rise from a plantarflexed position. This impairment would affect descending stairs, walking or running downhill, or landing from a jump. A decrease in the ability of the plantar flexors to absorb impact forces when landing from a jump would increase the load on the bony structures of the ankle and increase the demand on the knee joint. Such an effect would have significant performance implications in sports such as basketball, volleyball, and gymnastics, in which correct landing is critical, and warrants further examination. Strength testing in end-range plantar flexion and functional tests in plantar flexion, such as the decline heel rise, may be useful additions to a patient's clinical evaluation before returning to sports after Achilles repair.

It remains to be determined if end-range plantarflexion weakness can be avoided after Achilles tendon repair. If end-range plantarflexion weakness were owing to a lengthened or more compliant Achilles tendon, 2 preventive strategies would be to (1) use stronger repair techniques and (2) decrease postoperative Achilles tendon stress. Epitendinous augmentation of 4-strand modified Kessler suture repairs has been shown to increase the failure force of the repair from 164 to 323 N.[8] The authors of this article now use epitendinous augmentation for all Achilles repairs. Keeping all patients in a cam walker until at least 8 weeks and maintaining a heel lift in the patient's shoe for at least 1 month after the cam walker is discontinued may also be beneficial interventions. It is common practice to perform Achilles (gastrocnemius-soleus complex) stretches to mobilize the joint during tendon healing. The authors of this article allow early motion in plantar flexion but now do not emphasize Achilles stretching in rehabilitation in an attempt to avoid excessive elongation and plastic deformation of the repaired Achilles tendon. This stretching is often avoided up until 12 weeks postoperatively, as previous studies have shown that most elongation occurs in the first 12 weeks after Achilles repair.[14,16]

It also remains to be determined if end-range plantarflexion weakness can be reversed. It has long been known that immobilization of muscles in a shortened position leads to a reduction in serial sarcomeres with an associated leftward shift in the length-tension curve (increased force at short lengths – decreased force at long lengths). The addition of intermittent muscle stimulation during immobilization does not reverse the serial sarcomere loss.[21] Therefore, relative plantar flexor shortening by inserting a heel lift in the shoe, combined with aggressive end-range plantarflexion strengthening, may be beneficial. However, any beneficial length-tension–mediated changes would presumably be easily reversed with return to normal activities.

In conclusion, disproportionate weakness in end-range plantar flexion (shortened muscle), decreased passive joint stiffness in dorsiflexion, and the inability to perform a heel rise when initiated from a plantarflexed position were apparent in patients who had returned to full activities after Achilles tendon repair. These impairments will have important functional implications for activities such as descending stairs and landing from a jump. Weakness in end-range plantar flexion may be an unrecognized problem after Achilles tendon repair.

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