INTRODUCTION

Achilles tendon ruptures are life altering injuries which lead to short term physical deficits and potential long term decreases in strength and physical activity level. Plantar flexion weakness at the end range of motion is still observed more than a year after surgery and the cause of remaining deficits and the optimal treatment for Achilles rupture is currently under debate. A solid understanding of the physiologic reaction to a complete rupture and the anatomical variation caused by surgical repair is needed to improve current rehabilitation. The purpose of this study was to relate the lengthening of the Achilles tendon post rupture to muscle activation patterns during walking to discern whether remaining gait abnormalities are due to physical alteration or neural inhibition.

METHODS

EMG data was collected at 1200hz from the medial and lateral gastrocnemius (MG, LG), soleus (SL), and tibialis anterior (TA) muscles during walking from 4 subjects, with an Achilles tendon rupture, at 6 and 12 months post-repair along with 5 healthy control subjects (age: 26.2 ± 6.4yrs, height: 1.76±0.1m, weight: 81.44±20.6kg) using a MA-300 system (Motion Lab Systems, Baton Rouge, LA). Each subject signed an inform constant prior to testing. Bipolar silver/silver chlorides EMG surface electrodes were placed on each of the muscles. Peak EMG values were determined in Visual 3D (C-Motion Inc., Bethesda, MD) by creating a linear envelope using low and high pass filtering, DC offset removal, and full wave rectification. EMG data was normalized to the maximum EMG found during maximum isometric testing.

The Achilles tendon length (ATL) of the injured and uninjured side was measured using a combination of B-mode ultrasound imaging at 10 MHz with a 60mm transducer using a ProSound SSD-5000 (Aloka, Tokyo, Japan) and motion analysis using an 8-camera motion capture system (Qualisys Motion Capture System, Gothenburg, Sweden) collecting motion at 50hz. The tendon was scanned between the calcaneal osteotendinous junction and the musculotendinous junction. The combination of motion capture and the outputted ultrasound files allowed for the length of the Achilles tendon to be determined. Test-retest reliability of this method has found an ICC=0.97 with no significant differences (p=0.889) between the two test occasions.

RESULTS AND DISCUSSION

The Achilles tendon of those patients injured had a significantly increased (p=0.05) ATL compared to the uninjured side at both the 6 and 12 month evaluation with an increase of 3.56±0.75cm and 3.12±0.96cm, respectively (Table 1). There was no significant difference (p=0.05) between the right and the left side in the healthy control group. Six (6) months post-surgery, the peak normalized EMG of the LG during walking on the injured side (0.95) was significantly increased (p=0.05) compared to the EMG of the unaffected side (0.81). The activity of the MG during walking at 12 months was also significantly larger on the injured side (0.91) compared to the uninjured side (0.72). Overall, there was general increase of muscle activity on the injured side of the triceps surae muscles at 6 months post-surgery and an even greater increase of activity at 12 months (Figure 1). Conversely, there was no change in EMG activity of the TA at 6 or 12 months. The healthy controls showed no difference of EMG activity between legs.
The increased muscle activity seen at 6 and 12 months post-surgery indicate, following an Achilles tendon rupture, the calf muscles are not inhibited during gait. Since these patients all had a significantly longer Achilles tendon on the injured side, the gastrocnemii and soleus muscles could instead be contracting more in an attempt to compensate for the increase in tendon slack during walking. The healthy controls had no ATL difference between sides and showed no disparity in peak EMG.

**CONCLUSIONS**

The increased ATL and EMG signals from the muscles used to stretch the tendon indicate that loss of functionality is primarily caused by anatomical changes in the tendon and the appearance of muscle weakness is due to force transmission from the inhibition. During gait, patients’ compensate for an elongated Achilles tendon following surgical repair by greater activation of the gastrocnemii muscles to account for the additional tendon slack before motion is produced at the joint.

**REFERENCES**


**ACKNOWLEDGEMENTS**

The authors would like to acknowledge the Swedish Research Council for its financial support.

**Table 1.** Mean Achilles tendon lengths of subjects at 6 and 12 months post-Achilles rupture repair and healthy controls. Note: The injured side of the subjects post-surgery is significantly larger than the unaffected side while there is no difference between the left and right sides of the healthy control with the differences in bold.

<table>
<thead>
<tr>
<th></th>
<th>6 Months Injured</th>
<th>6 Months Uninjured</th>
<th>Difference at 6 Months</th>
<th>12 Months Injured</th>
<th>12 Months Uninjured</th>
<th>Difference at 12 Months</th>
<th>Healthy Left</th>
<th>Healthy Right</th>
<th>Difference in Healthy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Achilles Length (cm)</td>
<td>24.46</td>
<td>20.90</td>
<td>3.56</td>
<td>23.83</td>
<td>20.72</td>
<td>3.12</td>
<td>20.33</td>
<td>19.77</td>
<td>0.56</td>
</tr>
</tbody>
</table>

**Figure 1.** The averaged normalized EMG from the lower leg during one gait cycle of walking from 4 Achilles repaired subjects. The injured limb is graphed in red and the unaffected limb in black for each of the muscles. The astrix (*) represents a significant difference in peak EMG magnitude between limbs.