Relationships Between Biomechanics, Tendon Pathology, and Function in Individuals With Lateral Epicondylitis

Lateral epicondylitis (LE) is a prevalent and costly musculoskeletal disorder of the common extensor tendon, characterized by degeneration of the tendon and frequently reported pain at the lateral aspect of the elbow.\[^{1,2,5}\] In addition, biomechanical and sensorimotor deficits can occur and adversely impact upper extremity function.\[^{4,5}\] These functional deficits may interfere with occupational tasks and activities of daily living, resulting in significant individual and occupational costs.\[^{16,45}\]

Because the pathophysiology of LE is not well understood, treatment remains challenging, and LE is prone to recurrence.\[^{1,2,3}\]

Tendon changes due to LE include dense populations of fibroblasts, vascular hyperplasia, and disorganized collagen.\[^{10,39,43}\] The common extensor tendon origin in individuals with LE is usually thickened and shows increased signal intensity on magnetic resonance imaging (MRI). The region of greatest signal abnormality is usually at the origin of the extensor carpi radialis brevis tendon from the lateral epicondyle of the humerus. The areas of increased signal intensity within the diseased tendon usually correspond to areas of mucoid degeneration and neovascularization on histopathologic analysis.\[^{27,43,44}\] Ultrasound also has been used to study LE, and findings include the presence of intratendinous calcification, tendon thickening, adjacent bone irregularity, focal hypoechoic regions, and diffuse heterogeneity of the common extensor tendon.\[^{3,10,31}\]

**STUDY DESIGN:** Single-cohort descriptive and correlational study.

**OBJECTIVES:** To investigate the relationships between tendon pathology, biomechanical measures, and self-reported pain and function in individuals with chronic lateral epicondylitis.

**BACKGROUND:** Lateral epicondylitis has a multifactorial etiology and its pathophysiology is not well understood. Consequently, treatment remains challenging, and lateral epicondylitis is prone to recurrence. While tendon pathology, pain system changes, and motor impairments due to lateral epicondylitis are considered related, their relationships have not been thoroughly investigated.

**METHODS:** Twenty-six participants with either unilateral (n = 11) or bilateral (n = 15) chronic lateral epicondylitis participated in this study. Biomechanical measures (grip strength, rate of force development, and electromechanical delay) and measures of tendon pathology (magnetic resonance imaging and ultrasound) and self-reported pain and function (Patient-Rated Tennis Elbow Evaluation) were performed. Partial Spearman correlations, adjusting for covariates (age, gender, weight, and height), were used to evaluate the relationship between self-reported pain, function, and biomechanical and tendon pathology measures.

**RESULTS:** Statistically significant correlations between biomechanical measures and the Patient-Rated Tennis Elbow Evaluation ranged in magnitude from 0.44 to 0.68 (P < .05); however, no significant correlation was observed between tendon pathology (magnetic resonance imaging and ultrasound) measures and the Patient-Rated Tennis Elbow Evaluation (r = -.02 to 0.31, P > .05). Rate of force development had a stronger correlation (r = 0.54-0.68, P < .05) with self-reported function score than with grip strength (r = 0.35-0.47, P < .05) or electromechanical delay (r = 0.5, P < .05).

**CONCLUSION:** Biomechanical measures (pain-free grip strength, rate of force development, electromechanical delay) have the potential to be used as outcome measures to monitor progress in lateral epicondylitis. In comparison, the imaging measures (magnetic resonance imaging and ultrasound) were useful for visualizing the pathophysiology of lateral epicondylitis. However, the severity of the pathophysiology was not related to pain and function, indicating that imaging measures may not provide the best clinical assessment. J Orthop Sports Phys Ther 2013;43(6):368-378. Epub 18 March 2013. doi:10.2519/jospt.2013.4411

**KEY WORDS:** grip strength, hand function, rate of force development, tennis elbow

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Pain is the primary symptom of LE. The pain that patients with LE experience may be due to changes in the nervous system, as a result of neuronal tissue changes as well as nociceptive and non-nociceptive processes. In patients with LE, pain can be assessed using pressure pain threshold and self-reported measures such as the visual analog scale and the Patient-Rated Tennis Elbow Evaluation (PRTEE). The PRTEE is a commonly used, valid, reliable, and sensitive clinical instrument for assessment of pain and disability in individuals with chronic LE.

Pain-free grip strength is the most commonly assessed motor impairment, and recently the effects of LE on reaction time, rate of force development, and electromechanical delay have been investigated. Rate of force development is considered to be a measure of the ability to rapidly generate strength and is associated with higher functional performance. Electromechanical delay represents the duration of the excitation contraction coupling in the muscle and the time to take up the slack in the elastic structures of the muscle-tendon unit. Chourasia et al found a lower rate of force development and longer electromechanical delay in individuals with LE compared to controls. In addition, Bisset et al reported longer reaction times in individuals with LE compared to controls.

While tendon pathology, pain system changes, and motor impairments due to LE are considered related, their relationship has not been investigated thoroughly. For example, Clarke et al observed a positive association between ultrasound findings and improvements in self-reported pain and function, but changes in motor performance were not evaluated. Similarly, Bisset et al investigated the effects of various interventions for LE on global improvement, grip strength, and sensorimotor measures but did not assess morphological changes in the common extensor tendon.

It is noteworthy that the majority of studies on LE only include patients with LE of the dominant arm and exclude those with bilateral LE or unilateral LE of the nondominant arm. It is important that the relationships between the various components of LE be investigated in a heterogeneous sample. This may help to gain a better understanding of LE to improve assessment and treatment outcomes.

The overall objective of the study was to evaluate the relationships between self-reported pain and function (PRTEE) and measures of tendon pathology and biomechanics in individuals with LE. Secondary analyses evaluated the relationships between biomechanical and tendon pathology measures. This information may help provide a better understanding of the effect of LE on function and its association with biomechanical measures.

**METHODS**

**Participants**

Twenty-nine patients with LE were recruited and enrolled from various outpatient clinics in Madison, WI from June 2009 to February 2010. These patients were participating in a therapeutic trial investigating the efficacy of prolotherapy for LE. Diagnostic criteria for LE included the presence of lateral elbow pain for more than 3 months, tenderness to palpation over the lateral epicondyle and/or extensor mechanism, and pain present on at least 2 of the following provocation tests: resisted extension of the wrist or fingers, resisted supination, and passive stretch of the wrist extensors or supinator muscle. Exclusion criteria consisted of coexisting or previous medical history of rheumatoid or inflammatory arthritis, chronic pain diagnoses, diabetes mellitus, pregnancy, systemic nervous disease, neuropathy, or acute trauma to the fingers or hands. Additional exclusion criteria were prior upper extremity injury, concurrent upper extremity injury, unresolved litigation, and comorbidities that could interfere with the ability to participate in the study. Two participants were excluded because they reported concurrent upper extremity injury. Data from 1 participant were excluded because of instrumentation malfunction. Of the 26 eligible participants, 11 had unilateral symptoms and 15 had bilateral symptoms (Table 1).

Informed consent was obtained for all participants in this study, in accordance with the study protocol, as approved by the University of Wisconsin-Madison Health Sciences Institutional Review Board.

**Self-Reported Measures**

**PRTEE Questionnaire** Participants completed the PRTEE, a condition-specific questionnaire that assesses both elbow pain and function. The PRTEE has good test-retest reliability, although reliability is less for patients with work-related LE (intraclass correlation coefficient [ICC] = 0.80) versus non–work-related LE (ICC = 0.94). It has also been used to determine the effects of different interventions for LE.

The PRTEE consists of a 5-item pain subscale and a 10-item function subscale. Each subscale is scored from 0 to 50, with 0 being the best score and 50 the worst score. The PRTEE composite score is the sum of the pain and function subscales and ranges from 0 to 100, with 0 being the best score and 100 the worst score.

**Visual Analog Scale** All participants were asked to rate the average lateral elbow pain intensity for the previous week using a 10-cm line ranging from zero (no pain) to 10 (most pain).

**Biomechanical Measures**

Biomechanical measures included pain-free grip strength, rate of force development, and electromechanical delay. These measures were collected bilaterally, and the results have been reported elsewhere. Only the results from the affected arm were used for the correlational analyses reported in this paper. In cases of bilaterally affected participants, the results from the more affected arm,
as determined by the visual analog scale scores, were used. When both arms were equally affected, the results from the dominant arm were used.

**Pain-Free Grip Strength** Pain-free grip strength is a valid and reliable (ICC = 0.97) measure and is commonly used in research and clinical assessment of LE.²⁻⁴⁻⁵⁻⁶⁻⁹⁻¹³⁻¹⁴ For testing, as recommended for evaluation of grip strength in patients with LE,¹⁴⁻¹⁵ the participants were seated in a chair with the elbow in an extended position. The participants were then instructed to squeeze the Baseline (Fabrication Enterprises, Inc, White Plains, NY) dynamometer for 5 seconds, avoiding discomfort. Three replications with a 60-second interval between trials were performed. The average of the 3 replications was used as the pain-free grip strength.

**Rate of Force Development** The multiaxis profile (MAP) dynamometer was used for measurement of rate of force development.²⁴ The MAP dynamometer has demonstrated excellent test-retest reliability (ICC = 0.99) and excellent concurrent validity when compared to the Baseline dynamometer (r = 0.88-0.90).²⁴ When both arms were equally affected, the results from the dominant arm were used.

The participants were seated in a chair with the elbow in an extended position. Upon receipt of a randomly timed visual stimulus, participants were instructed to squeeze the handle as quickly and as hard as possible without pain for 5 seconds.⁷ The rate of force development was calculated by taking the time derivative of the force signal (FIGURE 1).¹ Rate of force development was measured at 30, 50, and 100 milliseconds from the onset of contraction. The onset of contraction was defined as a rise of 1 N from baseline level.³⁰ Peak rate of force development was also measured. This method is consistent with that of others for evaluating force development.¹² Three repetitions, with a 60-second interval between them, were performed, and the average of the 3 repetitions was used for data analysis. The signals from the MAP dynamometer were sampled at a rate of 1000 samples per second, using a USB-6009 card (National Instruments Corporation, Austin, TX).

The MAP dynamometer also provides a measure of pain-free grip strength, but, due to a different handle geometry compared to the Baseline dynamometer, the grip strength values are different.²⁴ We reported both grip strength values using the variable names "pain-free grip strength-Baseline" and "pain-free grip strength-MAP."

**Electromechanical Delay** Electromechanical delay was measured using the MAP dynamometer force output and the raw electromyographic (EMG) signal from the extensor carpi radialis muscle. EMG signal was measured using a TeleMyo 2400 EMG system (Noraxon USA Inc, Scottsdale, AZ). Electrodes were placed on the extensor carpi radialis at one third of the distance from the proximal end of a line from the lateral epicondyle to the distal end of the radius.²⁶ Electrodes were placed on the extensor carpi radialis with the forearm in neutral position. The reference EMG electrode
Activation was defined as a deviation of samples per second. The onset of muscle signals were sampled at the rate of 1500. The input impedance much greater than 100 MΩ.

The EMG amplifier had a gain of 1000, an input impedance much greater than 100 Ω, and a common-mode rejection ratio greater than 100 dB. The signals were sampled at the rate of 1500 samples per second. The onset of muscle activation was defined as a deviation of ±15 μV in the EMG signal from resting baseline level.

The time between the onset of muscle activation based on the change in EMG signal intensity and the onset of force as measured with the MAP dynamometer is considered the electromechanical delay (FIGURE 1). Sufficient practice was provided to all participants to become comfortable with the testing procedures prior to completing 3 repetitions. The average of the 3 repetitions was used for data analysis.

Tendon Pathology Measures

Tendon pathology was assessed using MRI and ultrasound. For those with unilateral LE, imaging was conducted on the affected arm. For individuals with bilateral LE, ultrasound was conducted on the most affected arm (as determined by the visual analog scale scores) and MRI was conducted bilaterally.

Magnetic Resonance Imaging

The MRI examination was performed using an ARTOSCAN (Esaote SpA, Genoa, Italy) 0.17-T extremity scanner. Axial and coronal intermediate-weighted fast spin-echo (FSE) and short tau inversion recovery (STIR) sequences of the elbow were used for semiquantitative assessment of disease severity (FIGURE 2). Intermediate-weighted FSE scan parameters were as follows: repetition time, 2050 milliseconds; echo time, 18 milliseconds; slices, 7; gap, 1.0 mm; thickness, 3.5 mm; readout field of view, 180; encoding field of view, 180; samples, 192; encoding number, 192. A semiquantitative grading scale was used to estimate the severity of chronic degeneration and pathologic changes in the common extensor tendon origin. The grading scale is as follows:

- Grade 0: normal common extensor tendon that is of uniform low signal intensity on intermediate-weighted FSE and fat-suppressed, T2-weighted STIR images.
- Grade 1: common extensor tendon with mild tendinopathy that is thickened and has intermediate signal intensity on intermediate-weighted FSE and STIR images.
- Grade 2: common extensor tendon with moderate tendinopathy that is thickened and shows focal areas of intense fluid-like signal intensity on STIR images, which comprise less than 50% of the total cross-sectional diameter of the tendon.
- Grade 3: common extensor tendon with severe tendinopathy that is thickened and shows focal areas of intense fluid-like signal intensity on STIR images, which comprise more than 50% of the total cross-sectional diameter of the tendon.

Ultrasound

All ultrasound exams were performed at the University of Wisconsin Sports Clinic using an iU22 xMATRIX system (Philips Healthcare, Andover, MA). A fellowship-trained musculoskeletal radiologist with 5 years of ultrasound experience performed all diagnostic exams. Diagnostic ultrasound images were obtained with the patient in the seated position and the elbow resting on a table at 90° of flexion. Ultrasound images were obtained of the common extensor tendon origin in orthogonal planes, long and short axis. All images were recorded in the radiology picture-archiving and communication system. Ultrasound-based diagnostic features of LE included thickening of the common extensor tendon, focal hypoechoic regions with tissue heterogeneity, neovascularity (“neovessels”), and intrasubstance clefts or calcification (FIGURES 2).
Grading of LE-related structural elbow scales allow a semiquantitative severity of hypoechogenicity. These 2 scales allow focal hypoechogenicity; and grade 3, severe diffuse hypoechogenicity. These 2 scales allow a semiquantitative severity grading of LE-related structural elbow changes.

Statistical Analysis
Spearman correlation coefficients were calculated to detect possible monotonic relationships between the PRTEE scores (pain, function, and composite) and the biomechanical (grip strength, rate of force development, and electromechanical delay) and imaging (MRI and ultrasound) measures. Both full (usual) and partial correlations, adjusting for baseline covariates (age, gender, weight, and height), were calculated. For secondary analyses, full and partial Spearman correlation coefficients were calculated among and between biomechanical and imaging measures. P values were calculated without adjustment for multiplicity. Data analysis was conducted using the R language and environment for statistical computing (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

The descriptive statistics for the self-report, biomechanical, and tendon pathology measures are presented in Table 2.

PRTEE Questionnaire
The mean ± SD values for the PRTEE composite, pain, and function scores were 44.3 ± 18.8, 23.9 ± 8.3, and 20.4 ± 11.4, respectively. Figure 5 shows the relationship between the PRTEE function and pain component scores, with a correlation coefficient of 0.76 (P < .01) and a partial correlation coefficient of 0.73 (P < .01).

PRTEE and Biomechanical Measures
Table 3 shows full and partial Spearman correlation coefficients of PRTEE components and composite scores with biomechanical measures. Partial Spearman correlation coefficients adjusted for covariates are reported in the text.

All partial correlation coefficients were in the expected directions, with negative correlation coefficients indicating that higher PRTEE scores were associated with lower grip strength and rate of force development. Positive partial correlation coefficients between PRTEE scores and electromechanical delay indicated that higher PRTEE scores were associated with higher electromechanical delay. Nineteen of 21 partial correlation coefficients were found to be nominally statistically significant (P < .05). Statistically significant partial correlation coefficients between biomechanical and PRTEE measures ranged in magnitude from 0.44 to 0.68 (P < .05), with the greatest partial correlation coefficient observed between rate of force development at 100 milliseconds and PRTEE composite score (r = -0.68, P < .01). Figure 6 shows the relationship between the PRTEE composite score and peak rate of force development.

PRTEE and Imaging Parameters
Correlation coefficients between the imaging parameters and PRTEE components and composite scores are shown in Table 4. None of the 12 partial correlation coefficients was found to be statistically significant. Figure 7 shows the distribution of PRTEE pain and function scores by MRI score.

Secondary Associations
Biomechanical Measures
Higher grip strength was found to be associated with higher rate of force development (Table 5). All 3 partial correlation coefficients among biomechanical measures were found to be statistically significant (P < .01). The greatest correlation occurred between pain-free grip strength-MAP and pain-free grip strength-Baseline (r = 0.74, P < .01). Relationships of pain-free grip strength measures with rate of force development at 30, 50, and 100 milliseconds (not shown) were similar to the relationships observed with peak rate of force development.

Imaging Measures
Only 2 of 6 partial correlation coefficients were found to be nominally significant (P < .05) (Table 6). The strongest correlation was that observed between neovascularity and MRI score (r = 0.55, P < .01).
Biomechanical and Imaging Measures
There was no statistically significant association between the biomechanical and the ultrasound imaging measures, and none of the 12 partial correlation coefficients were nominally statistically significant (Table 7). MRI score was found to be consistently negatively associated with biomechanical measures, and all 3 partial correlation coefficients were nominally statistically significant ($P < .05$), including a strong observed negative partial correlation coefficient ($r = -0.72, P < .01$) between MRI score and MAP grip strength.

DISCUSSION

We found that the biomechanical measures of grip strength and rate of force development were associated with measurements of self-report pain and function as assessed by the PRTEE. No statistically significant association was observed between imaging measures (ultrasound and MRI) and measurement of self-report pain and function as assessed by the PRTEE.

The PRTEE was previously reported to have significant but low association with pain-free grip strength ($r = 0.35-0.40, P < .01$). It was hypothesized that maximal grip strength may not be required for function. Instead, alternative biomechanical measures, such as the rate of force development or submaximal strength, may be more important for function and thus may have a stronger correlation with PRTEE scores.

Although we did not measure submaximal strength, our results suggest that the rate of force development may have a greater role in determining function in patients with LE than maximal grip strength. In the current study, we found that rate of force development was highly correlated with the PRTEE, and it had a higher correlation with the PRTEE than pain-free grip strength. This is consistent with other studies in which the rate of force development was associated with higher functional performance for the upper extremity as well as the lower extremity. Andersen et al. found that rate of force development had a stronger association with self-reported pain than maximal strength. In our prior research, we found that rate of force development was significantly reduced in those with LE compared to matched controls. To perform activities of daily living, a threshold level of strength is required. Greater strength beyond the threshold alone may not necessarily im-

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th><strong>Descriptive Statistics for Self-Report, Biomechanical, and Imaging Measures (n = 26)</strong></th>
</tr>
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<tbody>
<tr>
<td><strong>Value</strong></td>
<td></td>
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<tr>
<td><strong>Self-report pain and function scores</strong></td>
<td>Visual analog scale (0-10) 5.9 ± 1.7 (4.0-10.0)</td>
</tr>
<tr>
<td><strong>Biomechanical measures</strong></td>
<td>RFD at 30 ms, N/s 393 ± 239 (48-806)</td>
</tr>
<tr>
<td></td>
<td>RFD at 100 ms, N/s 554 ± 336 (44-1072)</td>
</tr>
<tr>
<td></td>
<td>Pain-free grip strength-Baseline, N 208 ± 136 (40-536)</td>
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<tr>
<td></td>
<td>EMD, s 0.065 ± 0.031 (0.04-0.16)</td>
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<tr>
<td>Ultrasound hypoechogenicity, n‡ Grade 0 7</td>
<td></td>
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<tr>
<td></td>
<td>Grade 1 7</td>
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<tr>
<td></td>
<td>Grade 2 8</td>
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<tr>
<td></td>
<td>Grade 3 1</td>
</tr>
<tr>
<td>Ultrasound neovascularity, n‡ Grade 0 12</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 1 7</td>
</tr>
<tr>
<td></td>
<td>Grade 2 4</td>
</tr>
<tr>
<td></td>
<td>Grade 3 0</td>
</tr>
</tbody>
</table>
| Ultrasound tendon thickness, mm*‡ 5.8 ± 0.8 (4.2-7.1) | Abbreviations: EMD, electromechanical delay; MAP, multiaxis profile dynamometer; MRI, magnetic resonance imaging; PRTEE, Patient-Rated Tennis Elbow Evaluation; RFD, rate of force development.

*Values are mean ± SD (range).
†1 participant declined MRI.
‡3 participants did not complete ultrasound assessment.
TABLE 3

<table>
<thead>
<tr>
<th>Measure</th>
<th>Full Correlation Coefficient</th>
<th>Partial Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PRTEE Pain</td>
<td>PRTEE Function</td>
</tr>
<tr>
<td>Pain-free grip strength-Baseline</td>
<td>-0.36</td>
<td>-0.48*</td>
</tr>
<tr>
<td>Pain-free grip strength-MAP</td>
<td>-0.49*</td>
<td>-0.44*</td>
</tr>
<tr>
<td>RFD at 30 ms</td>
<td>-0.55*</td>
<td>-0.57*</td>
</tr>
<tr>
<td>RFD at 50 ms</td>
<td>-0.57*</td>
<td>-0.59*</td>
</tr>
<tr>
<td>RFD at 100 ms</td>
<td>-0.64*</td>
<td>-0.65*</td>
</tr>
<tr>
<td>Peak RFD</td>
<td>-0.60*</td>
<td>-0.63*</td>
</tr>
<tr>
<td>Electromechanical delay</td>
<td>0.22</td>
<td>0.43*</td>
</tr>
</tbody>
</table>

Abbreviations: MAP, multiaxis profile dynamometer; PRTEE, Patient-Rated Tennis Elbow Evaluation; RFD, rate of force development.

*P<.05.

†P<.01.

These findings may be relevant for physical therapy interventions for LE. Resistance training activities for the forearm muscles as well as grip-strengthening activities are common physical therapy interventions, with improvements in grip strength often used as an outcome measure in clinical research.5,41

While a strong association exists between grip strength and rate of force development, exercises that address deficits in grip strength may not necessarily address the deficits in rate of force development. The ability to rapidly produce force is most affected by exercises that incorporate a velocity-dependent component and not solely resistive strengthening.6,37,31

Although the effects of velocity-dependent training in LE have not been specifically studied, a number of other studies have found improvements in rate of force development for other muscle-tendon groups following interventions that include velocity-dependent training. Bottaro et al6 reported significant increases in arm and leg muscular power and functional performance for older men following 10 weeks of high-velocity power training compared to no increase in muscular power following resistance training. Fielding et al17 reported similar results for older women. Hakkinen and Komi23 also reported significant increases in rate of force development, but not in maximal strength, for knee extension following explosive-type strength training. Conversely, Hakkinen and Komi23 observed significant increases in knee extension strength, but not in rate of force development, following resistance training and suggested that specific training-induced adaptations in the neuromuscular system may be responsible for these changes in performance. Neuromuscular training may also help in addressing the deficits in reaction time. Linford et al34 found that a neuromuscular training program consisting of sensorimotor, strength, and power components for the lower extremity resulted in a significant decrease in reaction time, with a trend toward an increase in electromechanical delay. Grosset et al30 found that for the lower extremity, 10 weeks of plyometric training caused an increase
Miller et al. reported that ultrasound and MRI for diagnosing LE. Previous studies have found differences in the sensitivity and specificity of ultrasound and MRI for diagnosing LE. Miller et al. reported that ultrasound had 64% to 82% sensitivity and 67% to 100% specificity and MRI had 90% to 100% sensitivity and 83% to 100% specificity to diagnose LE, and suggested that sonography might be as specific but not as sensitive as MRI for this purpose. Sonography might have contributed to the lack of significant association.

The partial correlations of MRI score with biomechanical measures are noteworthy, particularly the correlation of MRI score to MAP grip strength. It should be noted, however, that none of the full correlation coefficients was nominally statistically significant, and our data suggest that the interrelationship between MRI score, biomechanical parameters, and our selected covariates may be complex. Study of these associations in a larger sample is needed.

Our research, along with that of others, found that the severity of ultrasound and MRI findings for LE does not correlate with clinical symptom severity and function. Overall, our results suggest that use of imaging measures with ordinal scales may be best suited for diagnosis of disease rather than for assessment of subtle differences in disease severity.

**Limitations**

Participants in this study had chronic LE, and it is possible that over time they might have developed adaptive motor patterns to adjust for functioning with LE, whereas individuals with acute LE might not have developed adaptive motor patterns. Also, the severity of LE observed on imaging measures may not be

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**TABLE 4**

<table>
<thead>
<tr>
<th>Measure</th>
<th>PRTEE Pain Correlation Coefficient</th>
<th>PRTEE Function Correlation Coefficient</th>
<th>PRTEE Composite Correlation Coefficient</th>
<th>PRTEE Pain Partial Correlation Coefficient</th>
<th>PRTEE Function Partial Correlation Coefficient</th>
<th>PRTEE Composite Partial Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ultrasound thickness</td>
<td>0.44*</td>
<td>0.22</td>
<td>0.26</td>
<td>0.28</td>
<td>0.16</td>
<td>0.17</td>
</tr>
<tr>
<td>Ultrasound neovascularity</td>
<td>0.14</td>
<td>0.17</td>
<td>0.13</td>
<td>-0.02</td>
<td>0.10</td>
<td>0.03</td>
</tr>
<tr>
<td>Ultrasound hypoechogenicity</td>
<td>0.25</td>
<td>0.31</td>
<td>0.26</td>
<td>0.26</td>
<td>0.31</td>
<td>0.29</td>
</tr>
<tr>
<td>MRI score</td>
<td>0.45*</td>
<td>0.40</td>
<td>0.40</td>
<td>0.31</td>
<td>0.31</td>
<td>0.27</td>
</tr>
</tbody>
</table>

*P<.05.*

**FIGURE 7.** Box plots of PRTEE pain (A) and function scores (B) versus MRI scores. Scale used for MRI scores: grade 1, mild tendinopathy; grade 2, moderate tendinopathy; grade 3, severe tendinopathy. The median is the dark band in the box. The bottom and top of the box represent the 25th and 75th percentile, respectively. The whiskers extend to the most extreme data point, which is no more than 1.5 times the interquartile range (25th-75th percentile) from the box. Points beyond the whiskers represent the outliers. Abbreviations: MRI, magnetic resonance imaging; PRTEE, Patient-Rated Tennis Elbow Evaluation.
as extreme in individuals with acute LE. Therefore, these results cannot be generalizable to patients with acute LE. Future studies involving a larger number of participants with varied duration of symptoms may help in further elucidating the relationship between biomechanics, tendon pathology, and function in individuals with LE.

Although the assessors were not blinded to group status, because all participants had LE, they were blinded to the results of the other outcome measures. A biostatistician not involved in data collection completed all data analysis. To minimize assessor bias during biomechanical measurement, a standard operating procedure was used. Interrater reliability for measurement of biomechanical variables (EMG and force onset) for 2 assessors was found to have an ICC of 0.99.

CONCLUSION

Biomechanical measures (pain-free grip strength, rate of force development, electromechanical delay) have the potential to be used as outcome measures to monitor progress in LE. In comparison, imaging measures (MRI and ultrasound) were useful for visualizing the pathophysiology of LE. However, the severity of the pathophysiology was not
related to pain and function, indicating that imaging measures may not provide the best clinical assessment.

**KEY POINTS**

**FINDINGS:** Rate of force development has greater association with function than maximal strength. Imaging measures of tendon pathology were not significantly associated with self-report symptoms.

**IMPLICATIONS:** Physical therapy interventions that include velocity-dependent training may result in improvements in rate of force development and function.

**CAUTION:** Study participants had chronic LE. Results of the study may not be generalizable for individuals with acute LE.

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