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Original Article

Lateral epicondylalgia exhibits adaptive muscle activation strategies based on wrist posture and levels of grip force: a case-control study

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Abstract

Objectives: To investigate forearm muscle activity in individuals with lateral epicondylalgia (LE) when gripping at different wrist postures, and investigate the association between muscle activity and clinical characteristics of LE. **Methods:** Eleven LE and 11 healthy participants performed isometric handgrips at 15% and 30% of maximum grip force (MVC). Gripping was performed in wrist extension, wrist flexion, and wrist neutral. Surface electromyography was collected from six forearm muscles. Standard clinical and tendon structural measures for LE were obtained. **Results:** LE group had reduced magnitude of extensor carpi radialis brevis (ECRB) with increased contribution of extensor carpi ulnaris (ECU) during 15% MVC. However, during 30% MVC the LE group had reduced flexor carpi radialis and flexor digitorum superficialis activity, which was coupled with increased contribution from extensor digitorum communis (EDC) and ECU. Although ECRB and ECU activity differed in wrist flexion compared to other wrist postures for controls, different wrist posture had no effect on forearm muscle activation in LE. Pain and disability, and tendon thickness had significant associations with EDC and ECRB activity respectively in LE. **Conclusion:** Individuals with LE use different neuromuscular strategies when gripping with different wrist postures which appears to be dependent on the level of grip force.

Keywords: Grip, Wrist Posture, Forearm Muscles, Motor Control

Introduction

Lateral Epicondylalgia (LE) is a degenerative condition characterised by increased thickness and tearing in the common extensor tendon[1], widespread pressure and thermal hyperalgesia[2,3], and the presence of neuromotor dysfunction- particularly of extensor carpi radialis brevis (ECRB)[4-9]. Arguably, the most disabling clinical feature of LE is pain during hand grip activities. This in turn can modify gripping behaviour, as individuals with LE often exhibit lower levels of force during gripping in order to prevent pain[10].

When healthy individuals perform power gripping, the largest grip forces are typically observed when the wrist is in ~35 degrees of extension[11]. This contrasts to the gripping characteristics of individuals with LE who grip with ~11° less wrist extension compared to controls[4]. The role of the wrist and finger extensor muscles in stabilising the wrist during gripping is well documented[12]. Although the neuromotor control of individual muscles, such as ECRB, have previously been examined[6,7,13], additional insight may be gained by assessing how other muscles of the forearm activate in the presence of ECRB dysfunction, particularly in relation to changes in wrist posture. It is likely that differences in wrist extensor and flexor muscle coordination may emerge with alternative wrist postures. In particular, as the kinematic and clinical manifestation of LE occurs about the flexion-extension axis of the wrist, LE-related differences in forearm muscle coordination may be evident when gripping in wrist flexion or wrist extension.

Therefore, the primary aim of this study was to examine the absolute magnitude and relative contribution of ECRB, extensor digitorum communis (EDC), extensor carpi ulnaris

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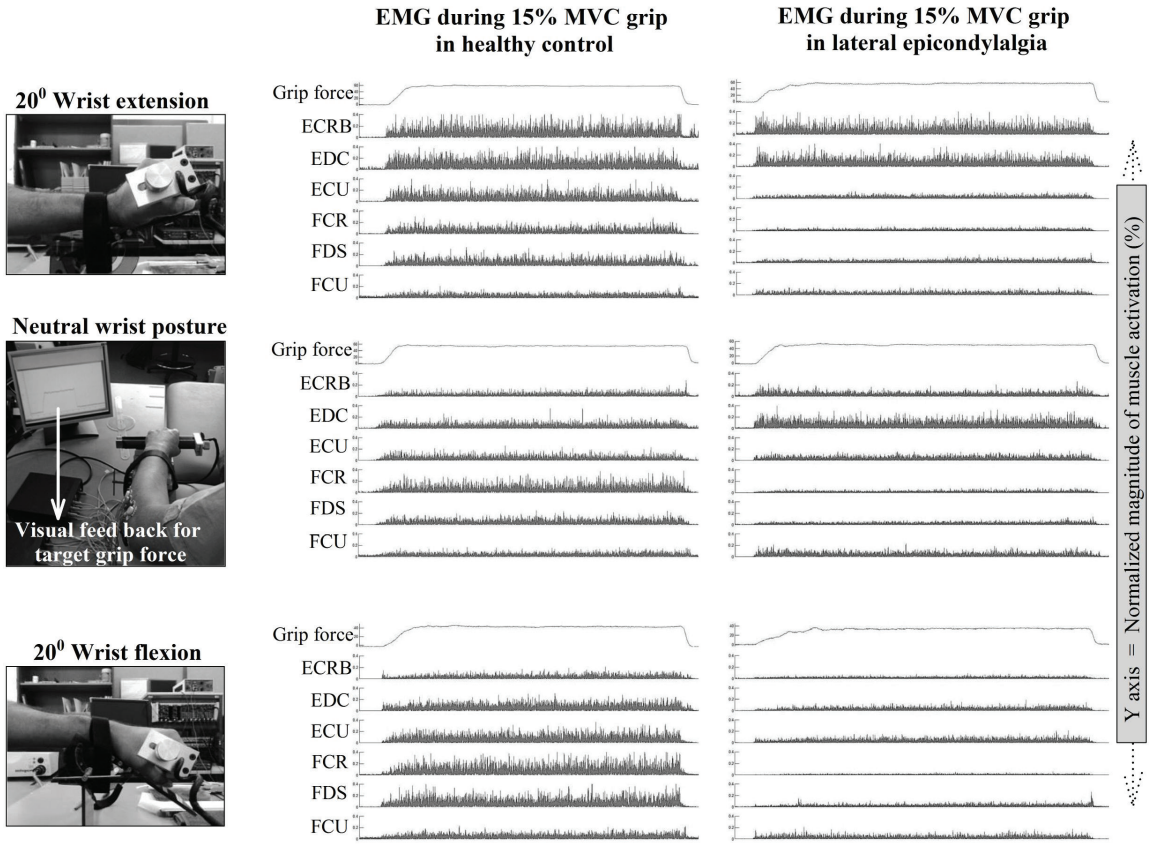

Figure 1. The experimental setup with wrist posture positioned in 20° wrist flexion, neutral, and 20° wrist extension. Visual feedback was provided on a PC monitor where a target line was presented at 15% or 30% maximal grip strength. Force and full-wave rectified EMG data in the right panels are representative of a single 15% grip trial from one individual with LE and a matched healthy control.

(ECU), flexor digitorum superficialis, (FDS), flexor carpi radialis (FCR), and flexor carpi ulnaris (FCU) in individuals with LE during isometric gripping performed in three different wrist postures. The secondary aim of this study was to determine the association between forearm muscle activation and clinical characteristics and common extensor tendon structural characteristics in people with LE. It was hypothesised that compared to healthy controls, the magnitude of ECRB activation and the relative contribution of ECRB to gripping would be significantly decreased regardless of wrist posture or the level of grip force in people with LE. It was also hypothesised that the reduced magnitude of ECRB activation and its relative contribution will be associated with increased pain, disability and altered tendon structural characteristics in people with LE.

Material and methods

Participants

Eleven individuals with LE (age range 32 to 65 years, 8 males, 10 right hand dominant) and eleven age-, sex- and limb-matched healthy controls were recruited from the general community. This was based on 80% power at the 0.05 significance level to achieve the minimum between-group effect size difference of 0.8 for ECRB muscle activation[8]. To be recruited into the LE group, participants needed to have a clinical diagnosis of LE confirmed by pain over the lateral epicondyle for greater than six weeks, which was provoked by at least two of the following: palpation, resisted wrist, index and/or middle finger extension, or gripping. The exclusion criteria were any other neuromusculoskeletal injury, treatment for elbow pain within the previous three months, a history of surgery, fractures or dislocations of the elbow, systemic conditions, and the use of central nervous system depressive medications. In addition to the above exclusion criteria, the healthy controls had no previous history of LE. All participants were instructed to avoid strenuous gripping tasks and anti-inflammatory medications 48-hours prior to testing. Written informed consent was obtained from all participants, and all testing procedures were approved by the institutional ethics committee in accordance with the Declaration of Helskini.

Clinical assessment

Standard clinical outcomes of LE were collected one week prior to the gripping experiment, including duration of pain, pain intensity in the past 24 hours (Numeric pain rating scale, NPRS), pain-free grip strength (PFG, MIE digital grip dynamometer, UK)[14], and Patient Rated Tennis Elbow Evaluation (PRTEE)[15]. Sensory measures of pressure pain threshold (PPT)[14] and cold pain threshold (CPT)[3] were also recorded. Structural characteristics within the common extensor tendon were reported using a standard ultrasound assessment (Ultrasonix, Richmond, BC). All images were de-identified prior to the assessment of sagittal plane tendon thickness (distance between the periosteal border of capitulum to the outer surface of the tendon) and the level of intra-tendinous hypoechoic changes[16,17], using OsiriX Imaging Software (Pixmeo SARL, version 7, 2003-2016).

Instrumentation

A digital hand grip dynamometer was used for all experimental tasks (MIE Ltd, Leeds UK). Participants sat comfortably with the shoulder in a neutral position and the affected elbow (or matched limb for controls) positioned in 65° flexion. The forearm was rested in full pronation on a custom designed plastic frame (Figure 1). The distal forearm was fixed to the frame with a non-compliant strap and participants were instructed to grip without lifting the hand from the frame. A goniometer was used to ensure wrist position was constant throughout testing. Surface EMG was used to measure muscle activity of the wrist extensors (ECRB, ECU), wrist flexors (FCR, FCU), and finger extensor (EDC) and flexor (FDS). Following skin preparation, the respective muscles were identified using recommended surface EMG electrode placement procedures[18,19]. The bipolar Ag/AgCl electrodes (Kendall Arbo, 24 mm) were placed over each muscle with an inter-electrode distance of 20 mm. A ground electrode was placed over the acromion. During electrode placement the investigators carefully monitored EMG activity for each muscle. In particular, controlled movements of the wrist and fingers were performed, and EMG activity was monitored for each electrode to identify if crosstalk was present. Wrist and finger movements were performed both passively and under resistance, and if crosstalk was obvious the EMG electrodes were replaced in a more appropriate position. Dynamometer and EMG data were acquired simultaneously at 1000 Hz using 16-bit Power 1401 interface and custom Spike2 software (Cambridge Electronic Design). All surface EMG signals were amplified by 1000 and band-pass filtered using cut-off frequencies of 3 and 500 Hz.

Experimental procedures

Due to the pain-provoking nature of maximal gripping in LE, MVC was assessed one week prior to the main experiment. During this session, participants performed three maximal grips each of the following postures: a neutral wrist, 20° wrist flexion, and 20° wrist extension. Participants were given 2-minute rests between contractions. The order of wrist postures was randomized and the MVC was calculated as the peak force for each wrist posture. The severity of pain during MVC testing was measured using the NPRS.

The main experiment consisted of performing five trials of gripping at 15% and 30% MVC in each wrist posture. The order of testing was randomized. The 30% MVC condition was selected as the highest force because it is estimated to be the maximum pain-free force achievable by people with LE[20]. The target force was presented on a monitor 1 m in front of the participant (Figure 1). Participants were instructed to squeeze the dynamometer to reach the target line over a 2 s period and then sustain the target force for 6 s. All participants were allowed a single practice trial, and ratings of pain (NPRS) were assessed after each target grip force. Upon completion of the total of 30 grip trials, MVC was again obtained to determine if the testing procedures compromised the ability of the participant to perform the tasks.

Surface EMG pre-processing and analysis

All data analyses were performed using custom Matlab software (Mathworks Inc., R2013a). For each experimental trial, 4 s of steady state EMG data was extracted from the middle of the sustained contraction. The EMG signal was band pass filtered using a zero lag, 2nd order Butterworth filter with cut-off frequencies set to 10 and 400 Hz. EMG amplitude of each muscle was normalized to the peak amplitude of the same muscle's MVC for each wrist posture. Two variables were obtained from the processed EMG data: 1) the amplitude of normalized EMG of each muscle during the grip tasks, and 2) the relative contribution of each muscle to the overall grip task. The amplitude of individual muscle activation was computed as the root mean square of the EMG signal using 25 ms non-overlapping windows across the 4 s of data. The relative contribution of each muscle to the grip task was calculated as the proportion of each muscle activation to the summed activity of all six muscles involved in the experiment.

Statistical analysis

Demographic, clinical and tendon structure measures were compared between groups using one-way analysis of variance (ANOVA). Paired student t-tests were used to assess within-group differences in maximal grip strength and PFG. Prior to statistical analysis, each target grip trial data with an EMG RMS of 1.5 times the inter-quartile range of the group were considered as outliers and removed from subsequent analyses. Repeated-measure ANOVA with a between-subjects factor of group (LE, control) and a within-subject factor of wrist posture (extension, flexion, neutral) was employed to examine the amplitude of individual muscle activation and relative contribution of each muscle to the grip task. If a main effect of group was detected, pairwise comparisons were performed between groups for each muscle, separately for each wrist posture and target force.

Table 1. Demographic, clinical and tendon structural characteristics for the lateral epicondylalgia (LE) and healthy control (HC) group. Data are presented as the mean (SD) unless otherwise specified.

		LE group n=11		HC group n=11		P value (LE vs HC)	
Demographic characteristics	Age, years	42 (11)		42 (11)		0.97	
	Sex, N	8 Male / 3 Female		8 Male / 3 Female		1	
	Height, cm	174 (9)		177 (6)		0.46	
	Weight, kg	80 (16)		73 (13)		0.31	
	Hand dominance, N	10 Right / 1 Left		10 Right / 1 Left		1	
	Affected arm, N	9 dominant / 2 non-dominant		-		-	
		LE group		HC group		P value (LE vs HC)	
		Test limb	Non-test limb	Test limb	Non-test limb	Test limb	Non-test limb
Clinical characteristics	Duration of condition, months	8 (7)	-	-	-	-	-
	Worst pain past 24 hours, NPRS 0-10 scale	5 (2)	-	-	-	-	-
	Pain during MVC grip,						
	NPRS 0-10 scale	7 (2)	-	-	-	-	-
	PRTEE, 0-100 scale	28 (12)	-	-	-	-	-
	PFG, Newtons	126 (102)*	246 (99)	-	-	-	-
	PPT, kPa	231 (84)	352 (120)	373 (121)	382 (97)	0.005	0.53
	CPT, °C	11 (12)	14 (11)	6 (6)	8 (8)	0.15	0.98
Ultrasound features	CET echo-intensity, 0-3 scale	2.3 (0.9)	0.6 (0.8)	0.3 (0.5)	0.3 (0.5)	0.001	0.21
	CET thickness, mm	6.5 (1.1)	5.5 (0.6)	4.9 (0.7)	4.9 (0.7)	0.001	0.05
		Day-1	Week-1	Day-1	Week-1	Day-1	Week-1
MVC grip	Wrist neutral, Newtons	245 (89) ᵗᵗ	259 (62)	296 (108) ᵗᵗ	261 (98)	0.24	0.55
	20° Wrist flexion, Newtons	174 (81) ᵗ	168 (57)	230 (87) ᵗ	205 (91)	0.13	0.26
	20° Wrist extension, Newtons	274 (85) ᵗᵗ	274 (88)	286 (81) ᵗᵗ	303(117)	0.75	0.53

*NPRS - numerical pain rating scale; MVC - Maximal voluntary contraction; PRTEE - Patient Rated Tennis Elbow Evaluation; PFG - pain-free grip strength; PPT - pressure pain threshold; CPT - cold pain threshold; CET - common extensor tendon; CET echo-intensity scale: 0 = Absent, 1 = <30% of hypoechoic change, 2 = 30 to 50% of hypoechoic change, 3 = >50% of hypoechoic change in the tendon region; WN – Neutral wrist; WF – Wrist flexion; WE – Wrist extension; Day-1: Base line maximal grip strength; Week-1: Maximal grip strength measured on the day of experiment. *significant difference between test limb and non-test limb within the LE group (P<0.05); ᵗᵗsignificant difference in maximal grip strength in neutral wrist and wrist extension compared to wrist flexion posture (marked with "ᵗ") within the LE group and control group (P<0.05). Note: there were no between-group differences in maximal grip strength at any wrist posture.*

If a main effect of posture was identified, univariate ANOVA with Tukey's post-hoc multiple comparisons were used to determine which wrist posture affected the dependent measures. Linear regression analyses were used to identify the association of clinical and tendon structure measures with EMG variables. All statistics were performed using IBM® SPSS® Statistics (version 22) with alpha levels set at <0.05.

Results

Participant characteristics

Demographic, clinical and structural characteristics for LE and control participants are summarised in Table 1. PFG was significantly reduced compared to maximal grip force in the LE group (p=0.014), however there were no significant differences between groups in maximal grip force or EMG amplitude during maximal gripping for any wrist posture or testing session (Table 1, p>0.05). None of the LE participants reported pain during the 15% MVC target grip trials, however, 6/11 LE participants reported mild discomfort (NPRS ranged 0 to 2/10) over the elbow/forearm during the 30% MVC grip trials. Of the total number of the grip trials (660), 8% were identified as outliers and were removed before statistical analyses.

Analysis of the duration and rate of grip force development across all wrist postures revealed no between group differences for 15% MVC and 30% MVC gripping task ($p \geq 0.5$). The average duration of grip force development

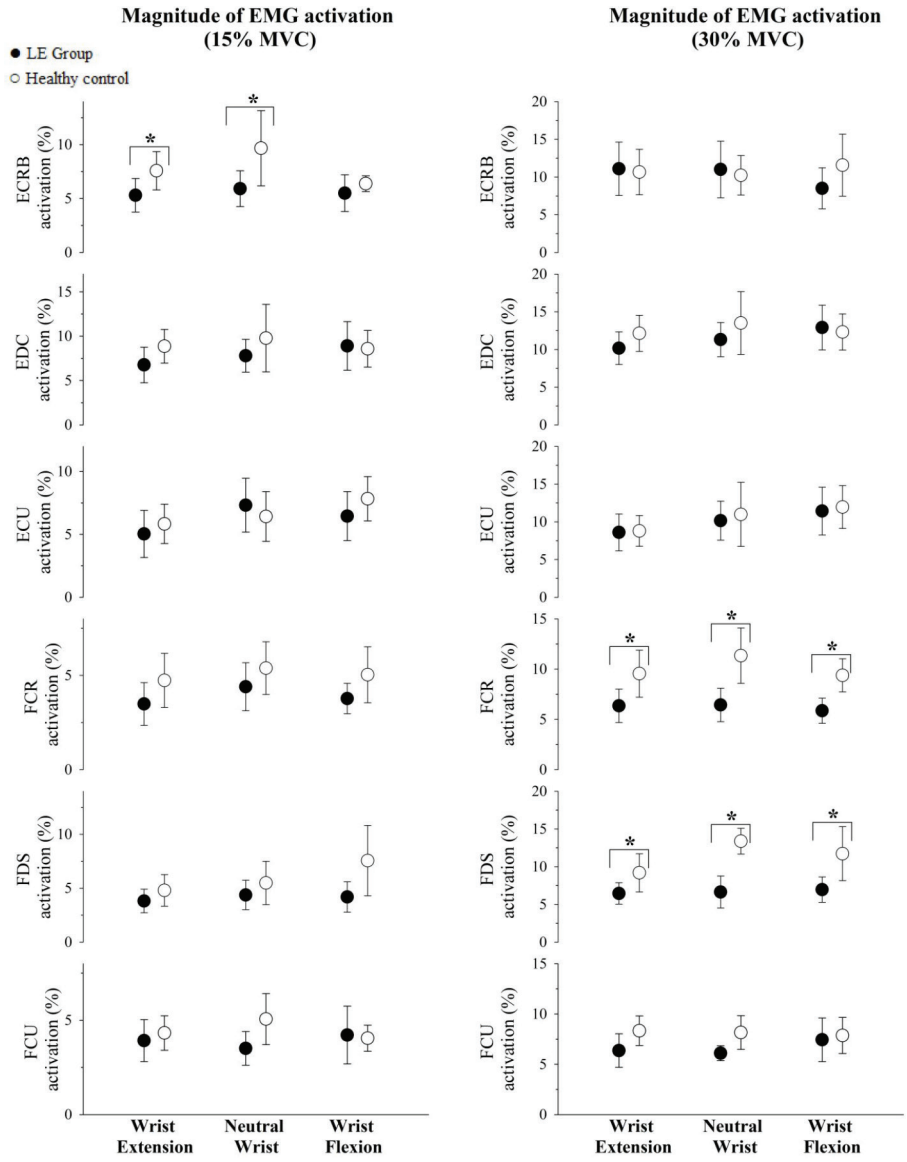

Figure 2. The magnitude of individual forearm muscle activity between LE and healthy control groups at 20° wrist extension, neutral, and 20° wrist flexion during 15% and 30% target grip force. Data points are mean and 95% confidence intervals. ECRB: extensor carpi radialis brevis, EDC: extensor digitorum communis, ECU: extensor carpi ulnaris, FCR: flexor carpi radialis, FDS: Flexor digitorum superficialis, FCU: Flexor carpi ulnaris. *Significant difference between groups (p<0.05)

measured during 15% and 30% MVC were 1.6±0.5 s and 1.8±0.9 s (for LE group), and 1.4±1.1 s and 1.7±1.2 s (for control group). Similarly, the mean rate of force development during 15% and 30% MVC were 23.4±10.2 $N.s^{-1}$ and 44.3±23.9 $N.s^{-1}$ (for LE group), and 31.4±14.5 $N.s^{-1}$ and 55.2±24.1 $N.s^{-1}$ (for control group).

Magnitude of individual muscle activation during gripping

Of the total number of the grip trials (660), 8% were identified as outliers and were removed before statistical analyses. The LE group had significantly lower ECRB activity during 15% MVC (p=0.021), with pairwise comparison showing decreased ECRB during wrist extension (p=0.044) and neutral wrist (p=0.043) postures compared to controls (Figure 2). The magnitude of ECRB activation was not different between groups at 30% MVC. However, the LE group had a significantly lower activation of FCR (p = 0.001) and FDS (p=0.001) compared to the controls at 30% MVC. Pairwise comparisons revealed that LE FCR and LE FDS activation were significantly decreased in wrist extension (FCR: p=0.022, FDS: p=0.048), wrist flexion (FCR: p=0.001, FDS: p=0.014) and in neutral wrist posture (FCR: p=0.003, FDS: p=0.001) compared to controls (Figure 2).

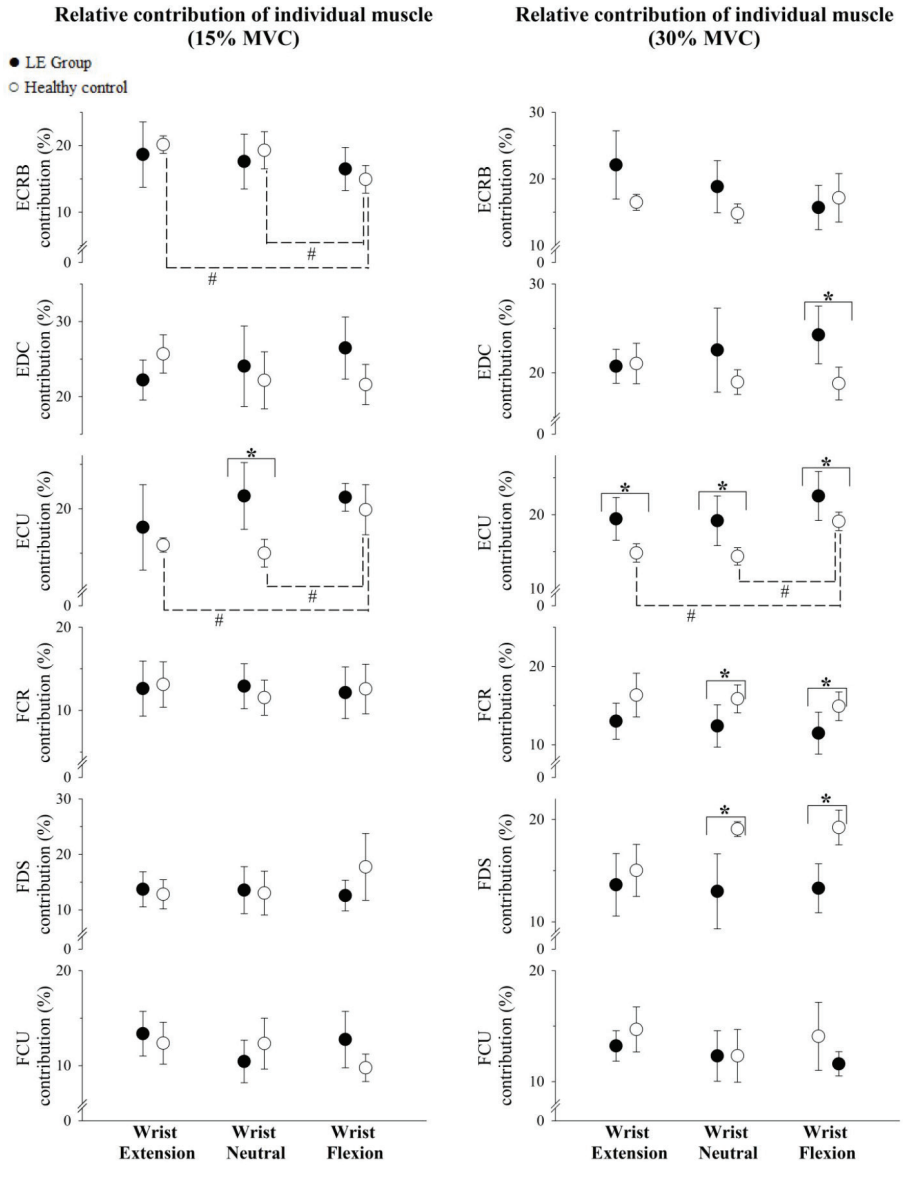

Figure 3. The relative contributions of individual muscle activity, as a percentage of total muscle activity during gripping for LE and the healthy control groups at 20° wrist extension, neutral, and 20° wrist flexion, during 15% and 30% target grip force. Data is mean and 95% confidence intervals. ECRB: extensor carpi radialis brevis, EDC: extensor digitorum communis, ECU: extensor carpi ulnaris, FCR: flexor carpi radialis, FDS: Flexor digitorum superficialis, FCU: Flexor carpi ulnaris. *Significant difference between groups (p<0.05). #Significant difference in the relative contributions of the target muscle between wrist postures (marked as dashed lines) in healthy controls (p<0.05).

Relative contribution of each muscle to the overall grip task

The relative contribution of ECU at 15% MVC grip was significantly increased in LE compared to controls (p=0.01), with pairwise comparisons showing significantly increased contribution of LE ECU to the gripping only at the neutral wrist posture (p=0.002, Figure 3). During the 30% MVC grip, the relative contribution of EDC (p = 0.017) and ECU (p=0.001) muscles to gripping were significantly increased in LE compared to controls. Pairwise comparisons revealed significantly increased relative contribution of LE EDC only in wrist flexion (p=0.004), whereas the relative contribution of LE ECU was significantly increased in the neutral (p=0.007), extension (p=0.004) and flexion (p=0.042) wrist postures compared to controls (Figure 3).

Differences were also identified for the wrist and finger flexors at 30% MVC, where the relative contribution of FCR (p=0.003) and FDS (p=0.001) were significantly decreased in LE compared to controls. Pairwise comparisons revealed

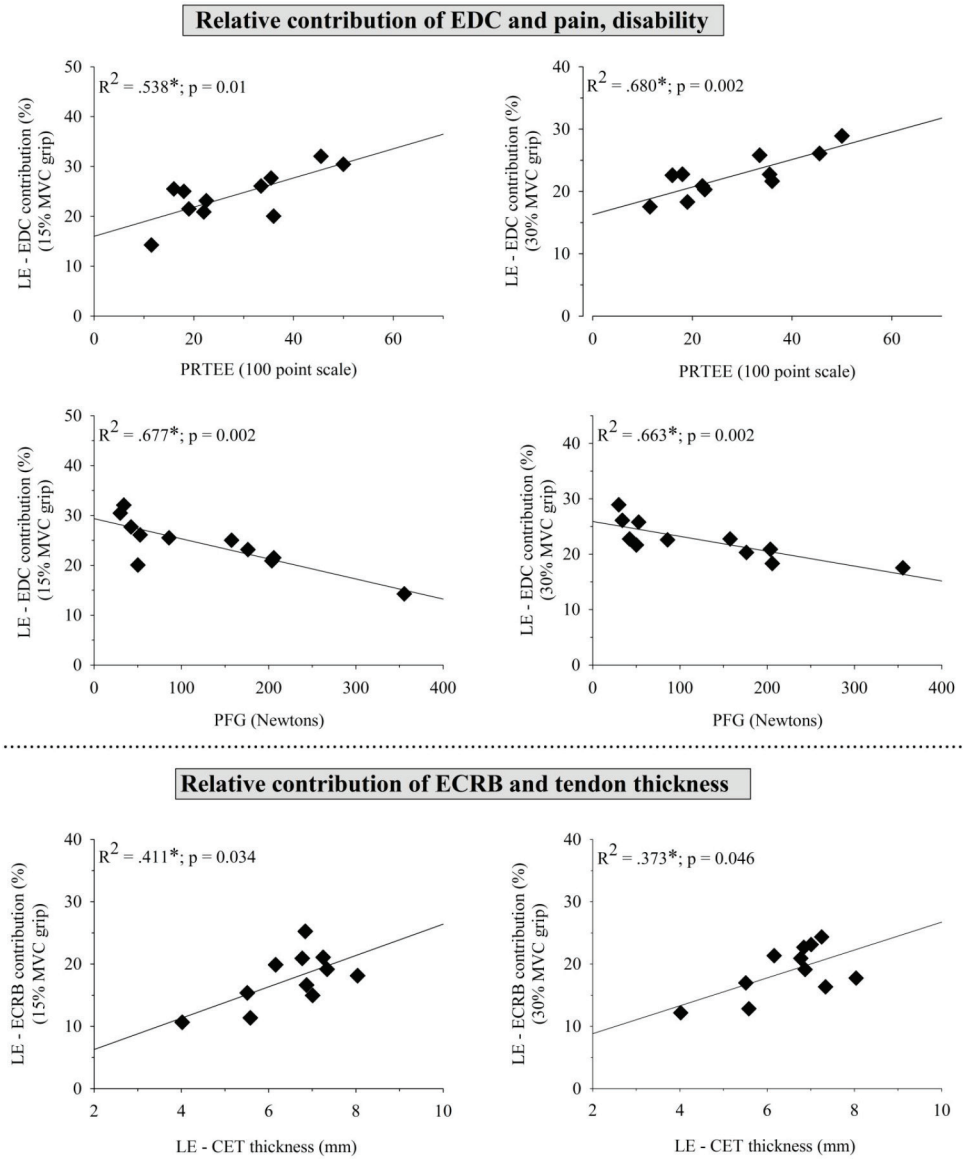

Figure 4. Association of clinical measures and diagnostic ultrasound measures with the relative contribution of the extensor digitorum communis (EDC) and extensor carpi radialis brevis (ECRB) muscle activation, averaged across the three wrist postures in the LE group. PRTEE: Patient-Rated Tennis Elbow Evaluation, PFG: Pain-free grip strength, CET: common extensor tendon, R^2= Coefficient of correlation. *Significant association between the clinical measures and muscle activation pattern in LE (p<0.05).

significantly decreased LE FCR and LE FDS contributions in wrist flexion (FCR: p=0.028; FDS: p=0.001) and neutral wrist postures (FCR, p=0.028; FDS, p=0.002) compared to controls (Figure 3).

Effect of wrist posture on the relative contributions of wrist extensors

A main effect of wrist posture was identified for the relative contribution of ECRB (p=0.016) and ECU (p=0.018) during 15% MVC grip. However, post hoc analyses showed that only the control group exhibited significant differences, with increased contribution of ECRB in wrist extension (p=0.002) and neutral wrist (p=0.009), compared to wrist flexion, and increased ECU in wrist flexion compared to wrist extension (p=0.003) and neutral wrist (p=0.001, Figure 3). This indicates that gripping in healthy individuals is characterised by the different muscle activity for different postures, whereas the LE group does not have the same strategy.

Similar to the 15% MVC, the main effect of wrist posture was found for the relative contribution of ECU during the 30% MVC (p=0.001). Once again, only controls had a different

activity for different postures, with significantly increased relative contribution of ECU in wrist flexion compared to wrist extension (p=0.001) and neutral wrist (p=0.001, Figure 3) identified on post hoc tests.

Associations of clinical and tendon thickness measures with EMG variables

As wrist posture had no effect on the EMG-derived variables for individuals with LE, the amplitude of each muscle and the relative contribution of each muscle during the grip tasks were averaged across the wrist postures for the LE group prior to further analyses. Increased relative contribution of EDC was significantly associated with increased pain and disability (PRTEE) and decreased PFG in LE (Figure 4). Among the three wrist and finger extensors that share the common extensor tendon, only the relative contribution of the ECRB showed a significant association with increased tendon thickness in LE (Figure 4).

Discussion

This is the first study to evaluate the effects of wrist posture on magnitude of muscle activity and relative contribution of individual forearm muscles during a gripping task in people with LE. At 15% MVC the amplitude of ECRB activity was significantly lower and there was greater relative contribution from ECU in LE compared to controls. At 30% MVC there was a reduction in flexor activity (FCR and FDS) with greater relative contribution from the extensors (EDC, ECU) in the LE group. In addition, posture significantly influenced extensor muscle activity in healthy controls at both 15% and 30% MVC, but not in people with LE.

LE participants in this study presented with significant tendon degeneration, mechanical hyperalgesia, and reduced PFG strength, however it is important to note that no participant reported pain during the 15% MVC grip. As such, it is likely that the observed change in ECRB during this task represent chronic pain-related adaptations in motor control rather than an acute response to nociceptive stimuli during testing. Although reduced ECRB activity may be mediated by local tendon or muscle pathology, adaptations in the CNS may also be observed via changes in muscle activity. For example, an LE case-control experiment has previously shown significant reductions in ECRB motor unit firing rate during isometric (5 to 10% MVC) wrist extension[21]. In addition to altered motor unit firing rate, chronic musculoskeletal pain can limit central drive to the muscle as part of an anti-nociceptive protective mechanism[22], which will be reflected by declines in EMG amplitude.

LE is a chronic condition implicated by dysfunction of the ECRB, so the reduction in FCR and FDS activation in the LE group during the 30% MVC grip task is an interesting finding. While the decreased magnitude of FDS activation may be a physiological consequence to decrease the demand on the painful synergistic wrist extensors[22], it may also be due to centrally driven motorneuron inhibition that occurs during grip tasks which are habitually painful in LE[22-24]. In fact, mild pain reported by some LE participants during the 30% MVC grip may have triggered inhibitory mechanisms of the muscles responsible for gripping (i.e. the flexors). It appears that regardless of wrist posture in people with LE, muscle activation adapts differently in ECRB, FCR and FDS based on the level of grip force exertion.

Estimating the relative contribution of each muscle's activation to the summed activation of all muscles provides an understanding of compensatory adaptation that occurs within the synergistic and antagonistic forearm muscles. During the 15% MVC grip, individuals with LE exhibited greater synergistic contribution from ECU across wrist postures compared to the controls, which is consistent with a previous finding of increased contribution of ECU during 20% MVC isometric gripping and wrist extension in LE compared to controls[6,8]. In addition, the contribution of flexor muscles to the overall gripping strategy differed between LE and controls during higher level grip force (30% MVC). While the EDC and ECU activation were higher relative to the summed muscle activity in LE, FCR and FDS were concomitantly *lower* in LE group compared to the controls. The relative contribution of flexor digitorum profundus (FDP) has previously been shown to increase at 20% MVC gripping in LE[6], however we did not measure FDP activity due to methodological concerns with surface EMG. It is possible that an increased contribution from flexor digitorum profundus may compensate for decreased contribution from FDS and FCR in people with LE, which may maintain the overall force being generated during gripping. Our finding of decreased flexors and concomitantly increased extensors contribution to the total summed activity when gripping at higher forces (30% MVC) suggest that the coordination between the synergistic forearm extensors and flexors in individuals with LE varies depending on grip force.

Recent work by Heales et al.[6,13] examined coordination of forearm muscles during submaximal gripping in different shoulder, elbow and forearm positions. Consistent with our findings, Heales et al.[6] reported the relative contribution of ECRB to total EMG was less in LE compared to controls. However, in contrast to our results, they found no significant difference in ECRB activity between arm positions. This lack of difference in ECRB EMG activity between shoulder and elbow positions is perhaps not unexpected, given that the primary role of ECRB is to stabilise the wrist by counteracting the wrist flexion moment created by contraction of the finger flexors during gripping[12]. In addition, change in sarcomere length within ECRB is most influenced by changes in wrist position rather than changes in elbow or forearm position[25]. As discussed earlier, we found a significant effect of wrist posture on ECRB and ECU activation in healthy controls, but not in LE, reflecting reduced variability in the activation of wrist extensor muscles in LE. Heales' group[6] did not find any such difference between LE and control groups based on changes in arm position. Nor did they find any difference in the number of muscle synergies during a gripping task, between LE and Controls[13]. In contrast, we have recently identified fewer number of forearm muscle synergies during

grip force development to 15% MVC in LE[26], reflecting that even with pain-free gripping, changes in motor control that reflects reduced variability is a feature of chronic LE. Perhaps the difference in findings between our work and that of Heales' group is the test conditions of upper limb position[6,13]. We deliberately chose to investigate the effect of wrist position (flexion, neutral, extension), whereas Heales' group[6,13] investigated the effects of shoulder (flexion, neutral), elbow (flexion, extension) and forearm (supination, neutral, pronation) positions. It appears that differences in motor control between LE and healthy controls are associated with changes in wrist position rather than changes in position of other upper limb joints.

We found that in individuals with LE, more severe pain and disability (i.e. higher PRTEE and lower PFG) were associated with a greater contribution from EDC during both the 15% and 30% MVC grip. This may be an adaptive strategy to reduce the load on the pathological portion of the common extensor tendon associated with the ECRB. Alternatively, it is possible that the increased EDC contribution in people with LE might be a pre-existing trigger of painful tendinopathy, as previous evidence has suggested[27]. Likewise, the common diagnostic ultrasound features of LE[16,17], such as increased tendon thickness and intra-tendinous hypoechoic features (representing disorganized tendon collagen) were observed in our LE cohort compared to controls. While our finding of increased tendon thickness in LE supports the results of previous work[17], it is in contrast to a previous study in which blinded assessment revealed no difference in tendon thickness between the affected side in LE and the matched side in healthy controls[28]. However, tendon thickness showed a positive linear association with ECRB contribution in LE, but not in healthy controls. While the overall ECRB contribution did not differ between the LE and control groups in this study, greater tendon thickness in LE may reflect a degree of matrix disorganisation as well as a proportion of intact, aligned fibrillar collagen[29]. The intact portion of the tendon is capable of transmitting force, thereby maintaining the relative contribution of the ECRB muscle to gripping.

The current study was a cross-sectional design that compared two different groups of subjects. A consequence of this design is that we cannot definitively express whether our results are a cause or effect of the chronic pain. Although EMG assessment using surface electrodes is a standardized, non-invasive method for measuring muscle activity, there is a risk of EMG crosstalk between the small superficial forearm muscles. Furthermore, measurements of deeper muscles such as FDP are limited using surface electrodes. In addition, we did not assess for the presence or absence of palmaris longus, which when present, lies superficial to FDS, and might influence the recorded activity of the flexor muscles. Notwithstanding these limitations, using a standardized EMG electrode placement method and use of an isometric grip task instead of a task that involves limb movement helps to minimise the negative effects of EMG crosstalk[30]. Finally, pain during strong contractions may compromise individuals with LE to maximally grip, which may be problematic when using MVC for normalization. However, there were no significant between- or within-group differences in maximal grip force or EMG amplitude during MVC for any wrist posture or testing session, which suggests that our use of MVC grip was justified.

Conclusion

Our hypothesis that ECRB absolute amplitude and relative contribution is decreased regardless of grip force and wrist posture, in only partially confirmed. This study showed that the forearm muscle activity pattern adapts differently in people with LE based on the level of grip exertion. People with LE adapt a simplified motor strategy for wrist extensors when gripping with different wrist postures, compared to controls. The preliminary evidence of association between the severity of pain and disability and altered forearm muscle activity indicates the need for muscle-specific motor control interventions in LE. Further investigations are required to determine whether changing altered motor control through intervention improves clinical outcomes in people with LE.

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