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Muscle Activation During Landing Before and After Fatigue in Individuals With or Without Chronic Ankle Instability

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Context: Ankle instability is a common condition in physically active individuals. It often occurs during a jump landing or lateral motion, particularly when participants are fatigued.

Objective: To compare muscle activation during a lateral hop prefatigue and postfatigue in individuals with or without chronic ankle instability (CAI).

Design: Cross-sectional study.

Setting: Sports medicine research laboratory.

Patients or Other Participants: A total of 32 physically active participants volunteered for the study. Sixteen participants with CAI (8 men, 8 women; age = 20.50 ± 2.00 years, height = 172.25 ± 10.87 cm, mass = 69.13 ± 13.31 kg) were matched with 16 control participants without CAI (8 men, 8 women; age = 22.00 ± 3.30 years, height = 170.50 ± 9.94 cm, mass = 69.63 ± 14.82 kg) by age, height, mass, sex, and affected side.

Intervention(s): Electromyography of the tibialis anterior, peroneus longus, gluteus medius, and gluteus maximus was measured before and after a functional fatigue protocol.

Main Outcome Measure(s): Activation of 4 lower extremity muscles was measured 200 milliseconds before and after landing from a lateral hop.

Results: We observed no interactions. The group main effects for the peroneus longus demonstrated higher muscle activation in the CAI group (52.89% ± 11.36%) than in the control group (41.12% ± 11.36%) just before landing the lateral hop ($F_{1,30} = 8.58$, $P = .01$), with a strong effect size (d = 1.01). The gluteus maximus also demonstrated higher muscle activation in the CAI group (45.55% ± 12.08%) than in the control group (36.81% ± 12.08%) just before landing the lateral hop ($F_{1,30} = 4.19$, $P = .049$), with a moderate effect size (d = 0.71). We observed a main effect for fatigue for the tibialis anterior, with postfatigue activation higher than prefatigue activation ($F_{1,30} = 7.45$, $P = .01$). No differences were present between groups for the gluteus medius.

Conclusions: Our results support the presence of a centralized feed-forward neuromuscular alteration in patients with CAI, not only in the ankle-joint muscles but also in the proximal hip muscles. These results may have implications for rehabilitation programs in these patients.

Key Words: ankle injuries, hip, proprioception, postural control, feed-forward mechanism

Key Points

- Just before landing a lateral hop, activation values in the peroneus longus and gluteus maximus muscles were higher in participants with chronic ankle instability than in control participants.
- After repeated ankle trauma, proximal and distal neuromuscular alterations may result from a centralized feed-forward mechanism to prepare the lower extremity for landing without injury.
- More research involving functional activity with perturbation is needed to help investigators address neuromuscular deficits in patients with chronic ankle instability.

Ankle sprains are one of the most common injuries in activity, and reinjury rates as high as 73% have been reported.[1] When a substantial lateral ankle sprain occurs, a common outcome is repeated giving way of the ankle during activities. Termed *chronic ankle instability* (CAI), it is characterized by residual lateral instability[2] categorized as (1) mechanical instability related to anatomical changes in tissues surrounding the ankle, (2) functional or perceived instability related to neuromuscular changes,[2] or (3) recurrent sprains in which a patient experiences repeated inversion injury with activity. Individuals with CAI may be subcategorized into 1 of these categories or a combination of the 3.[3–5]

Much still needs to be understood about why patients continue to experience residual effects after an ankle sprain. Numerous authors[6–10] have proposed altered peripheral neuromuscular control due to ankle injury. Specifically, impairments are related to proprioception, neuromuscular control, or strength, leading to deficits[2,3] not only at the ankle joint but of the entire lower kinetic chain.[11–13] Wyke[14] proposed that damaged mechanoreceptors in a joint capsule could disrupt the central nervous system's feedback control of joint positioning and movement. This

disruption could, in turn, create changes in the movement of the entire lower extremity.[15] Caulfield and Garrett[16] further substantiated these changes, reporting differences in the kinematics of both the ankle and the knee before jump landing. Researchers[13,17,18] have proposed that residual symptoms in individuals with CAI not only include changes at the ankle due to poor afferent feedback information but also a neuromuscular reorganization to a centralized feed-forward mechanism during dynamic tasks to avoid reinjury. Beckman and Buchanan[11] found changes in the firing of the hip muscles in individuals with CAI compared with healthy controls. Proximal neuromuscular control at the hip, specifically the gluteus medius (Gmed) and gluteus maximus (Gmax), contributes to the positioning of the lower extremity[19–21] and may be affected in individuals with CAI. Continuing to investigate the possibility of these changes in those with CAI is important to help us understand the best means of implementing interventions that may prevent subsequent injury.

Another factor possibly contributing to CAI is muscle fatigue, which has been demonstrated to alter motor control,[22] joint position sense,[23] and muscle response during activity.[24] These alterations due to fatigue can lead to improper positioning of the lower extremity and difficulty responding quickly to changes in movements, which can predispose patients to injuries.[22,25] In addition, moderate and major injuries more commonly occur at the end of athletic practices and games.[26] Physically active patients often perform many of the mechanisms of ankle sprain, namely changing direction and jump landing; therefore, fatigue may be an additional factor leading to CAI that has not been studied extensively in patients with the condition.

Researchers[24,27] investigating the effects of fatigue on the lower extremity have found decreased muscle activation and increased postural sway after fatiguing protocols. To our knowledge, no one has examined whether fatigue is associated with altered electromyography (EMG) activity in the hip and ankle muscles in this patient population during a functional task. Examining the prefatigue and postfatigue activation of the proximal muscles of the Gmed and Gmax and the distal muscles of the tibialis anterior (TA) and peroneus longus (PL) during a lateral hop may identify mechanisms that can lead to ankle sprain over time. Acquiring data from the hip and ankle muscles during this activity may contribute to a better understanding of the mechanism of ankle injury and aid in designing rehabilitation protocols to prevent recurrent sprains. Therefore, the purpose of our study was to examine the prefatigue and postfatigue EMG activity of the TA, PL, Gmed, and Gmax in individuals with or without CAI before and during the landing of a lateral hop.

METHODS

We implemented a cross-sectional, repeated-measures design to examine prefatigue and postfatigue EMG of the hip and ankle musculature in participants with or without CAI during a lateral hop.

Participants

A total of 32 individuals volunteered for the study. Sixteen participants with CAI (8 men, 8 women; age = 20.50 ± 2.00 years, height = 172.25 ± 10.87 cm, mass = 69.13 ± 13.31 kg) were matched with 16 volunteers without CAI serving as control participants (8 men, 8 women; age = 22.00 ± 3.30 years, height = 170.50 ± 9.94 cm, mass = 69.63 ± 14.82 kg) by age, height, mass, sex, and affected side. Participants reported to the sports medicine research laboratory for a single session.

The participants with CAI presented with a history of ankle injury resulting in an antalgic gait or pain for 24 hours or longer and 2 or more self-reported episodes of the ankle giving way in the 6 months before the study. These participants needed to score 90% or less on the Foot and Ankle Disability Index (FADI; mean = 82.0% ± 16.19%) or 80% or less on the FADI-Sport[28] (mean = 64.19% ± 20.36%) for inclusion in the study. Control-group participants did not have previous ankle sprains and had completed the FADI (mean = 100% ± 0%) and FADI-Sport (mean = 100% ± 0%). These tools helped to establish that the participants had similar levels of instability during activities of daily living (FADI) and sport activity (FADI-Sport). Control participants were assigned an "injured" limb, which was compared with the injured side of their matched participants in the CAI group.

Neither group had a previous lower extremity fracture or surgery or any vestibular changes, including concussions, within the 6 months before the study, and they reported being *physically active*, which was defined as participating in 20 to 30 minutes of cardiovascular activity at least 3 times each week. To verify the level of physical activity, all participants completed the Participation Activity Readiness Questionnaire and answered *no* to all questions. All patients completed the Tegner Activity Scale to document weekly physical activity; all answered *level 5* or above, which indicated they were minimally involved in recreational activity on uneven ground at least twice weekly. On the basis of a previous study,[29] a power-analysis calculation[30] suggested that 15 participants in each group would be sufficient (power = 0.80, $P < .05$). All participants provided written informed consent, and the study was approved by the University of Toledo Institutional Review Board (No. 106670).

Equipment

A force platform (model NC-4060; Bertec Corporation, Columbus, OH) was integrated with MotionMonitor software (Innovative Sports Training, Inc, Chicago, IL) to record initial contact (IC) during the hopping task. We collected EMG data using the Telemyo 2000 System (Noraxon USA, Inc, Scottsdale, AZ) with an input impedance of greater than 100 mΩ and a common-mode rejection ratio of greater than 100 dB. Dual circular silver/silver chloride disposable electrodes with adhesive areas of 4 × 2.2 cm, conductive area of 1 cm, and interelectrode distance of 2 cm (Noraxon USA, Inc) were used. The EMG data were sampled at 1000 Hz and stored on a personal computer for offline analysis. A floor-mat–activated timing device (Lafayette Instrument Co, Lafayette, IN) was used to time the fatiguing protocol. Files were exported to Excel (version 2010; Microsoft Corporation, Redmond, WA) for data reduction and analysis.

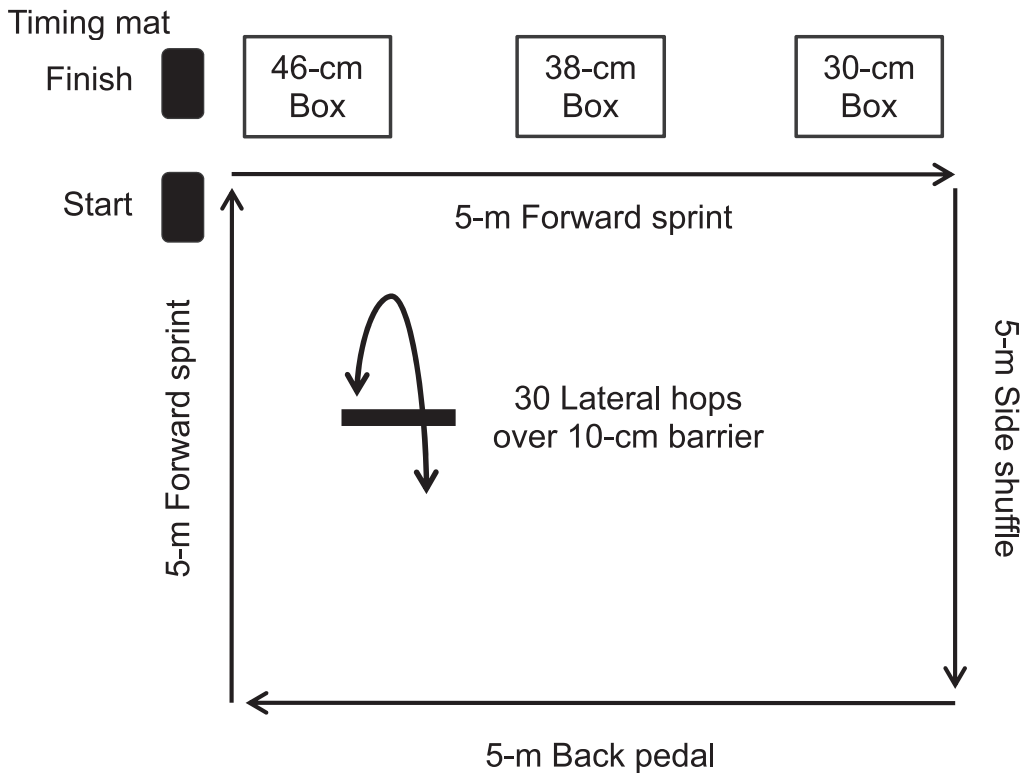

Figure. Functional fatigue protocol.

Procedures

We tested the injured limb of the CAI group and the matched limb of the control group. We shaved the skin superficial to the 4 muscles when necessary and lightly abraded and cleaned it with alcohol. The electrodes were positioned in the direction of the muscle fibers that were being measured, which was consistent with established protocols.[31] For the hopping portion, an investigator (K.A.W.) wrapped the TA and PL electrodes and leads with nonadhesive tape (Powerflex; Andover Healthcare, Inc, Salisbury, MA) to avoid movement both prefatigue and postfatigue.

At prefatigue, we measured the length of participants' fibulas and marked this distance with 1 piece of tape on the floor and 1 piece on the force platform. We then instructed individuals to use the test limb to perform 5 lateral hops onto and off of the force platform a distance equal to the length of their fibula and over a barrier that was 5 cm high.

The subsequent fatigue protocol[32] consisted of 5 × 5-m cone drills involving combinations of forward sprints, lateral shuffles, pivoting, and backward running. Next, participants completed 30 2-footed lateral hops over a 10-cm barrier, followed by 3 successive step-ups onto and 2-footed hop-downs from boxes measuring 30, 38, and 46 cm high (Figure). Participants used the floor-mat–activated timing device to time the fatigue trials. They had 20 seconds between fatigue bouts to return to the starting position.

We instructed the participants to repeat the protocol until *fatigue*, which was defined as (1) 50% increase in their fastest time to complete the course, (2) inability to repeatedly clear the 10-cm barrier on the lateral jumps, (3) inability to step onto the plyometric box, or (4) unwillingness to continue. The average number of completed fatigue protocols was 8.4. Two participants ended the protocol due to reason 1; 1, due to reason 2; 1, due to reason 3; and all other participants, due to reason 4. Immediately postfatigue, participants completed the same 5 1-footed lateral hops as during the pretest while EMG data were collected.

Data Processing

The EMG signals from the TA, PL, Gmed, and Gmax were collected at 1000 Hz for the periods of prelanding (200 milliseconds pre-IC to IC) and postlanding (IC until 200 milliseconds post-IC), filtered using a fourth-order band-pass filter with cutoffs at frequencies of 10 Hz and 500 Hz, smoothed using a 50-millisecond root mean square algorithm, and full-wave rectified. The prefatigue and postfatigue data were normalized to the mean peak EMG amplitude of the corresponding prefatigue and postfatigue 5 hopping trials for each participant.[29]

Statistical Analysis

Means and standard deviations of the normalized percentage of average peak muscle activation were used for analysis. Dependent variables were amplitude of the TA, PL, Gmed, and Gmax prelanding and postlanding. For each dependent variable, we performed a separate group-by-fatigue repeated-measures analysis of variance. Effect sizes were calculated using the Cohen d with 95% confidence intervals (CIs) and interpreted as *small* (0.2), *medium* (0.5), or *large* (0.8).[33] Data were analyzed using SPSS (version 15.0; SPSS Inc, Chicago, IL). We set the α level a priori at .05.

Table 1. Muscle-Activation Prelanding Interaction Results

Muscle	Group	Muscle Activation, %, Mean ± SD		$F_{1,30}$ Value	P Value	Postfatigue Between-Groups Comparison	
		Prefatigue	Postfatigue			Effect Size	95% Confidence Interval
Tibialis anterior	Chronic ankle instability	40.22 ± 3.90	44.15 ± 6.53	0.01	.94	−0.27	−0.95, 0.44
	Control	42.50 ± 6.86	46.22 ± 8.35				
Peroneus longus	Chronic ankle instability	48.07 ± 7.70	57.71 ± 25.43	1.35	.25	0.81[a]	0.09, 1.53
	Control	40.96 ± 13.72	41.29 ± 11.63				
Gluteus medius	Chronic ankle instability	42.17 ± 6.53	42.02 ± 7.38	0.34	.56	−0.27	−0.97, 0.42
	Control	46.53 ± 13.81	44.70 ± 11.41				
Gluteus maximus	Chronic ankle instability	40.55 ± 4.83	50.55 ± 23.98	2.84	.10	0.72[b]	0.00, 1.43
	Control	36.84 ± 11.93	36.77 ± 11.36				

[a] Indicates strong effect size.
[b] Indicates moderate effect size.

RESULTS

Our results for the prelanding and postlanding comparisons are presented in Tables 1 through 4. We noted no interactions. However, during prelanding postfatigue, the PL had notably higher muscle-activation levels in the CAI group (57.71% ± 25.43%) than in the control group (41.29% ± 11.63%). Activation levels for the Gmax were also higher in the CAI group (50.55% ± 23.98%) than in the control group (36.77% ± 11.36%). Although not different, the effect sizes for the PL ($P = .25$, $d = 0.81$) and Gmax ($P = .10$, $d = 0.72$) were moderate to strong (Table 1).

The group main effects for the PL demonstrated higher muscle activation in the CAI group (52.89% ± 11.36%) than in the control group (41.12% ± 11.36%) just before landing the lateral hop, with a strong effect size ($P = .01$, $d = 1.01$; Table 2). The Gmax also demonstrated higher muscle activation in the CAI group (45.55% ± 12.08%) than in the control group (36.81% ± 12.08%) just before landing the lateral hop, with a moderate effect size ($P = .049$, $d = 0.71$; Table 2). We observed a main effect for fatigue of the TA, which demonstrated higher activation postfatigue than prefatigue (Table 3). No differences were demonstrated in group or fatigue for the Gmed (Table 3). The only difference for the postlanding phase was in the TA, which demonstrated higher activation postfatigue than prefatigue across groups (Table 4).

DISCUSSION

Activation of the PL and Gmax was higher in the CAI than in the control group during the prelanding phase of a lateral hop. When we introduced functional fatigue, we also observed differences with moderate to strong effect sizes in the postfatigue results for the PL and Gmax, demonstrating clinical importance.

The higher muscle-activation values in the CAI group just before landing the lateral hop may help to support the theory suggested by Delahunt et al,[13] who proposed a centralized feed-forward mechanism to explain changes seen in patients with CAI. Authors of several subsequent studies[2,9,13,16,18,29,34] have attributed findings to this theory of the feed-forward mechanism. The theory is based on the premise that, due to injury, the normal reaction pattern of the muscles that protect the ankle in healthy patients is too slow to prevent injury positions in patients with CAI; therefore, a centralized feed-forward neural adaptation is implemented to protect the ankle from injury both proximally and distally. We instructed participants to hop laterally and return to a specific location. In concept, this activity would allow them to attempt to implement any protective neuromuscular control necessary to stabilize during the task, which is often associated with an ankle-sprain mechanism. These muscles may have been activated to a greater extent in the CAI group to help place the lower limb in a more protected position prelanding.[35]

Distal Alterations

When considering how these changes may appear in the distal segment of the lower kinetic chain, researchers have found that patients with CAI demonstrate changes at the ankle compared with healthy participants when preparing for the foot to contact the ground.[9,16,17,36] Similar to our findings for jump-landing preparation, Gutierrez et al[9] reported that patients with CAI demonstrated increased PL activity when preparing to land from a drop jump on a supinating surface. Whereas we used a lateral-hop landing,

Table 2. Muscle-Activation Prelanding Results by Group

Muscle	Muscle Activation, %, Mean ± SD		$F_{1,30}$ Value	P Value	Effect Size	95% Confidence Interval
	Chronic Ankle Instability	Control				
Tibialis anterior	42.19 ± 5.48	44.36 ± 5.48	1.25	.27	−0.39	−1.09, 0.31
Peroneus longus	52.89 ± 11.36	41.12 ± 11.36	8.58	.01[a]	1.01[b]	0.27, 1.75
Gluteus medius	42.10 ± 9.37	45.62 ± 9.37	1.13	.30	−0.37	−1.06, 0.33
Gluteus maximus	45.55 ± 12.08	36.81 ± 12.08	4.19	.049[a]	0.71[c]	−0.01, 1.42

[a] Indicates difference.
[b] Indicates strong effect size.
[c] Indicates moderate effect size.

Table 3. Muscle-Activation Prelanding Results by Time

Muscle	Muscle Activation, %, Mean ± SD		$F_{1,30}$ Value	*P* Value	Effect Size	95% Confidence Interval
	Prefatigue	Postfatigue				
Tibialis anterior	41.36 ± 3.95	45.19 ± 5.30	8.61	.01[a]	−0.80[b]	−1.52, −0.80
Peroneus longus	44.52 ± 7.87	49.50 ± 13.98	1.55	.22	−0.43	−1.13, 0.27
Gluteus medius	44.35 ± 7.64	43.36 ± 6.79	0.48	.50	0.13	−0.56, 0.83
Gluteus maximus	38.70 ± 6.44	43.66 ± 13.27	2.76	.11	−0.45	−1.17, 0.24

[a] Indicates difference.
[b] Indicates strong effect size.

they[9] instructed patients to land on a surface that allowed the ankle to supinate. Both circumstances are common mechanisms for ankle injury, so it is not surprising that similar findings would be demonstrated when patients anticipated a challenge at landing. The theory of a centralized feed-forward mechanism could explain a strategy to protect the joint when landing by attempting to place the ankle in a more stable position, as evidenced by higher activation of the PL in this case. Levin et al[36] also recently found changes in EMG firing of the PL, but the changes were in the contralateral limb of patients with CAI. Their results showed that patients with CAI had higher levels of contralateral PL activity just before jump landing than did healthy control participants. The authors also proposed that centralized feed-forward mechanisms were responsible for these changes. In another study, Caulfield and Garrett[16] instructed participants to perform a drop jump and noted earlier ground reaction force peaks in patients with CAI than in control participants. These authors also attributed this alteration to the feed-forward response mechanism of the neuromuscular system compensating for previous ankle injury by modifying ankle-joint landing. These data contribute to the premise that patients with CAI demonstrate different motor-behavior patterns than control participants when preparing their ankles for landing. Further prospective studies are necessary to determine whether this is an adapted response due to injury or whether patients had these differences before injury, which would suggest a predisposition to CAI. Caulfield and Garrett[16] did not collect kinematic data to determine whether the foot actually was positioned differently in patients with CAI than in healthy controls.

Delahunt et al[17] obtained kinematic data for a lateral hop of 30 cm and reported that participants with CAI displayed a less everted position of the ankle than did control participants. Caulfield and Garrett[37] noted the same positioning just before foot contact during gait. This observation seems counterintuitive given the findings of the studies discussed earlier with higher PL activity prelanding, theoretically placing the foot in a more everted position. The increased PL activation in our CAI group may suggest the neuromuscular system is attempting to evert the ankle to avoid an inversion motion during the lateral hop landing but is unable to move the foot into a more everted position.

Another protective tactic is a more dorsiflexed position, which allows for a more stable, close-packed orientation of the ankle joint. Researchers[37] have found that patients with CAI demonstrate increased dorsiflexion before landing from a jump compared with healthy controls. The higher postfatigue activation of the TA just before landing was observed across groups and may indicate that participants with or without CAI both attempt to increase ankle stability to handle landing. Whereas the TA is the muscle most responsible for ankle dorsiflexion, it also has a medially located insertion, allowing it to contribute to midfoot inversion. Activation of the PL possibly prevents the TA from placing the foot in a more inverted position but still allows for more dorsiflexion.

Proximal Alterations

Participants with CAI demonstrated differences not only in the lower leg and ankle but also in the proximal musculature compared with healthy controls. One of the aims of our study was to investigate the role of the proximal musculature in a landing task for participants with or without CAI. The Gmed and Gmax are involved in positioning the femur, which subsequently affects positioning of the ankle through the kinetic chain. Landing from a lateral hop requires control of hip flexion and rotation, in part by the Gmed and Gmax.[20] The increased Gmax activity of the CAI group observed prelanding may suggest an effort to position the lower extremity more under the center of mass, thereby creating slight hip extension. The Gmax also may be more highly activated as it prepares to limit internal femoral rotation at landing, which would put greater stress on the ankle to stabilize than if the femur was positioned properly under the body. These explanations are speculative, given that we did not quantify kinematic patterns in this study.

Table 4. Muscle-Activation Postlanding Results for Fatigue

Muscle	Muscle Activation, %, Mean ± SD		$F_{1,30}$ Value	*P* Value	Effect Size	95% Confidence Interval
	Prefatigue	Postfatigue				
Tibialis anterior	42.36 ± 5.37	45.73 ± 5.51	7.45	.01[a]	−0.60[b]	−1.31, 0.10
Peroneus longus	46.70 ± 5.55	45.71 ± 6.49	0.64	.43	0.16	−0.53, 0.85
Gluteus medius	43.44 ± 7.56	42.70 ± 6.04	0.23	.64	0.20	−0.50, 0.89
Gluteus maximus	41.46 ± 7.25	40.24 ± 6.70	0.44	.51	0.17	−0.52, 0.86

[a] Indicates difference.
[b] Indicates moderate effect size.

Whereas we are not aware of other researchers who reported Gmax activation during a lateral hop in patients with CAI compared with healthy controls, investigators have observed changes in proximal activity in this patient population. Rios et al[38] addressed the activation of the proximal muscles during another functional task, kicking a soccer ball, in individuals with or without CAI. Increased activation in the proximal muscles for the CAI group was present compared with the control group. They attributed these findings to an attempt by the proximal portion of the leg to maintain stability, which may protect the previously injured ankle.[38] Our results confirmed these findings of increased Gmax activity in patients with CAI prelanding, which may suggest a feed-forward mechanism to help maintain stability during the lateral jump landing. Van Deun et al[39] found that patients with CAI demonstrated later onset times for the hip than did control participants when transitioning from a double- to a single-legged stance. Whereas Bullock-Saxton et al[12] observed participants in an open chain position, they also noted changes in Gmax activation in patients with CAI compared with control participants, as evidenced by delayed activation during prone hip extension. Continued research is needed to determine whether these muscle-activation patterns are contributing to the movement patterns at the ankle in patients with CAI in conjunction with kinematic variables. Although it would seem that more muscle changes might be demonstrated in the Gmed due to the frontal-plane movement of the lateral hop, we did not observe differences in this muscle. The altered neuromuscular system of participants with CAI may defer to the larger, more powerful Gmax muscle. Overall, researchers have demonstrated neuromuscular changes in the activation timing of the Gmed and Gmax in participants with CAI compared with healthy participants,[11,12,38,39] but more research is necessary to investigate patterns of feed-forward neuromuscular control demonstrated in the proximal joints during dynamic movements.

Fatigue

We selected our fatigue protocol to simulate the demands of physical activity—including elements of sprinting, cutting, and lateral shuffles and an extended period of lateral hopping—with the intention of targeting and fatiguing the lower extremity. Evidence[22,23,26] has pointed toward greater injury risk and decreased neuromuscular control in fatigued participants, which may result in greater changes in muscle activation when comparing CAI and control participants.

Whereas we observed no interactions for fatigue (Table 1), the effect sizes for both the PL and Gmax were strong and moderate, respectively, with the CAI group demonstrating higher activation than the control group for both muscles in the postfatigue state. The effect sizes indicated some clinical importance of these comparisons. As muscles fatigue, contractile capability decreases, resulting in additional motor recruitment and increased frequency of firing.[40] The increase in EMG amplitude during fatigue may represent the recruitment of more motor units as the force-producing capabilities of type II fibers are diminished.[41] Increased amplitude in the EMG signal of a fatigued participant with CAI can also be explained by the reduced conduction velocity of the muscle action potential seen with fatigue, which widens the pulse and increases the area under the curve, resulting in a larger mean amplitude of the rectified EMG signal.[42] The results are varied as to whether fatigue is controlled centrally or peripherally or by a combination of the two.[43] Researchers[43] have determined that feeling fatigued, as in this study, is a complex process in which the body integrates sensory information to determine its ability to maintain homeostasis. Regardless of the cause, the clinically important changes in muscle activation in the CAI group may imply that rehabilitation programs should include functional exercise during a fatigued state to help overcome this deficit. More specifically, they could also demonstrate that the PL and Gmax should be targeted with more repetitions during therapeutic exercise in patients recovering from ankle instability to help avoid early fatigue when returning to activity.

Postlanding

After ground contact, greater TA activation was observed postfatigue than prefatigue across groups (Table 4). The TA may fatigue fastest and be most involved in landing, leading to higher activation postlanding for all participants, so clinicians do not need to focus on postlanding techniques related to the TA in patients with CAI. The lack of differences for all other muscles during the postlanding phase suggested that the CAI and control groups managed the jump landing similarly over ground contact and weight acceptance despite the altered neuromuscular control systems of participants with CAI, perhaps due to the planned nature of the task.

Limitations

We attempted to maintain an objective fatigue protocol by using participants' times as an indicator of fatigue, yet those who did not give full effort may still have experienced various levels of fatigue. Despite including only physically active volunteers, participants may still have had various levels of fitness or may not have given their full effort. Another limitation was the lack of kinematic data, which may have helped to explain positioning of the participants at landing, adding depth to the conclusions; however, given the demands of the fatigue protocol performed between the prehopping and posthopping measurements, maintaining the proper marker positions would have been challenging. An additional limitation may have been the controlled task during which participants could concentrate solely on hopping and landing; because they are not generally focusing on a landing task in real-life activity, this may not reflect how the maneuver would occur then. To better reflect actual participation, researchers should include an unexpected perturbation during activity, which may help to override some of the preplanned feed-forward mechanisms and demonstrate more alterations in neuromuscular control from a reactive mechanism. Finally, the smaller sample size may have resulted in a type II error, as evidenced by some of the findings that were not different but had associated moderate to strong effect sizes with confidence intervals that did not cross zero. These relationships suggest the need for continued work in this area with larger sample sizes.

Clinical Implications

Our findings have several clinical implications. The neuromuscular changes that participants with CAI demonstrated while performing a common functional athletic task indicate that alterations after ankle sprain occurred not only distally at the ankle but also proximally at the hip. Clinicians treating patients with CAI may not only need to focus on exercises at the ankle but also may need to consider the entire lower extremity chain and specifically gluteal muscle firing. Finally, functional fatigue may need to be incorporated into rehabilitation protocols because neuromuscular alterations were demonstrated in effect sizes in participants with CAI postfatigue.

CONCLUSIONS

During the prelanding phase of a lateral hop, higher activation values were observed in both the PL and Gmax muscles of participants with CAI than in control participants. These proximal and distal neuromuscular alterations may result from a centralized feed-forward mechanism developed after repeated ankle injuries that attempts to prepare the lower extremity for an injury-free landing. Moderate to strong effect sizes suggested that these values increased in the CAI group after completing a functional fatigue protocol.

Whereas researchers continue to show alterations in the proximal and distal musculature during dynamic tasks in individuals with CAI, more research using functional activity with perturbation is necessary to find more consistent results in these participants, so that more concrete conclusions can be drawn to address the neuromuscular deficits in this pathologic condition.

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