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Chronic Ankle Instability and Neural Excitability of the Lower Extremity

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Context: Neuromuscular dysfunction of the leg and thigh musculature, including decreased strength and postural control, is common in patients with chronic ankle instability (CAI). Understanding how CAI affects specific neural pathways may provide valuable information for targeted therapies.

Objective: To investigate differences in spinal reflexive and corticospinal excitability of the fibularis longus and vastus medialis between limbs in patients with unilateral CAI and between CAI patients and participants serving as healthy controls.

Design: Case-control study.

Setting: Research laboratory.

Patients or Other Participants: A total of 56 participants volunteered, and complete data for 21 CAI patients (9 men, 12 women; age = 20.81 ± 1.63 years, height = 171.57 ± 11.44 cm, mass = 68.84 ± 11.93 kg) and 24 healthy participants serving as controls (7 men, 17 women; age = 22.54 ± 2.92 years, height = 172.35 ± 10.85 cm, mass = 69.15 ± 12.30 kg) were included in the final analyses. Control participants were matched to CAI patients on sex, age, and limb dominance. We assigned "involved" limbs, which corresponded with the involved limbs of the CAI patients, to control participants.

Main Outcome Measure(s): Spinal reflexive excitability was assessed via the Hoffmann reflex and normalized to a maximal muscle response. Corticospinal excitability was assessed using transcranial magnetic stimulation. *Active motor threshold* (AMT) was defined as the lowest transcranial magnetic stimulation intensity required to elicit motor-evoked potentials equal to or greater than 100 μV in 5 of 10 consecutive stimuli. We obtained motor-evoked potentials (MEPs) at percentages ranging from 100% to 140% of AMT.

Results: Fibularis longus MEP amplitudes were greater in control participants than in CAI patients bilaterally at 100% AMT (control involved limb: 0.023 ± 0.031; CAI involved limb: 0.014 ± 0.008; control uninvolved limb: 0.021 ± 0.022; CAI uninvolved limb: 0.015 ± 0.007; $F_{1,41} = 4.551$, $P = .04$) and 105% AMT (control involved limb: 0.029 ± 0.026; CAI involved limb: 0.021 ± 0.009; control uninvolved limb: 0.034 ± 0.037; CAI uninvolved limb: 0.023 ± 0.013; $F_{1,35} = 4.782$, $P = .04$). We observed no differences in fibularis longus MEP amplitudes greater than 110% AMT and no differences in vastus medialis corticospinal excitability ($P > .05$). We noted no differences in the Hoffmann reflex between groups for the vastus medialis ($F_{1,37} = 0.103$, $P = .75$) or the fibularis longus ($F_{1,41} = 1.139$, $P = .29$).

Conclusions: Fibularis longus corticospinal excitability was greater in control participants than in CAI patients.

Key Words: transcranial magnetic stimulation, Hoffmann reflex, lateral ankle sprain

Key Points

- Corticospinal excitability in the fibularis longus at transcranial magnetic stimulation intensities of 100% and 105% of active motor threshold was higher in the healthy control group bilaterally than in the chronic ankle instability group.
- Transcranial magnetic stimulation intensities at 110% or more of the active motor threshold did not result in differences between groups.
- Corticospinal excitability of the quadriceps did not differ between groups.
- Spinal reflexive excitability of the fibularis longus and quadriceps did not differ between groups.

Ankle sprains are common musculoskeletal injuries, with an estimated 23 000 injuries per day in the United States.[1] Recurrent ankle sprains have been reported to occur in as many as 80% of patients with ankle injuries.[2] Multiple recurrent ankle sprains are thought to be a complication of chronic ankle instability (CAI),[3] which is a multifactorial pathologic condition hypothesized to originate from both mechanical insufficiencies and functional deficits.[3,4] Mechanical insufficiencies include pathologic joint laxity and altered arthrokinematics; functional deficits may include impaired postural control and decreased strength and neuromuscular control.[3,4] Chronic ankle instability results in self-reported disability, and the cumulative effect of multiple ankle sprains may hasten the progression of joint degeneration and osteoarthritis. Therefore, advancing rehabilitative approaches is critical to improve disability and decrease the risk of multiple ankle sprains in individuals with CAI.

Current nonoperative approaches to improve functional deficits in CAI patients have targeted clinical impairments associated with altered movement strategies that may increase the risk of ankle sprain.[5,6] Patients with CAI have

Table 1. Participant Demographics

Characteristic	Group	
	Chronic Ankle Instability	Healthy Control
Sex, No.		
Male	9	7
Female	12	17
	Mean ± SD	
Age, y	20.81 ± 1.63[a]	22.54 ± 2.92
Height, cm	171.57 ± 11.44	172.35 ± 10.85
Mass, kg	68.84 ± 11.93	69.15 ± 12.30
Foot and Ankle Ability Measure Sport[b]	61.75 ± 14.92	100.00 ± 0.00

[a] Indicates difference between groups ($P < .05$).
[b] Range, 0%–100%.

been observed to exhibit impaired postural control,[7] decreased muscle strength,[8] and altered ankle range of motion during jogging[9] and landing tasks.[10–12] They also exhibit less control of their center of pressure relative to the boundaries of their feet during single-limb stance[13] and take longer to stabilize after landing from a jump[14–16] than healthy control participants. Patients with CAI have exhibited decreased plantar-flexor[17] and ankle-evertor muscle strength[8] and delayed muscle-firing patterns in the fibularis musculature when perturbed while walking.[18] Ankle-dorsiflexion deficits[9] and increased subtalar-inversion and shank external-rotation ranges of motion have been demonstrated during both walking and jogging in CAI patients compared with healthy control participants.[19]

Altered muscle function after joint injury has been hypothesized to have neural origins rooted partially in a clinical impairment known as the *arthrogenic muscle response*.[20] This impairment is characterized by an abnormal facilitation or inhibition of neural drive to the undamaged musculature surrounding an injured joint. The central nervous system controls muscle contraction and modulates movements via spinal reflexive and corticospinal pathways.[21] Patients with ankle instability have decreased spinal reflexive excitability of the fibularis longus and soleus muscles, measured via the Hoffmann reflex (H-reflex), compared with healthy counterparts.[22] Similarly, corticospinal excitability of the fibularis longus in CAI patients has been shown to be diminished when compared with healthy participants assessed using transcranial magnetic stimulation (TMS).[23] Neuromuscular control adaptations in joints proximal to the ankle also have been demonstrated in patients with CAI, manifesting as deficits in force production,[17,24] changes in kinematic patterns,[10,11,14,25–27] and deficits in muscle-activation patterns[12,28–31] about the knee and hip during slow and dynamic tasks. Whereas these alterations are observed consistently, the source of these changes has not been established.

Pathologic ankle conditions result in spinal-level pathway alterations,[22,32] which can lead to feed-forward patterns that present as changes in knee and hip neuromuscular control.[11,14,27] Sedory et al[31] reported that the excitability of multiple muscle groups proximal to the ankle was altered in people with CAI, suggesting that higher brain centers may be influencing motor function. In addition, Heroux and Tremblay[33] suggested that cortical excitability is altered in the quadriceps musculature after knee injuries. However, to our knowledge, few researchers have evaluated the effects of ankle instability on corticomuscular control in this population. These theories of the influence of higher brain centers have been developed using biomechanical research tools that provide indirect information about nerve function. To fully appreciate these theories, it is necessary to directly compare the nerve pathway function between the pathologic ankles, as well as proximal to the ankles, of CAI patients and the ankles and proximal regions of healthy populations. Understanding how both spinal reflexive and cortical excitability are affected in proximal and distal musculature is important for developing multimodal interventions that can target the origins of neuromuscular dysfunction at multiple points throughout the injured extremity. Therefore, the purpose of our study was to determine if corticospinal and spinal reflexive excitability of the fibularis longus and quadriceps differed between individuals with CAI and healthy control participants. We hypothesized that both spinal reflexive and corticospinal excitability would differ in the fibularis longus and the vastus medialis between those with CAI and their healthy control counterparts.

METHODS

In this case-control study, we collected all outcome measures bilaterally and randomized the order of tests (spinal reflexive, corticospinal) and limb (dominant, nondominant). Electrodes were placed over the vastus medialis and fibularis longus during the first test and remained affixed to the skin for the second test. The investigator (M.M.M.) assessing corticospinal and spinal reflexive excitability was blinded to group assignment.

Participants

Twenty-six CAI and 26 control participants volunteered. During the study, outcomes for 5 CAI patients and 2 control participants either could not be elicited or were unusable, yielding 21 CAI patients and 24 control participants (Table 1). No participant had a history of orthopaedic injury or operation to the knee or hip joints or fracture of a lower extremity bone. We excluded individuals who had a head injury in the 12 months or concussion in the 6 months before the study; history of vestibular disorder, epilepsy, stroke, cardiac condition, cancer, cranial neurosurgery, intracranial clip, psychiatric disorder, or migraine; cardiac pacemaker or implanted defibrillator; or were pregnant or breastfeeding. All CAI participants had a history of at least 1 acute lateral ankle sprain resulting in swelling, pain, and temporary loss of function but not within the 3 months before the study. They also reported more than 2 episodes of the ankle "giving way" in the 6 months before the study. All CAI participants scored 80% or less on the Foot and Ankle Ability Measure (FAAM) Sport instrument.[34] Control participants had no history of injury to either ankle and scored 100% on the FAAM Sport. Control participants were matched based on age, sex, and limb dominance. *Limb dominance* was defined as the limb that participants preferred to kick a ball. An "involved" limb that corresponded with a CAI patient's involved limb was assigned to each control participant. All participants were instructed to refrain from consuming caffeine within the 12 hours before the procedures started. All participants provided written informed consent, and the study was approved by The University of Toledo Institutional Review Board.

Spinal Reflexive Excitability

For spinal reflexive excitability testing of the vastus medialis, participants were instructed to lie supine on a padded plinth. A 2-mm shielded disc-stimulating electrode (model EL254S; BIOPAC Systems, Inc, Goleta, CA) was positioned over the femoral nerve during vastus medialis testing and over the sciatic nerve in the popliteal space for fibularis longus testing. A 5-cm, round, self-adhesive ground electrode (DURA-STICK II; Chattanooga Group, Hixson, TN) for the stimulating electrode was placed over the hamstrings musculature during vastus medialis testing and over the quadriceps during fibularis longus testing.

Analog-to-digital signal conversion was processed with a 16-bit convertor (model MP150; BIOPAC Systems, Inc). We used Acqknowledge BIOPAC software (BIOPAC Systems, Inc) that interfaced with a 200-V maximum stimulus isolation adaptor (STIMISOC; BIOPAC Systems, Inc) to visualize the signals and to manipulate the stimulus. Electromyographic (EMG) signals were sampled at 2000 Hz with amplification set at a gain of 1000 (model EMG100C; BIOPAC Systems, Inc). We measured peak-to-peak Hoffmann reflexes (H-reflexes) and increased the stimulus intensity by increments of 2 V until a maximal H-reflex was observed. Three H-reflexes were recorded, averaged, and normalized to the maximal muscle response (M), theoretically representing the ratio of the motoneuron pool reflexively activated to the amount of the motoneuron pool available (H : M ratio). Maximal muscle responses were determined after identification of the H-reflex by continuing to increase the stimulus until M-wave amplitude was elicited. Higher H-reflex values and H : M ratios indicated greater spinal reflexive excitability.

Corticospinal Excitability

Two 10-mm pregelled Ag/AgCl electromyography electrodes (BIOPAC Systems, Inc) were positioned 1.75 mm apart over the bellies of the vastus medialis and the fibularis longus muscles and 2 to 3 cm distal to the fibular head. A ground electrode was placed over the medial malleolus of the nondominant limb. Patients wore Lycra (Invista, Wichita, KS) swim caps that we used to mark reference lines for placement of the stimulating coil. We drew 1 line bisecting the hemisphere sagitally and 1 line bisecting this line from the apex of 1 ear to the other. The lines intersected over the vertex of the skull, and we used the intersection as our reference point to the motor cortex. Participants wore disposable earplugs (Aearo Company, Indianapolis, IN) to muffle the sound of the stimulation.

Assessment of corticospinal excitability was performed using TMS over the motor cortex. Transcranial magnetic stimulation produces a brief magnetic stimulus from a coil placed over the scalp that excites brain tissue and descending neural tissue, eliciting motor-evoked potentials (MEPs) in the peripheral musculature. To determine the position at which TMS would be performed for each muscle, a double-cone coil (Magstim Company Ltd, Whitland, Carmarthenshire, Wales, UK) was positioned over the contralateral vertex of the cranium relative to the test limb.[35] A Magstim Rapid stimulator (Magstim Company Ltd) was used to produce a maximum magnetic stimulus of 1.4 T. The coil was moved anteriorly or posteriorly approximately 1 cm until the greatest MEP was elicited at a constant stimulus intensity. The coil was secured against the scalp at this position using supporting clamps and remained there during testing for each muscle.

During corticospinal excitability testing of the vastus medialis, participants were seated in an isokinetic dynamometer (System II Pro dynamometer; Biodex Medical Systems, Shirley, NY) with hips flexed to 85°, knees flexed to 90°, and the distal lower leg secured to a padded movement arm. The testing position of the fibularis longus was the same as for the vastus medialis except that the knee and ankle were flexed to 10° with the calcaneus secured in a rubber heel cup mounted on a flat platform. To standardize volitional muscle contraction during corticospinal testing procedures, participants performed contractions (knee extension and ankle plantar flexion, respectively) at 5% of their maximal voluntary isometric contractions (MVICs) with their upper extremities crossed over their chests.

Active motor threshold (AMT) was determined as the lowest intensity required to elicit an MEP peak-to-peak amplitude that was 100 μV or greater in at least 5 of 10 trials. It provides an estimate of excitability of intracortical synapses and descending interneuronal relays.[36] A higher AMT indicates less excitability, as greater stimulus intensity was required to elicit an MEP of at least 100 μV. The MEP amplitudes provide a measure of the magnitude of corticospinal tract excitability,[36] which we normalized to maximal M (MEP : M ratio) measured during spinal reflexive excitability testing. Larger MEP : M ratios corresponded to greater excitability. After determining AMT, we recorded and averaged 5 MEPs at stimulus intensities of 100%, 105%, 110%, 120%, 130%, and 140% of AMT. Recording MEP amplitudes at these intensities is considered a *stimulus response*, which indicates if MEP amplitude increases as TMS intensity increases.[37] The increase in the MEP amplitude has been attributed to the stimulation of inherently less excitable neurons in the motor cortex.

Data Analysis

Independent-samples *t* tests were performed to examine differences between CAI and control participant demographics. We conducted 8 separate 2 × 2 (limb × group) analyses of covariance, with age entered as a covariate, for each outcome measure: bilateral vastus medialis and fibularis longus AMT; MEPs at 100%, 105%, 110%, 120%, 130%, and 140% AMT; and H : M ratio. We set the α level a priori at .05. Tukey post hoc multiple comparison tests were applied when we observed interactions.

RESULTS

Our control participants were older than the CAI patients ($t_{43} = -2.496$, $P = .02$, Table 1).We noted no limb × group interaction effects for spinal reflexive and corticospinal excitability of the vastus medialis and fibularis longus muscles. No group effects existed for corticospinal excitability in the vastus medialis (Table 2). We observed group effects, indicating greater corticospinal excitability in the fibularis longus of the control group, at 100% ($F_{1,41} = 4.551$, $P = .04$) and 105% of AMT ($F_{1,35} = 4.782$, $P = .04$; Table 3).

Table 2. Active Motor Thresholds, Motor-Evoked Potential Amplitudes, and Spinal Reflexes of the Vastus Medialis

		Group						
		Chronic Ankle Instability			Healthy Control			
		Mean ± SD			Mean ± SD			
Variable		n	Involved Limb	Uninvolved Limb	n	Matched Involved Limb	Matched Uninvolved Limb	*P* Value	*F* Value
Active motor threshold, % T		21	40.10 ± 13.11	39.48 ± 13.76	24	40.79 ± 10.24	40.17 ± 7.61	.73	0.125
Motor-evoked potential, μV[a]									
Active motor threshold, %									
	100	19	0.019 ± 0.027	0.024 ± 0.030	23	0.017 ± 0.027	0.018 ± 0.019	.75	0.099
	105	19	0.031 ± 0.054	0.031 ± 0.045	23	0.023 ± 0.023	0.024 ± 0.038	.55	0.372
	110	18	0.026 ± 0.036	0.033 ± 0.046	23	0.030 ± 0.038	0.031 ± 0.038	.78	0.078
	120	16	0.029 ± 0.033	0.043 ± 0.050	22	0.037 ± 0.035	0.048 ± 0.057	.41	0.706
	130	15	0.038 ± 0.039	0.066 ± 0.082	22	0.053 ± 0.046	0.076 ± 0.085	.33	0.964
	140	15	0.055 ± 0.055	0.071 ± 0.070	22	0.087 ± 0.098	0.101 ± 0.115	.22	1.558
Hoffmann : maximal muscle response ratio		18	0.348 ± 0.287	0.286 ± 0.241	22	0.362 ± 0.414	0.353 ± 0.482	.75	0.103

[a] Values are normalized to maximal muscle response.

DISCUSSION

We observed that MEP amplitudes were lower bilaterally at 100% of AMT and at 105% of AMT in the fibularis longus of the CAI group than in the control group. No differences were evident in MEP amplitudes at the remaining intensities from 110% to 140% of AMT of the stimulus response curve for the fibularis longus. No other differences were noted bilaterally between the CAI and control groups for AMT or spinal reflex excitability for the fibularis longus. We observed no differences between the CAI and control groups for spinal reflexive or corticospinal excitability for the vastus medialis.

Decreased MEPs at lower TMS intensities (100% and 105% of AMT) indicated that patients with CAI may generate smaller-amplitude motor responses in the fibularis longus with low levels of excitation to the primary motor cortex.[38] Smaller amplitudes suggested that a smaller portion of the fibularis longus motoneuron pool that arises from the primary motor cortex was excited with low levels of TMS. Decreased MEPs at 100% and 105% of AMT in CAI participants may have indicated inhibited motor output during rhythmic movements, such as walking or running, that require activation of the cortical neurons of the primary motor complex.

Patients with functional ankle instability demonstrated an inability to reproduce force output during eversion contractions at 30% or less of maximal voluntary effort compared with control participants.[39] In addition, increased self-reported disability was associated with more errors when attempting to contract ankle-evertor muscles to match force loads at a low percentage of MVIC.[40,41] The corticospinal differences that we observed for the fibularis longus at lower stimulus intensities (100%–105% of AMT) may have been the mechanism dictating the inability of CAI patients to accurately reproduce sufficient voluntary muscle tension during submaximal contractions. The inability to generate adequate eversion muscle contractions when low percentages of maximal contraction capacity are necessary, such as during gait, may be related to the risk of multiple ankle sprains during common activities of daily living.

Given the retrospective nature of a case-control design, the causality between decreased corticospinal excitability and pathologic ankle conditions remains unknown. Resting motor thresholds of the fibularis longus were bilaterally

Table 3. Active Motor Thresholds, Motor-Evoked Potential Amplitudes, and Spinal Reflexes of the Fibularis Longus

		Group						
		Chronic Ankle Instability			Healthy Control			
		Mean ± SD			Mean ± SD			
Variable		n	Involved Limb	Uninvolved Limb	n	Matched Involved Limb	Matched Uninvolved Limb	*P* Value[a]	*F* Value
Active motor threshold, % T		21	55.48 ± 9.21	52.24 ± 8.88	24	54.21 ± 10.21	52.25 ± 10.91	.93	0.008
Motor-evoked potential, μV[b]									
Active motor threshold, %	100	20	0.014 ± 0.008[c]	0.015 ± 0.007[c]	24	0.023 ± 0.031	0.021 ± 0.022	.04	4.551
	105	18	0.021 ± 0.009[c]	0.023 ± 0.013[c]	20	0.029 ± 0.026	0.034 ± 0.037	.04	4.782
	110	16	0.033 ± 0.019	0.035 ± 0.020	18	0.032 ± 0.021	0.043 ± 0.061	.62	0.250
	120	11	0.050 ± 0.030	0.058 ± 0.041	12	0.064 ± 0.059	0.073 ± 0.077	.15	2.237
	130	5	0.072 ± 0.055	0.095 ± 0.095	10	0.086 ± 0.044	0.099 ± 0.092	.63	0.248
	140	4	0.090 ± 0.061	0.130 ± 0.119	5	0.081 ± 0.073	0.144 ± 0.164	.71	0.150
Hoffmann : maximal muscle response ratio		20	0.297 ± 0.175	0.270 ± 0.159	24	0.262 ± 0.178	0.31 ± 0.207	.29	1.139

[a] *P* values represent group effects.
[b] Values are normalized to maximal muscle response.
[c] Indicates difference between chronic ankle instability and healthy control groups ($P < .05$).

higher in patients with unilateral CAI than in control participants,[23] suggesting that unilateral injury may cause bilateral alterations of corticospinal excitability or that these alterations were present before injury and potentially led to the development of CAI. Bilateral deficits in quadriceps neuromuscular control have been reported after unilateral anterior cruciate ligament injury,[42] suggesting that bilateral neuromuscular alterations are common after unilateral injury at joints in addition to the ankle. Pietrosimone et al[43] suggested that unilateral therapeutic exercise in patients with chronic pathologic knee conditions will benefit the contralateral limb, indicating that changes in neuromuscular control on 1 side of the body will affect the contralateral limb. Research is needed to determine the origins of bilateral deficits after unilateral ankle injury and the most effective methods for treatment.

During more demanding activities, increased corticospinal excitability may be required. We found no difference between limbs or between groups in corticospinal excitability of the fibularis longus at stimulation intensities greater than 105% of AMT, suggesting that CAI patients exhibit voluntary control of the fibularis longus similar to control participants during higher levels of corticospinal stimulation. Whereas CAI patients and control participants exhibited similar motor output during high levels of corticospinal stimulation, CAI patients may need to generate greater-than-normal corticospinal excitability to overcome mechanical insufficiencies. The CAI patients with greater mechanical insufficiencies may have to initiate greater cortical control over movement to prevent sprains due to increased ankle laxity. Joint laxity is not universal among CAI patients, and our lack of laxity measures limited this interpretation. However, this presents direction for future investigation.

Interestingly, we did not find differences in spinal reflex or corticospinal excitability between limbs or groups in the quadriceps musculature. Sedory et al[31] observed differences in voluntary quadriceps activation in CAI patients. Voluntary quadriceps activation is dictated by the recruitment and firing rate of motor units during an MVIC.[44] We conducted our measurements at rest (spinal reflexes) and at 5% of an MVIC (corticospinal excitability). Therefore, the differences between our findings and those of previous researchers may be due to the outcome measures that were evaluated.

We did not observe differences in spinal reflex excitability between limbs or groups in either the vastus medialis or fibularis longus. Researchers[18,22] have reported decreased excitability in the fibularis longus of patients with chronic pathologic ankle conditions. They have suggested that decreased spinal reflex excitability of the fibularis longus is important,[18,32] as this muscle may slow ankle inversion and decrease the incidence or extent of injury related to lateral ankle sprains. Investigators[45,46] evaluating acute lateral ankle sprains have reported observations that were similar to ours, with no difference in spinal reflex excitability of the fibularis longus between groups or limbs. Whereas differences between outcomes in spinal reflex excitability of the fibularis longus in previous studies commonly were hypothesized to be due to different lengths of time since injury,[46] these differences also may be due to variations in the nervous system response to injury in individual CAI patients. Many researchers[18,22,45,46] have evaluated spinal reflex excitability after ankle injury in relatively small cohorts of injured patients ($N < 30$). In future studies with larger sample sizes, investigators may be able to determine if subgroups that display particular clusters of similar neurophysiologic alterations, which may manifest in specific patterns of spinal reflexive and corticospinal adaptations, can be identified after ankle injury. Identifying the neuromuscular patterns that predict chronic disability or ability to cope after acute ankle injury may help to direct more individualized rehabilitation.

In addition to our small sample size, our study had other limitations. Our TMS instrumentation limited the stimulus intensity delivered to no greater than 1.4 T. Given that a portion of our participants had high AMT intensities, we could not obtain MEPs throughout the entire stimulus-response curve, as doing so would have required a stimulus intensity exceeding 1.4 T. We could not include complete MEP stimulus-response curve data (up to 140% of AMT) for participants who demonstrated AMTs greater than 51%. Furthermore, we did not establish how spinal reflexive and corticospinal excitability relate to mechanical ankle laxity or self-reported measures of disability or if excitability outcomes are associated with biomechanical outcomes during functional tasks.

Researchers should evaluate the effect of alterations in the excitability pathways on the physical performance of athletic tasks and activities of daily life. One limitation of our study was that we do not know if alterations of these excitability measurements predict function. The goal of these case-control reports is to guide authors of future observational studies to evaluate the effect of interventions on improving disability in patients with CAI. Whereas traditional rehabilitation does not attempt to target excitability deficits, new rehabilitation paradigms have suggested that targeting excitability alterations may help to improve aberrant biomechanics and lead to better nonoperative therapeutic outcomes. New techniques using disinhibitory modalities to restore both spinal reflexive and corticospinal activity are being developed, and early efforts focused on the knee have demonstrated better outcomes than with traditional exercise.[47] Researchers should incorporate similar techniques into ankle rehabilitation strategies to manipulate excitability and better stabilize the lower extremity after ankle injury. They also should aim to determine if the degree of self-reported disability and instability within CAI patients relates to patterns of neuromuscular alterations. This may provide greater insight into the phenomenon of CAI and influence the development of optimal treatment strategies to improve outcomes in this population.

CONCLUSIONS

Corticospinal excitability in the fibularis longus was higher at TMS intensities of 100% and 105% of AMT in the control group bilaterally than in the CAI group. We did not observe differences between groups at TMS intensities of 110% of AMT or greater. No differences existed between groups for spinal reflexive excitability of the fibularis longus or the quadriceps musculature or for corticospinal excitability of the quadriceps.

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