Brain Regulation of Muscle Tone in Healthy and Functionally Unstable Ankles

Alan R. Needle, Jacqueline A. Palmer, Trisha M. Kesar, Stuart A. Binder-Macleod, and C. Buz Swanik

Context: Current research into the etiology of joint instability has yielded inconsistent results, limiting our understanding of how to prevent and treat ligamentous injury effectively. Recently, cortical reorganization was demonstrated in patients with ligamentous injury; however, these neural changes have not been assessed relative to joint laxity. Objective: The purpose of the current study was to determine if changes in cortical excitability and inhibition occur in subjects with functional ankle instability, as well as to investigate the relationship between these measures and joint laxity. Design: Posttest only with control group. Setting: University laboratory. Subjects: 12 subjects with no history of ankle sprain (CON) and 12 subjects with a history of unilateral functional ankle instability (UNS). Interventions: Subjects were tested for joint laxity using an instrumented ankle arthrometer. Cortical excitability and inhibition were assessed using transcranial magnetic stimulation (TMS) to obtain motor-evoked potentials and the cortical silent period from the lower leg muscles. Main Outcome Measures: Joint laxity was quantified as peak anterior displacement and inversion rotation. Active motor threshold, slope, and intensity at 50% of peak slope of TMS-derived recruitment curves were used to quantify cortical excitability from lower leg muscles, while the cortical silent period from the peroneus longus was used to represent intracortical inhibition. Results: No significant differences were observed between groups for laxity or cortical measures. CON demonstrated a significant relationship between laxity and tibialis anterior excitability, as well as laxity and silent period, while UNS ankles demonstrated significant relationships between peroneal and soleus excitability and laxity measures. Conclusion: Our results support relationships between laxity and measures of excitability and inhibition that differ between healthy and unstable subjects. Future research should further investigate the mechanisms behind these findings and consider cortical influences when investigating altered joint laxity.

Keywords: transcranial magnetic stimulation, cortical excitability, cortical inhibition, neuromechanical decoupling

The etiology behind recurrent joint instability, or sensations of “giving way” occurring after joint injury, has eluded researchers because alterations in mechanical laxity, joint proprioception, and neuromuscular control have inconsistently correlated with measures of joint stability and function. As a result, current treatment paradigms designed to minimize reinjury risk and prevent functional instability may be ineffective or inefficient. Research has recognized multiple neurological phenomena, including peripheral deafferentation and arthrogenic inhibition, that occur after joint injury; however, exploration of changes to the cerebral cortex is very limited. The brain has a well-established role in the regulation of muscle tone and joint stiffness, which is of utmost importance for the maintenance of joint stability. Thus, as neuromechanical coupling is deeply embedded in our motor-control strategies to prepare for and react to injurious forces, it is imperative to investigate how the brain’s regulation of dynamic joint restraint may be altered after injury.

Preliminary research has indicated that plastic changes occur in the brain after knee-joint injury, yet very few studies have investigated cortical activity in other joint-instability models. Ankle sprains are the most common injury observed in physically active people, with complaints of functional instability or recurrent “rolling” events developing in approximately 50% of patients. As functional instability may be present across various ranges of joint laxity and multiple ligaments, the ankle offers unique insights to the investigation of correlations between the central nervous system and the regulation of muscle tone. These relationships may provide a greater understanding of how the nervous system influences joint stability.
system controls joint stiffness and the dynamic restraint mechanism and whether neuromechanical decoupling occurs after injury.

Transcranial magnetic stimulation (TMS) is a well-established method used to investigate cortical excitability and inhibition. Consisting of brief magnetic pulses used to induce an electrical field in the brain, TMS has multiple uses for examining cortical function and measuring neuroplasticity. One common practice involves administering controlled pulses over the primary motor cortex and measuring the muscle response, or motor-evoked potential (MEP), through electromyographic recordings. The size and regulation of these responses across a range of stimulus intensities (stimulus-response curve) demonstrates the excitability of the primary motor cortex. Cortical excitability represents the ease with which motor neurons fire to control specific muscles, which in turn regulates muscle tone and joint stiffness. Increased cortical excitability to the quadriceps has been reported in patients with a deficient anterior cruciate ligament (ACL). Decreased excitability to the peroneus longus was observed in patients with functional ankle instability. These conflicting results may indicate differing roles of the bony architecture and capsuloligamentous and dynamic restraints that are associated with complaints of instability at the knee versus the ankle joint. To date, no studies have comprehensively investigated multiple parameters of the stimulus-response curve or changes to intracortical inhibition in the context of joint-laxity measures.

TMS may also be used to quantify cortical inhibition through measurement of the cortical silent period (CSP), a brief pause in voluntary muscle activity after the magnetic pulse. The CSP represents inhibitory pathways between the motor cortex, thalamus, and basal ganglia, where a shorter CSP is associated with less inhibition, higher facilitatory descending drive, and greater muscle tone. Although the CSP has been largely studied among patho-neurologic populations, such as patients with dystonia or cerebral palsy, intracortical inhibition may also play a profound role in the maintenance of joint stability by optimizing muscle tone in neurologically intact individuals. No differences in CSP have been observed from the quadriceps of ACL-deficient patients, but no reports exist on patients with unstable ankles or how the CSP duration may relate to joint laxity.

With increasing evidence emerging regarding cortical influences in patients with a history of joint injury, it is important to determine whether changes in corticomotor excitability are associated with alterations in joint laxity and stiffness. Variables derived from TMS have been related to the regulation of muscle tone in neurological populations; however, no studies have investigated its relationship to joint stiffness in populations with musculoskeletal pathologies. The purpose of this study was therefore to explore the relationship between ankle-joint laxity and measures of cortical excitability and inhibition and to observe if a decoupling between the neurological and mechanical properties of the joint may occur in patients with a history of ankle injury.

**Methods**

**Participants**

Twenty-four subjects from a university community were recruited for this study. After signing a university-approved informed-consent form, subjects were stratified into 2 groups using an ankle-injury history questionnaire and the Cumberland Ankle Instability Tool (CAIT). Control (CON) subjects had no history of ankle injury and a CAIT score greater than 27. Functionally unstable (UNS) subjects had a history of unilateral ankle injury and a CAIT score below 25. Subjects were excluded if they did not fit these group criteria or had a history of bilateral ankle injury, any fracture or surgery to either lower extremity, or injury to the lower extremity within 6 months of testing or met any exclusion criteria for TMS, including a history of seizures, electronic or metal implant, concussion within 6 months, current treatment for any psychiatric disorder, current pregnancy, or any other neurological disorder.

**Instrumentation**

Subjects were assessed for ankle laxity using an instrumented ankle arthrometer (Blue Bay Research, Milton, FL). The arthrometer consists of a load cell attached to an instrumented handle that detects anteroposterior force and inversion-eversion torque and a footplate connected to a shin pad via a 6-degree-of-freedom kinematic linkage system. Subjects were assessed for cortical excitability and inhibition using a magnetic stimulator (MagStim 200, MagStim Ltd, Wales, UK) with a double-conical coil. Electromyographic (EMG) recordings were obtained from the lower leg muscles using a 6-channel EMG system with active electrodes (B&L Engineering, Santa Ana, CA). Custom LabVIEW software (National Instruments, Austin, TX) was developed to set TMS intensities, trigger the stimulator, and collect EMG data. The order of testing (arthrometry or TMS first, left or right limb first) was counterbalanced across groups.

**Procedures**

For assessment of ankle laxity, subjects lay supine on a padded table with the calf supported in padding. The foot was secured to the arthrometer with heel and dorsal clamps, and the shin pad was secured to the lower leg. Three anterior translations to 130 N of force were applied, followed by 3 inversion rotations to 4.2 Nm of torque. Procedures were repeated on both limbs. Peak anterior displacement (mm) and peak inversion rotation (°) were extracted from arthrometry data using custom LabVIEW software.
For assessment of cortical excitability and inhibition, subjects were seated with the test leg secured (Figure 1). Surface EMG electrodes were placed on skin overlying the tibialis anterior, peroneus longus, and soleus muscles using standard preparation procedures. All subjects were asked to wear an elastic cap and familiarized to the TMS to reduce apprehension associated with testing. Once familiarized, the “hot spot,” (defined as the location where the maximum peak-to-peak MEPs from the muscles are observed) was located by identifying the vertex of the skull and placing the coil 1 cm anterior and 1 cm lateral. With the coil in this position, the intensity of the stimulator was increased gradually to determine the point at which a small muscle contraction was visible in the contralateral tibialis anterior. The intensity was then reduced by 5%, and the coil was moved in approximately a 5-mm radius to determine the point at which the largest MEP was observed on EMG. This spot was marked on the elastic cap and used for all recordings on that side.32

Once the hot spot was identified, a series of approximately 60 magnetic pulses, with intensities ranging from below an observable MEP to above the point of a maximal response for that individual, were applied in randomized order (to control for subject anticipation), with 5 seconds between pulses.21 Throughout this procedure, subjects were asked to pronate the ankle at 15% of maximal effort with real-time biofeedback of peroneus longus activity provided on a screen directly in front of the subject, to allow for calculation of the CSP. The peroneus longus was selected due to its role in maintaining dynamic ankle stability. Test procedures were repeated for each leg, with the order randomized.

**Data Analysis**

For extraction of cortical-excitability variables, peak-to-peak amplitudes of the MEPs were normalized to the largest observed MEP during the session and plotted against the stimulus intensity to form a stimulus-response curve (Figure 2). A curve was fitted to these data using a Levenberg-Marquardt nonlinear fit with a modified Boltzmann equation21:

\[
y = \frac{\text{MEP}_{\text{min}} + \text{MEP}_{\text{max}} - \text{MEP}_{\text{min}}}{1 + e^{m(I_{50}-I)}}
\]

This fit provided estimates of the minimum MEP (\(\text{MEP}_{\text{min}}\)), maximum MEP (\(\text{MEP}_{\text{max}}\)), slope (\(m\)), and the intensity at which the curve’s slope was the steepest (\(I_{50}\)). Using the fitted curve, the active motor threshold was calculated as the point at which the curve’s slope increased above 5% of its maximum. An \(R^2\) value greater than or equal to .75 was determined to be an acceptable fit.21 CSP was calculated only for the peroneus longus muscle (as precontraction was regulated in this muscle) and was manually determined from the end of the MEP until the resumption of baseline EMG activity (Figure 3).26 CSP was plotted against the stimulus intensities, and the maximum silent period, or the point at which the CSP plateaued, was extracted and used for analysis.

For analysis, asymmetry indices (AIs) between the involved and uninvolved sides were calculated using the following equation33: \(\text{AI} = (\text{uninvolved} - \text{involved}) / (\text{uninvolved} + \text{involved})\). These indices were used to normalize the difference that occurred between sides across subjects. A positive asymmetry index indicates that the value on the uninvolved side is higher, a negative value indicates that the involved side is higher, and if both sides are equal, the asymmetry index will equal zero. The involved limb for CON subjects was matched by gender and limb dominance to the UNS group. As measures of excitability and laxity are highly variable across a population, these indices provided within-subject normalization to an uninvolved side.33
Asymmetry indices for anterior displacement and inversion rotation between groups were compared using t tests. Asymmetry indices for TMS variables including active motor threshold, \( I_{50} \), and \( m \) were compared using a 2-way ANOVA with 1 within-subject factor (muscle, 3 levels) and 1 between-subjects factor (group, 2 levels). Asymmetry indices for CSP of the peroneus longus muscle between groups were compared using a t test. Pearson product–moment correlation coefficients were used to assess the relationships between laxity and TMS variables in each group. Fisher \( r \)-to-\( z \) transformations were used to determine if statistical differences existed in correlations across groups. An a priori level of significance (\( \alpha \)) was set at .05.

**Results**

There were no differences in group demographics between groups (Table 1). There were no significant between-groups differences (CON vs UNS) observed for laxity (Table 2) or TMS variables (Table 3).

Several significant correlation coefficients were revealed between laxity and excitability variables (Table 4). In the CON group, a significant positive correlation was observed between anterior displacement and tibialis anterior motor threshold (\( r = .667, P = .025 \)). A significant
Table 1  Group Demographics, Mean ± SD

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Unstable ankle</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (n)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>male</td>
<td>6</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>female</td>
<td>6</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>21.2 ± 2.6</td>
<td>20.9 ± 4.1</td>
<td>.857</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172.7 ± 8.5</td>
<td>170.6 ± 10.1</td>
<td>.600</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>70.0 ± 15.0</td>
<td>72.5 ± 15.0</td>
<td>.744</td>
</tr>
<tr>
<td>CAIT</td>
<td>29.6 ± 0.7</td>
<td>19.5 ± 3.7</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Abbreviations: CAIT, Cumberland Ankle Instability Tool.

Table 2  Laxity Results Between Groups, Mean ± SD

<table>
<thead>
<tr>
<th></th>
<th>Raw Values (Involved Side)</th>
<th>Asymmetry Indices</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Unstable ankle</td>
</tr>
<tr>
<td>Anterior-posterior displacement (mm)</td>
<td>6.02 ± 1.9</td>
<td>6.94 ± 2.6</td>
</tr>
<tr>
<td>Inversion-eversion rotation (°)</td>
<td>38.8 ± 10.7</td>
<td>37.0 ± 7.1</td>
</tr>
</tbody>
</table>

Table 3  Transcranial Magnetic Stimulation Results Between Groups, Mean ± SD

<table>
<thead>
<tr>
<th>Variable</th>
<th>Muscle</th>
<th>Raw Values (Involved Side)</th>
<th>Asymmetry Indices</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Control</td>
<td>Unstable ankle</td>
</tr>
<tr>
<td>Motor threshold</td>
<td>tibialis anterior</td>
<td>36.9 ± 12.2</td>
<td>41.2 ± 13.3</td>
</tr>
<tr>
<td></td>
<td>peroneus longus</td>
<td>33.3 ± 16.0</td>
<td>38.3 ± 11.0</td>
</tr>
<tr>
<td></td>
<td>soleus</td>
<td>39.9 ± 10.5</td>
<td>39.0 ± 10.5</td>
</tr>
<tr>
<td>Intensity of peak slope</td>
<td>tibialis anterior</td>
<td>67.2 ± 15.8</td>
<td>68.6 ± 13.5</td>
</tr>
<tr>
<td></td>
<td>peroneus longus</td>
<td>68.8 ± 20.1</td>
<td>63.4 ± 10.5</td>
</tr>
<tr>
<td></td>
<td>soleus</td>
<td>66.1 ± 16.0</td>
<td>62.3 ± 10.8</td>
</tr>
<tr>
<td>Slope</td>
<td>tibialis anterior</td>
<td>0.17 ± 0.07</td>
<td>0.22 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>peroneus longus</td>
<td>0.16 ± 0.09</td>
<td>0.21 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>soleus</td>
<td>0.20 ± 0.12</td>
<td>0.21 ± 0.07</td>
</tr>
<tr>
<td>Cortical silent period, s</td>
<td>peroneus longus</td>
<td>0.17 ± 0.09</td>
<td>0.24 ± 0.04</td>
</tr>
</tbody>
</table>

Note: Raw values for motor threshold and intensity of peak slope are presented as percentage of stimulator output. Asymmetry indices and slope are unitless.

A positive correlation was also observed between anterior displacement and peroneus longus CSP ($r = .644$, $P = .040$). Among UNS subjects, significant negative correlations were observed between anterior displacement for soleus active motor threshold ($r = -.753$, $P = .012$) and slope ($r = -.670$, $P = .048$). In addition, significant negative correlations were observed between inversion rotation and peroneus longus $I_{50}$ ($r = -.733$, $P = .016$), Figure 4). Significant between-groups differences were observed in the correlations between anterior displacement and tibialis anterior active motor threshold ($z = 2.24$, $P = .02$) and between soleus active motor threshold and anteroposterior displacement ($z = -2.80$, $P = .005$). A statistical trend was observed in the correlations between anterior displacement and soleus slope ($z = -1.73$, $P = .087$).
As the etiology of joint instability continues to elude researchers, it is essential to investigate how the central nervous system changes after injury. The current study is the first to investigate cortical changes as they relate to measures of joint laxity and to observe how these relationships differ between healthy and functionally unstable ankles. Although we observed no group differences in laxity, cortical excitability, and inhibition asymmetry values, we did observe relationships among laxity and cortical excitability and inhibition variables. Furthermore, the couplings between brain function and ankle laxity were different in healthy subjects than in those with unstable ankles. This suggests changes in the neuromechanical joint properties that may represent potential predispositions, adverse effects, or compensation strategies with unstable ankles.

Differences Between Groups
Our results indicate that the groups did not differ in side-to-side symmetry for laxity, excitability, or inhibition. This is consistent with previous findings suggesting that functional ankle instability may exist independent of mechanical laxity; however, our observations contradict previous research suggesting changes in excitability in patients with complaints of instability. Heroux and Tremblay

### Table 4: Correlations Between Laxity and Transcranial Magnetic Stimulation Asymmetries

<table>
<thead>
<tr>
<th>Group</th>
<th>Tibialis Anterior</th>
<th>Peroneus Longus</th>
<th>Soleus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MT</td>
<td>I₅₀</td>
<td>Slope</td>
</tr>
<tr>
<td>Control</td>
<td>AP Disp</td>
<td>.667*</td>
<td>.185</td>
</tr>
<tr>
<td></td>
<td>IE Rot</td>
<td>–.353</td>
<td>–.036</td>
</tr>
<tr>
<td>UNS</td>
<td>AP Disp</td>
<td>–.244</td>
<td>–.085</td>
</tr>
<tr>
<td></td>
<td>IE Rot</td>
<td>–.543</td>
<td>–.501</td>
</tr>
</tbody>
</table>

Abbreviations: MT, motor threshold; I₅₀, intensity of peak slope; CSP, cortical silent period; AP, Anterior-posterior; disp, displacement; IE, Inversion-eversion; rot, rotation; UNS, unstable ankle.

*Significant at $P < .05$.

Figure 4 — A significant correlation was observed between $I₅₀$ of peroneus longus and inversion rotation laxity in unstable subjects ($r = –.73$), while no significant correlation was observed among healthy subjects ($r = –.58$), suggesting an increased role of cortical influences in the regulation of joint laxity. Abbreviations: PL, peroneus longus; $I₅₀$, intensity of peak slope; CON, control; UNS, unstable ankles.
reported higher cortical motor-neuron excitability to the quadriceps in ACL-deficient subjects. While these subjects share a history of ligamentous injury, the knee represents an alternative model where a primary stabilizing ligament has been entirely ruptured and will not heal due to the neurovascular properties of the joint. Our data are also inconsistent with results from Pietrosimone et al, who observed decreased cortical excitability to the peroneus longus in patients with unstable ankles as determined through the resting motor threshold. Our research is the first study to investigate changes in multiple parameters of the stimulus-response curve in subjects with ankle instability, allowing for a broader understanding of the behavior of cortical motor neurons across a range of stimuli. In addition, while sample sizes were similar, the current study used facilitated contractions to determine the active motor threshold, as opposed to a resting motor threshold used previously in research. While both of these serve as meaningful measures of cortical excitability, the use of muscle contraction to facilitate MEPs may have made it more difficult to detect between-groups differences, as the increased motoneuron-pool activity makes it easier to provoke activation, masking baseline differences.

High variability across subjects may have contributed to the lack of significant group differences. Even across populations with no neurological impairment, it is normal to observe a wide range of values for TMS-derived stimulus-response curve parameters. This variability supports the idea that cortical excitability is highly individualized among patients and probably dependent on other variables. We attempted to control for these between-subjects differences by normalizing to an uninjured side; however, a high level of between-subjects variability was still observed in both laxity and TMS measures. These findings are consistent with previous studies that indicated no individual variable is consistently altered in subsets of unstable patients compared with healthy counterparts, as research has suggested that ankle instability may require several classification groups that should be assessed individually. Although no individual component of laxity, excitability, or inhibition differed across groups, it is important to compare how these variables might relate to each other differently in healthy populations than in those who have suffered a joint injury.

**Correlations Between Laxity and Excitability**

Among healthy ankles, less anterior displacement was related to greater tibialis anterior excitability, indicating a possible role of the tibialis anterior in regulating joint excursion. Although no studies have linked the active motor threshold to joint stiffness or muscle tone, previous studies have supported a key role of the tibialis anterior in regulating ankle dorsiflexion angle before heel strike, as this would place the ankle into a more stable position for the prevention of a roll-over event. This same relationship did not exist in unstable ankles, which suggests a potential loss of cortical regulation of joint stiffness through the tibialis anterior.

The unstable ankles demonstrated correlations between increased anterior displacement and higher soleus excitability (measured through the active motor threshold) and between increased inversion rotation and higher peroneal excitability (measured through $I_{50}$). Both of these excitability measures assess the membrane excitability of the cortical motor neurons, as opposed to increased recruitment that may be measured by the slope parameter. The laxity variables assessed in this study relate directly to the anterior-drawer and talar-tilt clinical tests and test joint excursion in the sagittal and frontal planes. Our results suggest that, in unstable ankles, higher laxity measures coexist with greater excitability of the muscle that directly resists motion in that plane (eg, increased soleus excitability was related to greater anterior laxity). These findings significantly differ from our observations of the healthy ankles, where higher excitability in a muscle not directly resisting displacement was observed to correlate with decreased joint laxity (eg, increased tibialis anterior excitability was related to less anterior laxity). Therefore, the cerebral cortices of people with ankle instability may have heightened excitability in muscles that can compensate for increased laxity and may increase muscle stiffness and the capacity to resist perturbations. This highlights a potential protective neural mechanism for stabilizing an injured joint, where a joint with less static restraint (increased mechanical laxity) may depend on faster and greater activation of motor neurons by the primary motor cortex for stabilization.

In contrast to these results, we also found that greater anterior displacement in the UNS group was correlated with lower soleus excitability as measured through the slope of the stimulus-response curve. As mentioned earlier, the slope is associated with recruitment of additional cortical motor neurons as opposed to changes in membrane excitability and has also been directly correlated to increases in muscle tone in pathoneurologic populations. Our findings are consistent with these results, as decreased laxity would be associated with increased muscle tone. However, because this relationship was only observed in unstable ankles, it could potentially demonstrate a higher dependence on cortical influences to the soleus for stabilization of the joint, a strategy that may be less efficient than the use of tibialis anterior observed in healthy individuals.

**Correlations Between Laxity and Inhibition**

A significant negative correlation existed between anterior displacement and the CSP from the peroneus longus in healthy subjects, indicating that higher inhibition is associated with more laxity. This measure of intracortical inhibition is representative of GABAergic pathways traveling between the primary motor cortex, thalamus, and basal ganglia and is necessary to decrease the descending drive that would cause overactivity of the
spinal stretch reflex and associated alpha–gamma coactivation.25 Research into pathoneurologic populations has observed that less inhibition results in abnormal increases in muscle tone as patients are no longer able to regulate their fusimotor drive, leading to spasticity.26–28 However, no studies have directly quantified the effect of inhibition on joint stiffness and muscle tone among non–neurologically impaired patients. No previous changes have been observed in the CSP of patients with joint instability14; however, no studies have collected laxity data in conjunction with measures of intracortical inhibition. While similar values existed in healthy and unstable joints, the lack of a correlation among functionally unstable patients may warrant further investigation into how functionally unstable ankles modulate cortical inhibition in accordance with their ankle laxity. Unfortunately, the sample size of this study is insufficient to draw conclusions related to between-groups differences. If regulation of muscle tone is not properly coupled to ankle laxity, the ability of the dynamic restraint system to appropriately regulate joint stiffness may be compromised.40

Limitations

While the current study provides valuable evidence of the role of muscle tone and stiffness regulation in the etiology of joint instability, it is important to acknowledge several limitations to the study. As this study is the first to investigate changes to parameters of the stimulus-response curve in this subset of patients, a convenient sample of subjects was used. A larger sample of subjects, as well as the addition of a group of subjects with a history of injury but no complaints of instability (copers) may provide valuable evidence on which strategies may be adaptive and maladaptive.41 In addition, while we attempted to control for subject variability by normalizing to an uninvolved side, previous research has suggested bilateral impairments in patients with ankle instability, so this normalization may limit the interpretation of group differences. This study also attempted to draw conclusions about cortical changes in this subset of patients; however, a stimulus was provided at the cortex, while measurements were from muscles in the lower leg. To better understand the relative contributions of the cortex and segmental levels to individualized changes, inclusion of the Hoffmann reflex to assess spinal excitability would be beneficial.42 Furthermore, as we used the peroneus longus to facilitate MEPs and measure the cortical silent period, future studies may investigate how differential activation of lower leg muscles may affect these correlations. Our results also demonstrated a relationship between inhibition and laxity; however, this only represents one pathway, while others may also be investigated with TMS.19,25

Conclusions

Our results provide evidence for a link between joint laxity and the central nervous system. Because no differences were observed in any measures of laxity or cortical excitability between groups, clinicians and researchers should be cautious in distinguishing these groups on individual mechanical and sensorimotor differences. However, our results did demonstrate potential differences in how the motor cortex regulates joint laxity in these groups. While restraint should be exercised in group generalizability, the results do provide preliminary evidence suggesting that this cortical regulation may be changing after injury or predispose individuals to recurrent joint instability. As limited research into cortical alterations has been conducted, it is difficult to hypothesize on potential rehabilitation implications. This research suggests that treatment of ankle sprains should focus on maintaining the link between the primary motor cortex and the joint’s laxity. Rehabilitation should focus on restoration of normal joint laxity and stiffness after injury. Future studies should investigate these variables prospectively or in the context of other clinical measures.

References

11. Swanik CB, Covassin T, Stearne DJ, Schatz P. The relationship between neurocognitive function


