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Cortical Activation During Single-Legged Stance in Patients With Chronic Ankle Instability

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Context: Chronic ankle instability (CAI) has been considered a neurophysiological condition, with dysfunctional somatosensory and motor system excitability. However, few researchers have explored the changes in cortical activation during balance tasks of patients with CAI.

Objective: To compare the cortical activity during single-legged stance among CAI, copers, and uninjured control participants and to compare dynamic balance across groups.

Design: Cross-sectional study.

Setting: Biomechanics laboratory.

Patients or Other Participants: A total of 22 participants with CAI (median [interquartile range]; age = 34.5 [11.0] years, height = 170.0 [15.8] cm, mass = 67.0 [16.2] kg), 17 copers (age = 27.0 [14.0] years, height = 170.0 [9.5] cm, mass = 66.5 [16.5] kg), and 21 uninjured control participants (age = 25.0 [10.5] years, height = 170.0 [11.0] cm, mass = 64.0 [16.5] kg).

Main Outcome Measure(s): Participants performed single-legged stance while cortical activation was tested with functional near-infrared spectroscopy. The peak oxyhemoglobin response of the activated cortex was calculated and compared across groups. The Y-Balance test outcomes and patient-reported outcomes were assessed and compared across groups.

Results: The CAI group had worse Y-balance test and patient-reported outcomes than the coper and uninjured control groups. Differences in the peak oxyhemoglobin response were observed for the primary somatosensory cortex (S1; $F_{2,57} = 4.347$, $P = .017$, $\eta_p^2 = 0.132$) and superior temporal gyrus (STG; $F_{2,57} = 4.548$, $P = .015$, $\eta_p^2 = 0.138$). Specifically, copers demonstrated greater activation in S1 and STG than the CAI ($d = 0.73$, $P = .034$, and $d = 0.69$, $P = .043$, respectively) and uninjured control ($d = 0.77$, $P = .036$, and $d = 0.88$, $P = .022$, respectively) groups. No differences were found in the cortical activation between CAI and uninjured control participants.

Conclusions: Copers displayed greater cortical activation in S1 and STG than CAI and uninjured control participants. Greater activation in S1 and STG suggested a better ability to perceive somatosensory stimuli and may represent a compensatory mechanism that allows copers to maintain good functional ability after the initial severe ankle sprain.

Key Words: coper, central nervous system, primary somatosensory cortex, superior temporal gyrus

Key Points

- The coper group had greater cortical activation than the chronic ankle instability and uninjured groups during single-limb stance.
- Increased cortical activation at the central nervous system level may be beneficial in enabling copers to maintain postural control after the initial severe ankle sprain.
- Activation of the primary somatosensory cortex and superior temporal gyrus during the balance task may be a potential treatment target for chronic ankle instability.

Ankle sprains are a common health problem, especially in those who are physically active. Up to 40% of patients who incur ankle sprains develop chronic ankle instability (CAI),[1] with residual symptoms such as feelings of instability, recurrent ankle sprains, and the ankle joint "giving way." Recurrent ankle sprains change the structural integrity of the ankle joint, reduce ankle stability, and cause deficits in neuromuscular control, increasing the incidence of early-onset osteoarthritis. The symptoms of CAI have marked effects on the activities of daily living[2] and should not be considered a simple injury or be treated lightly. On the contrary, CAI is a complex disorder resulting in both local and global difficulties in the sensorimotor system.

Copers are individuals who have had an initial ankle sprain, fully recovered, and not developed CAI.[3] Although researchers of most clinical and neuromuscular control CAI studies recruited uninjured individuals as the control group, copers are better choices for the control group to determine why a portion of individuals develop CAI whereas others fully recovered. Participants with CAI and copers had similar length changes in the anterior talofibular

ligament during ankle inversion and the anterior drawer test,[4] suggesting that mechanical laxity alone may not cause CAI.

Central nervous system (CNS) variations are present after acute and chronic ligamentous injuries, including ankle sprains and anterior cruciate ligament ruptures.[5] A number of authors[6–8] have identified CAI as a neurophysiological dysfunction, rather than a localized ligament injury disease, with evidence from direct neurophysiological measures of somatosensory function and corticomotor excitability. These CNS adaptations after ligamentous injury may negatively influence patient-reported outcomes and prolong full recovery.[5] Regarding somatosensory function, CAI has been associated with many sensorimotor impairments, such as impaired balance and decreased proprioception.[9] Needle et al[7] found that higher levels of load on the ankle ligaments could not be distinguished by the somatosensory cortex of those with CAI, potentially reducing the competence of the CNS in coping with joint loads. For corticomotor excitability, patients with CAI had less corticomotor excitability in the tibialis anterior muscle during single-legged stance,[8] resulting in slow motion control and a high risk of reinjury. These researchers[5–8,10] have suggested that CNS adaptation may harm the prognosis of CAI, and thus, evaluation and optimization are necessary.

Deficits in postural control are present in patients with CAI and have been demonstrated to be substantially related to the poor health-related quality of life associated with CAI.[2] The postural-control deficits of CAI are associated with the injury to the lateral ankle ligament and its somatosensory receptors.[11] These deficits also appear to be related to alterations in cortical activity.[12] However, few investigators[13] have focused on altered cortical activity in patients with CAI. Exploring the differences in cortical activity among patients with CAI, copers, and uninjured control participants during balance tasks will help to clarify the neural mechanisms of the postural-control deficit and improve balance and the prognosis in patients with CAI.

Because it is relatively robust despite motion artifact, functional near-infrared spectroscopy (fNIRS) can be used to measure blood oxygenation changes in cortical areas triggered by neural activity during a standing task.[14] Assessments of cortical activation during balance tasks allow considerable insight into the control of balance. We used oxyhemoglobin (HbO) as the main signal in this experiment because it is more sensitive than deoxyhemoglobin and has a better correlation with the blood oxygen level dependence signal,[14] which is a base standard estimate of the change in blood flow and is used in both fNIRS and functional magnetic resonance imaging measurements. Typical hemodynamic responses of HbO increase with neural activity and return to baseline afterward. The peak response of HbO is the most frequently chosen feature[15] as it is simple but effective.

Together, the evidence suggests that cortical activity is altered in patients with CAI, which may negatively affect their functional recovery. Evaluations of cortical activation could help to define CAI better than isolation of mechanical ankle instability. By comparing the cortical activation of patients with CAI and copers, we may identify the coping mechanisms absent in those with CAI, which may offer a potential therapeutic target for CAI. However, to date, researchers have not tended to focus on the CNS alterations during balance tasks due to the lack of copers as a control group and the limited availability of advanced technology. Therefore, the primary purpose of our study was to assess cortical activation during single-legged stance using fNIRS technology in individuals with CAI, copers, and uninjured control participants. Our secondary purpose was to compare dynamic balance across groups. We hypothesized that the copers and uninjured control groups would display greater activation than the CAI group in the cortical regions associated with the balance task. Additionally, we hypothesized that the coper and uninjured control groups would have better Y-balance test (YBT) outcomes than the CAI group.

METHODS

Design

We used a cross-sectional research design to compare variables across 3 groups. To determine their eligibility, we instructed participants to complete an online questionnaire. Sixty individuals (27 men and 33 women) participated in this study (Table 1). According to the recommendations of the International Ankle Consortium,[16] participants were entered into the CAI group (n = 22) if (1) they had a history of at least 1 significant lateral ankle sprain; (2) the initial sprain occurred ≥12 months before study enrollment; (3) the most recent injury had occurred more than 3 months before study enrollment; (4) they had a history of the previously injured ankle joint "giving way," recurrent sprain, "feelings of instability," or a combination of the 3; and (5) they had Cumberland Ankle Instability Tool (CAIT) scores <24. Inclusion criteria for the coper group (n = 17) were (1) a history of a moderate to severe lateral ankle sprain, including inflammatory symptoms (pain, swelling, discoloration, or being unable to bear full weight); (2) a return to at least moderate levels of weight-bearing physical activities without episodes of giving way, recurrent ankle sprain, or feelings of instability for ≥12 months; and (3) Foot and Ankle Ability Measure (FAAM) Activities of Daily Living subscale scores ≥99%, and FAAM Sports subscale scores ≥97%. Inclusion criteria for the control group (n = 21) were (1) no history of a lateral ankle sprain, (2) no history of the ankle giving way, and (3) CAIT scores ≥28.

No participant had a history of (1) previous surgery to either limb of the lower extremity; (2) previous avulsion or other fracture to either limb of the lower extremity requiring reduction; (3) acute injury to the musculoskeletal structures of other joints of the lower extremity in the previous 3 months that affected joint integrity and function and resulted in ≥1 interrupted day of desired physical activity; (4) diagnosed balance or vestibular disorder; or (5) diagnosed neurologic or mental condition. This study was approved by the ethics committee of Huashan Hospital, Shanghai, China (HIRB 2016M-008), and all participants signed an approved informed consent form before data collection.

Test Protocol

Patient-Reported Outcomes. The PROs used in this study were the Tegner Activity Scale, CAIT, Karlsson-Peterson score, and FAAM. The Tegner Activity Scale measures activity levels, the CAIT and Karlsson-Peterson scores reflect ankle symptoms, and the FAAM evaluates the ability to perform activities of daily living and sports.

Table 1. Demographics and Patient-Reported Outcomes

Characteristic	Group			*P* Value
	Chronic Ankle Instability (n = 22)	Coper (n = 17)	Uninjured (n = 21)	
Sex, males/females	9/13	2/15	9/12	.08
Limb dominance, dominant/nondominant[a]	14/8	12/5	21/0	.10
		Median [Interquartile Range]		
Age, y	34.5 [11.0]	27.0 [14.0]	25.0 [10.5]	.07
Height, cm	170.0 [15.8]	170.0 [9.5]	170.0 [11.0]	.26
Mass, kg	67.0 [16.2]	66.5 [16.5]	64.0 [16.5]	.35
		Mean ± SD		
Body mass index, kg/m^2	24.4 ± 3.6	24.2 ± 4.0	22.9 ± 3.5	.43
Tegner Activity Scale score	3.0 ± 2.25	5.0 ± 2.5	5.0 ± 3.0	.09
Cumberland Ankle Instability Tool score	10.0 ± 6.3[b,c]	27.0 ± 4.0	30.0 ± 1.5	<.001
Karlsson-Peterson score	61.0 ± 25.5[b,c]	100.0 ± 5.0	100.0 ± 5.0	<.001
Foot and Ankle Ability Measure score				
Activities of Daily Living	92.9 ± 10.7[b,c]	100.0 ± 0	100.0 ± 0	<.001
Sports	87.5 ± 16.4[b,c]	100.0 ± 0	100.0 ± 0	<.001

[a] Whether the test limb used by participants during the functional near-infrared spectroscopy measurement was the dominant or nondominant limb.
[b] Different from the coper group.
[c] Different from the uninjured control group.

Balance Testing. The YBT was used to evaluate dynamic balance as the participants stood on the center of the "Y" on 1 leg while pushing the indicator box with the other leg as far as possible along the following 3 directions: anterior, posteromedial, and posterolateral. Each participant performed 6 practice trials followed by 3 test trials. First, we normalized the average values of reach distances in the 3 directions to the limb length of the participant, and then we determined the mean normalized reach distances in the 3 directions and used that value as the composite reach distance. Finally, the limb symmetry index equals the value on the test side divided by the value on the contralateral side.

Functional Near-Infrared Spectroscopy Testing. Before the experiment, participants practiced single-legged stance to ensure that they could successfully perform the task. Three trials were conducted and recorded using fNIRS. Each trial consisted of a 30-second bipedal stance and a subsequent 30-second single-legged stance. Participants were allowed to raise their hands to keep balance during each task, but falling or touching down on the opposite limb resulted in a failed trial. With a failed trial, the data were discarded, the individual was given time to rest, and the task was reattempted until 3 consecutive trials were successful. Participants were allowed to fail a maximum of 3 trials. The HbO signals over the cerebral cortex of the frontal, parietal, occipital, and temporal lobes of both hemispheres were recorded using a 64-channel fNIRS system (NirScan; Danyang Huichuang Medical Equipment Co, Ltd) during the single-legged stance task (Figure 1). The wavelengths were 730 and 850 nm, and the sampling frequency was 11 Hz. Cortical activation during the task was measured via 24 sources and 24 detectors based on the internationally used 10/20 electrode distribution system (Figure 2). A 3-dimensional digitizer (Fastrak; Polhemus) was used to identify the positions of fNIRS optodes and reference points (Cz, Nz, Iz, AL, and AR) on the standard head model. The midpoint of the corresponding light source-detector pair was adopted as the position of the fNIRS channel.

For uninjured control participants, their dominant limb was the test limb. We defined the *dominant limb* as the limb with which each person preferred to kick a ball. Coper individuals used the limb with the lateral ankle sprain as the test limb. Members of the CAI group used the injured limb as the test limb, and for participants reporting bilateral instability, the limb with the lower CAIT score was the test limb.

Figure 1. Testing setup for single-legged stance with the functional near-infrared spectroscopy system affixed to the participant's head.

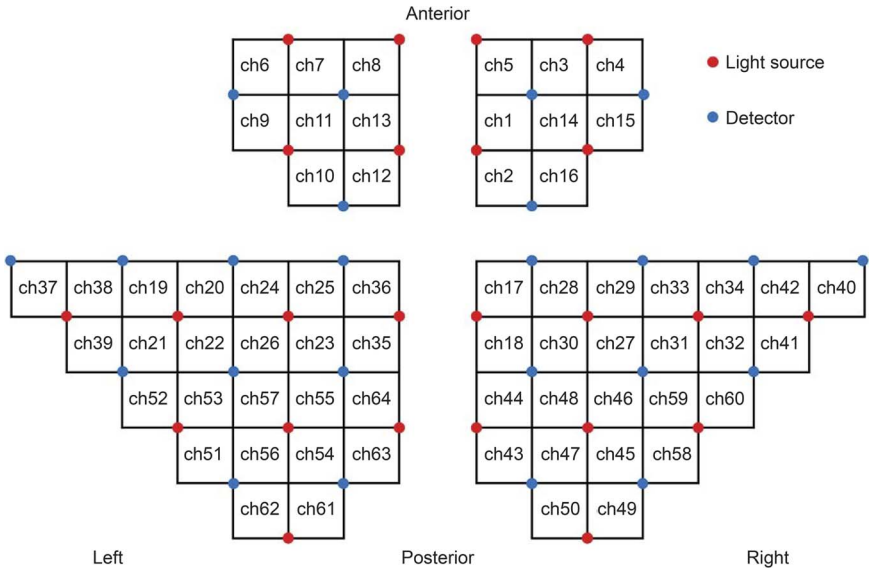

Figure 2. Montage of functional near-infrared spectroscopy. Abbreviation: ch, channel.

Data Reduction and Analysis

The differences between the task (single-legged standing) and baseline (bipedal standing) conditions were calculated to determine changes in HbO caused by the single-legged stance. To identify the activated channels during the single-legged stance, we analyzed the fNIRS data using the NIRS toolbox.[17] We developed a general linear model to estimate the task-related cortical activation and represented it with a β coefficient. Two-tailed, 1-sample Student *t* tests were calculated to identify the activated channels (β coefficient different from zero) at a group level. To avoid the error associated with multiple comparisons, we considered a threshold of a false discovery rate-corrected $q < 0.001$ statistically significant for the activated channels. Before the multiparticipant analysis, the fNIRS data of the single-legged stance performed with the left limb were flipped horizontally about the midline.

Four activated channels with precise locations in the left hemisphere were labeled regions of interest (ROIs): the left subcentral gyrus (channel 19), left primary somatosensory cortex (S1; channel 21), and left superior temporal gyrus (STG; channels 38 and 39).

The fNIRS data of the ROIs were then analyzed using the HomER2[18] toolbox of MATLAB (version R2013b; MathWorks Inc). The channels were deleted if their signal was too weak or too strong or if their SD was too great using the enPruneChannels function. The raw data were converted to optical density. Motion artifacts were marked and corrected. Next, we applied a bandpass filter between 0.01 and 0.1 Hz to avoid the effects of baseline drift and physiological noise. Finally, the optical density was converted to concentrations, and the block average was calculated given the stimulation conditions over the time range.

The peak response of HbO was computed. The peak response was the difference between the maximum value of HbO in the 45 seconds after the trial onset and the value of HbO at onset.

Statistical Analysis

Shapiro-Wilk tests identified normal distributions for peak values of HbO. All other demographic, patient-reported, and Y-balance outcomes were nonnormally distributed. Due to the presence of nonnormal distributions in most variables, we conducted between-groups comparisons via separate nonparametric Kruskal-Wallis tests. In the event of a significant Kruskal-Wallis test, we used separate Mann-Whitney U tests for pairwise comparisons. Post hoc tests were performed using the Bonferroni method. A χ^2 test was calculated to determine if a sex difference was present among groups. Because the test limb was not always the dominant limb, we used a χ^2 test to assess the differences in limb dominance between groups. Analysis of variance was computed to detect differences in peak HbO values among the groups, and η_p^2 ($\geq$0.01, *small*; $\geq$0.06, *medium*; $\geq$0.14, *large*) was determined. Post hoc tests were performed using the Bonferroni method and Cohen *d* ($\geq$0.2, *small*; $\geq$0.5, *medium*; $\geq$0.8, *large*). Significance was established at $P < .05$. All statistical tests were conducted using SPSS (version 26.0; IBM Corp).

RESULTS

The demographic data and PROs are shown in Table 1. The 3 groups were homogeneous for age, limb dominance, weight, and physical activity. The CAI group had worse PROs than the coper and uninjured control groups.

A between-groups comparison of YBT outcomes is provided in Table 2. The CAI group had a lower limb symmetry index and reach distance in the anterior direction than copers ($P = .002$ and $P < .001$, respectively) and the control group ($P = .003$ and $P < .001$, respectively). The CAI group had a lower composite reach distance than the copers ($P = .003$).

Cortical activation of the 4 ROIs is available in Table 3 and Figure 3. We found no differences in activation of the subcentral gyrus ($F_{2,57} = 2.340$, $P = .106$, $\eta_p^2 = 0.076$; Figure 3A) and STG detected by channel 38 ($F_{2,57} = 2.971$, $P = .059$, $\eta_p^2 = 0.096$; Figure 3C). Differences in activation in S1 ($F_{2,57} = 4.347$, $P = .017$, $\eta_p^2 = 0.132$; Figure 3B) and STG detected by channel 39 ($F_{2,57} = 4.548$, $P = .015$, $\eta_p^2 = 0.138$; Figure 3D) were found among groups. Post hoc analysis with Bonferroni correction revealed that copers had increased S1 activation compared with the CAI ($d = 0.73$,

Table 2. Y-Balance Test Outcomes

Y-Balance Test Measure	Group, Mean ± SD, %			*P* Value
	Chronic Ankle Instability (n = 22)	Coper (n = 17)	Uninjured (n = 21)	
Anterior direction, %LL	63.3 ± 8.3[a,b]	74.1 ± 8.5	72.6 ± 6.1	<.001
Limb symmetry index	93.9 ± 7.1[a,b]	99.8 ± 4.8	99.5 ± 4.7	.002
Posteromedial direction, %LL	112.6 ± 8.9	117.6 ± 9.1	113.4 ± 9.2	.207
Limb symmetry index	98.4 ± 4.5	99.0 ± 3.6	99.4 ± 4.2	.717
Posterolateral direction, %LL	115.7 ± 6.3	122.3 ± 8.9	118.2 ± 8.4	.066
Limb symmetry index	99.0 ± 3.2	100.1 ± 4.3	100.1 ± 3.1	.496
Composite, %LL	97.2 ± 6.9[b]	104.7 ± 7.8	101.4 ± 6.9	.013
Limb symmetry index	97.5 ± 3.5	99.6 ± 3.0	99.6 ± 1.9	.053

Abbreviation: LL, leg length.
[a] Different from the uninjured control group.
[b] Different from the coper group.

$P = .034$) and uninjured control ($d = 0.77$, $P = .036$) groups. Copers also demonstrated increased STG activation compared with the CAI ($d = 0.69$, $P = .043$) and uninjured control ($d = 0.88$, $P = .022$) groups. We observed no differences in S1 and STG activation between participants with CAI and uninjured control individuals.

DISCUSSION

Chronic ankle instability has been considered a neurophysiological disorder with balance deficits; however, few researchers have explored the brain activation of CAI during balance tasks using fNIRS technology. Authors[3] have proposed that copers rely on sensorimotor coping mechanisms during balance tasks after initial ankle sprains, which are absent in those with CAI. According to our results, the copers had increased activation in S1 and STG compared with the CAI group and the uninjured control group, suggesting that copers may use an altered cortical-activation strategy to maintain balance.

During the single-legged stance, S1 and STG were activated significantly. During balance tasks, the CNS requires afferent information from the somatosensory, visual, and vestibular systems, with healthy individuals especially relying on somatosensory information.[19] The S1 is an important brain region responsible for proprioception, and it perceives sensations on the contralateral side.[20] After stimulation, the peripheral somatosensory receptors relay through the dorsal spinal cord and terminate in the S1, where the stimuli are perceived.[21] Beyond S1, STG has also been associated with impaired proprioception,[22] and its activation is crucial for sensory integration, especially when balance is disturbed.[23]

The CAI group's activation of S1 and STG during the single-legged stance was less than that of the coper group, which may be the underlying cause of the former's dysfunction. In our study, the CAI group demonstrated diminished S1 and STG activation during balance tasks and less functional ability than the coper group based on the decreased scores in all 5 PROs as well as the YBT (lower composite reach distance and lower limb symmetry index and reach distance in the anterior direction). Other investigators of CAI also advocated that poor sensory integration may be central to the etiology of this condition. Participants with CAI used less somatosensory information during single-limb stance than uninjured control individuals,[24] and their somatosensory cortices were not able to distinguish higher levels of load on the ankle ligaments.[7] Another study of a musculoskeletal disorder also supports our findings. Valeriani et al[25] noted a cortical change in somatosensation that correlated with errors in joint position sense among participants with anterior cruciate ligament deficiency. Moreover, supportive results have been found in other populations, such as patients with cerebral palsy: patients with stronger somatosensory cortical activity displayed greater function and mobility.[24] Kurz et al[26] believed that motor control problems were related to poor sensorimotor integration. When combined with previous research, our results further contribute to the theory of poor sensory integration among individuals with CAI.

Copers' activation of S1 and STG during the single-legged stance was higher than that of the uninjured controls, which may be one of the important reasons for their better recovery. The lateral ankle sprain damaged the somatosensory receptors located in the ankle ligaments of both patients with CAI and copers, diminishing sensorimotor control.[5] Compared with the uninjured control group, our copers regained the same functional performance and displayed greater activation in S1 and STG during the balance task. Previous researchers determined that increased cortical excitability of S1 may promote somatosensory function in patients with strokes[27] and that less activity in STG was associated with greater proprioceptive error,[28]

Table 3. Oxyhemoglobin in the Subcentral Gyrus, Primary Somatosensory Cortex (S1), and Superior Temporal Gyrus (STG)

Oxyhemoglobin, μmol/L (channel)	Group, Mean ± SD			*P* Value
	Chronic Ankle Instability	Coper	Uninjured Control	
Subcentral gyrus (19)	1.04 ± 0.77	1.61 ± 1.57	0.89 ± 0.74	.106
S1 (21)	0.79 ± 0.72[a]	1.48 ± 1.12	0.79 ± 0.60[a]	.017
STG (38)	1.21 ± 0.96	1.73 ± 1.35	0.95 ± 0.63	.059
STG (39)	0.99 ± 0.87[a]	1.68 ± 1.13	0.91 ± 0.49[a]	.015

[a] Different from the coper group ($P < .05$).

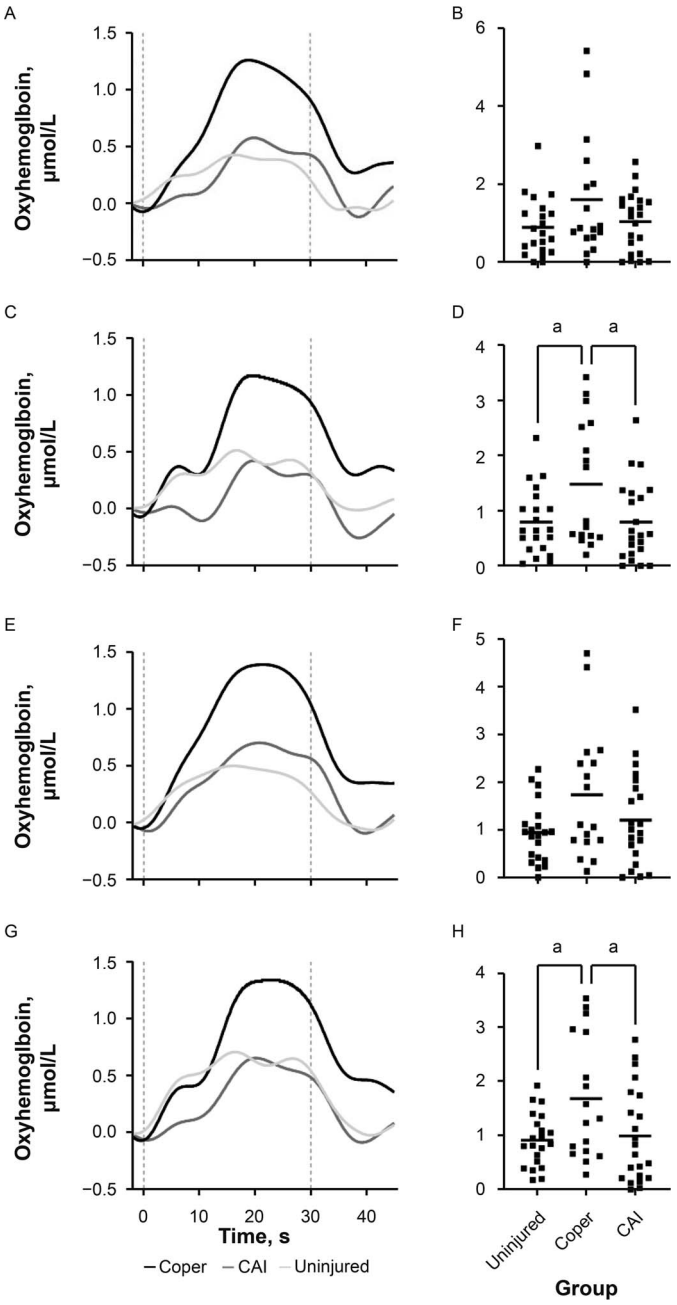

Figure 3. Time-course data and scatter-box plot of cortical activity. A and B, Subcentral gyrus (channel 19). C and D, Primary somatosensory gyrus (channel 21). E and F, Superior temporal gyrus (channel 38). G and H, Superior temporal gyrus (channel 39). The gray vertical lines represent the task onset (0 seconds) and end (30 seconds). Abbreviation: CAI, chronic ankle instability.
[a] Between-groups difference ($P < .05$).

suggesting that increased activation of S1 and STG may be beneficial for the proprioception of copers.

No difference in cortical activation during balance tasks was evident between the CAI and uninjured control groups. Earlier investigators also observed no difference in the somatosensory cortex between participants with CAI participants and uninjured control individuals during single-legged stance[12] and joint-loading tasks.[7] The possible reason is that cortical activation may differ between these groups, but the difference is too small to be detected by our current technology. Historically, uninjured control participants have been considered a comparison group for patients with CAI in CNS studies. However, uninjured control participants have never been exposed to ankle sprains, making it difficult to determine why those with CAI failed to recover after the initial ankle sprain.[3] Combined with our results, considering copers as a more appropriate comparison group may produce stronger and more relevant findings in CNS studies.

We propose that the coping mechanism absent in those with CAI is a successful reorganization of the sensorimotor system after the initial ankle sprain.[3] Based on our work, despite disrupted sensory feedback caused by the initial ankle sprain,[5] copers had better dynamic balance and enhanced S1 and STG activation. This altered cortical activation may be an adaptive change that promotes negotiating of dynamic tasks. When combined with the previous results,[5,7,24,25] this protective CNS adaptation may allow copers to better execute movements during balance tasks[8] and prevent repeated ankle sprains.[7]

Our findings demonstrate that increased activation of S1 and STG may be the mechanism of copers' balance control and a potential treatment target for the CAI population. Given that the CNS of copers appears to adapt more effectively after the initial ankle sprain, rehabilitation goals should not only be regaining appropriate action (increasing strength, balance, and power) but also enhancing the patient's proprioception.[29] For example, early mobilization instead of prolonged immobilization after ankle sprains and starting balance training as soon as weight bearing can be tolerated are beneficial to restoring perception.[30] We should also progressively increase the difficulty of balance exercises[29] (from simple to more complex tasks and from predictable to unpredictable environments) to promote the adaptation of the sensorimotor system to more complex tasks. Future researchers should incorporate longitudinal designs to determine whether existing treatments (eg, more complex balance-training protocols or transcranial electrical, magnetic, or direct current stimulation) can enhance cortical activation in CAI and develop rehabilitation strategies to improve S1 and STG stimulation. In addition, it is possible that copers have increased activation of S1 and STG before the initial ankle sprain, which may be the protective factor against recurrent ankle sprains. However, based on the results of our cross-sectional study, we were unable to tell whether the increased activation of S1 and STG occurred before or after the copers' initial ankle sprain. Prospective studies are needed to further explore this topic in the future. Another limitation of our study was that we did not consider the potential bias of leg dominance on cortical activation. Nonetheless, we found no differences in leg dominance among groups. Lastly, we assessed the limb symmetry index in the YBT of all participants, including individuals with bilateral instability, which may have caused an overestimation of the dynamic balance performance of those with CAI.

In conclusion, we observed that the coper group had greater cortical activation of S1 and STG during the balance task than the CAI and uninjured groups. Increased activation of S1 and STG may be the compensatory mechanism at the CNS level for the copers to maintain postural control after the initial severe ankle sprain. Activation of S1 and STG during the balance task may be a potential treatment target for CAI.

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