Lower-Limb Motor Assessment With Corticomuscular Coherence of Multiple Muscles During Ankle Dorsiflexion After Stroke

Rui Xu, Haichao Zhang, Xianle Shi, Jun Liang, Chunxiao Wan, and Dong Ming, *Senior Member, IEEE*

***Abstract*— Motor impairment after stroke is generally caused by damage to the neural networks that control movement. Corticomuscular coherence (CMC) is a valid method to analyze the functional connectivity of the corticospinal pathway between the cerebral cortex and muscles. However, current studies on CMC in stroke patients only focused on the upper limbs. The functional connectivity between the brain and lower limbs in stroke patients has not been well studied. Therefore, twelve stroke patients and fifteen healthy controls were recruited and their electroencephalogram (EEG) and electromyogram (EMG) of Tibialis Anterior (TA), Lateral Gastrocnemius (LG) and Medial Gastrocnemius (MG) during unilateral static ankle dorsiflexion were recorded. We found the mean beta and gamma CMC values of Cz electrode of stroke patients were significantly lower than those of healthy controls ($p < 0.05$). The brain topography showed significant coherence in the center of the cerebral cortex in healthy controls, while there was no significant coherence in stroke patients. For clinical assessment, there was a significant positive correlation between CMC and lower limb Fugl-Meyer Assessment (FMA) for Cz-TA in beta band ($r = 0.6296$, $p = 0.0282$), Cz-LG in beta band ($r = 0.6816$, $p = 0.0147$), and Cz-MG in gamma band ($r = 0.6194$, $p = 0.0317$). A multiple linear regression model was established between CMC and lower limb FMA ($R^2 = 0.6600$, $p = 0.0280$). Therefore, CMC between the cerebral cortex and lower limb muscles may be used as a new rehabilitation assessment biomarker in stroke.**

***Index Terms*— Corticomuscular coherence, static ankle dorsiflexion, stroke, motor assessment.**

I. Introduction

STROKE is a neurological disease characterized by blockage of blood vessels which seriously threatens human life and health [1]. Hemiplegia is the main manifestation of motor impairment after stroke [2]. Nearly 80% of the stroke survivors are affected by lower-limb motor dysfunction. Foot drop is their main clinical symptom, which is detrimental to patients' motor function [3]. In order to get the patients back to normal life as soon as possible, it is very important to fully evaluate the patients' motor function to plan an ideal physical therapy [4], [5]. Human electrophysiological parameters, such as electroencephalography (EEG) and electromyography (EMG), indicate a wealth of physiological and pathological information [6], [7]. Therefore, these indexes are usually used to monitor neural activity in the brain and predict motor rehabilitation in stroke patients [8], [9]. The combination of EEG and EMG can also reflect the information exchange between the brain and muscles, which indicates the level of brain control over muscles.

There are different levels of information exchange between brain and muscles during the movements [10], [11], [12]. Functional Corticomuscular Coupling (FCMC) [13], [14], which indicates that the control command is sent out from the cerebral cortex and transmitted to the corresponding body muscles and the muscles respond and transmit the feedback information to the brain. Corticomuscular coherence (CMC) is a method to analyze the functional connection of corticospinal pathway between central nervous system and motor muscle tissue [15], [16]. In 1995, Conway found synchronous cortical activity was coupled with motor output in the maintained voluntary contraction of muscle. He provided the evidence for the involvement of cortical neurons in the generation of motor-unit synchronization [17]. Motor dysfunction in stroke patients arises precisely because of problems in the brain's control of muscle pathways [18]. Therefore, exploring the

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This work involved human subjects in its research. Approval of all ethical and experimental procedures and protocols were granted by the Medical Ethics Committee of Tianjin Medical University General Hospital under Approval No. IRB2019-266-01, and performed in line with the Declaration of Helsinki.

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role of neural pathways under motor tasks will help to deeply study the mechanism of motor dysfunction and provide a new perspective for the evaluation of neuromotor function.

Generally speaking, during tonic contraction, stroke patients tended to have lower levels of CMC values than the general, and the CMC value of the affected hemisphere is significantly lower than that of the healthy hemisphere in stroke, which suggests EEG-EMG coherence provides an adequate index of the neural connections [19]. A similar study was done by Fang et al. They collected EEG signals and EMG signals from the anterior deltoid and brachialis muscles of upper limbs during reaching movements. And the CMC value was significantly lower in stroke patients than that in healthy controls in beta and lower gamma bands [13], as confirmed by Gao [20], Nielsen [21] et al. In addition, Krauth et al. found as stroke patients recovered, the CMC value increased during extending wrist and showed a significant correlation with motor function scale [22]. Von Carlowitz-Ghori et al. recorded EEG and EMG of the Abductor Pollicis Brevis (APB) muscle during thumb pressing, they found the CMC value was significantly increased after four weeks of rehabilitation intervention [23]. Belardinelli also confirmed that improved motor performance coincided with higher CMC values of the affected finger extensors [24]. The above findings may provide evidences that CMC of upper limbs can indicate the status of motor function rehabilitation in stroke patients.

Given these perspectives, it is of interest to study the link between CMC, the functional state of lower limb and how these are influenced by the task. Lower-limb CMC describes the information interaction between the cortex and the lower limb, which is more closely related to lower limb function. Although the motor cortex corresponding to lower limb is located in the inner side of the central sulcus, previous studies have found that it still can be recorded by scalp EEG. Spedden et al. found strong, positive associations between age and beta band coherence for Cz-TA during tonic ankle muscle contraction [25]. Úbeda et al. showed a significant coupling between EEG signals and motor unit spike trains at the target frequency during frequency-modulated isometric ankle dorsiflexions [26]. Ushiyama et al. analyzed CMC magnitude was associated with the amount of force fluctuation during tonic isometric voluntary ankle dorsiflexion [27]. However, these studies only revealed the general characteristics of lower limb CMC. The lower-limb CMC in the pathological state of stroke research is still under-investigated.

The above studies have proved that upper-limb CMC can be used as a biomarker to track the recovery process of stroke patients. However, the functional connection between the brain and lower-limb muscles in stroke patients is not clear. As demonstrated in a previous study, the lower-limb motor function of stroke was related to the function of the ankle joint which can not only adjust the body's walking posture but also maintain body stability [28]. Therefore, this study designed an experimental paradigm of ankle dorsiflexion. We aimed at observing the stroke patients' control of lower-limb movement during ankle dorsiflexion by EEG and EMG that have been previously shown to be synergistic, focusing on the control of lower-limb movement by the brain of stroke patients, so as to provide a new way for the rehabilitation evaluation of stroke patients.

TABLE I
DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF STROKE PATIENTS

Patient	Age	Sex	Lesion side	Days after stroke	FMA	NIHSS	BBS	Type
1	58	Male	Left	637	23	1	42	Hemorrhagic
2	57	Female	Right	167	20	1	52	Ischemic
3	64	Male	Right	177	19	2	41	Ischemic
4	46	Male	Right	108	19	8	6	Ischemic
5	51	Male	Left	84	24	2	51	Hemorrhagic
6	74	Male	Right	69	22	5	33	Ischemic
7	49	Female	Left	85	19	3	40	Ischemic
8	60	Female	Left	24	26	4	55	Ischemic
9	64	Female	Right	22	25	2	8	Ischemic
10	33	Male	Left	229	19	1	46	Hemorrhagic
11	32	Male	Left	185	25	0	55	Hemorrhagic
12	67	Male	Right	82	20	4	40	Ischemic

FMA = Fugl-Meyer Assessment, NIHSS = National Institute of Health Stroke Scale, BBS = Berg Balance Scale.

II. MATERIALS AND METHODS

A. Subjects

Stroke patients were included based on the following inclusion criteria: 1) first unilateral stroke; 2) unilateral limb hemiparesis due to stroke; 3) Mini-mental State Examination (MMSE) score > 24 (total score 30); 4) able to perform simple ankle dorsiflexion of the affected side. Exclusion criteria were: 1) recurrence after stroke; 2) having a history of neurological disease; 3) having cognitive impairment resulting in the inability to complete the experimental task.

Based on the above inclusion criteria and exclusion criteria, twelve stroke patients (4 females, mean age $\pm$ standard deviation: 55.58 ± 11.81 years old) were recruited at the Department of Rehabilitation, Tianjin Medical University General Hospital. The study also recruited 15 healthy controls with similar ages (8 females, mean age $\pm$ standard deviation: 49.20 ± 10.26 years old). There was no significant difference in age between the two groups ($p = 0.1456$). In addition, the motor function of the patient was evaluated by a professional physician according to the commonly used clinical scoring scales before the experiment. The National Institute of Health Stroke Scale (NIHSS), lower extremity Fugl-Meyer Assessment (FMA) and Berg Balance Scale (BBS) were used to assess the motor function of stroke patients. The demographics and clinical characteristics of stroke patients were shown in Table I.

All participants explicitly agreed to participate in the experiment, read and signed the informed consent for the clinical study. The Medical Ethics Committee of Tianjin Medical University General Hospital approved the clinical protocol and the informed consent for the study.

B. Experimental Paradigm

Before the formal collection of experimental data, the subjects were informed clearly of the precautions in the experiment. Firstly, the subject chose a comfortable posture. During the experiment, the subject was concentrated on the screen and tried to avoid blinking, swallowing and other

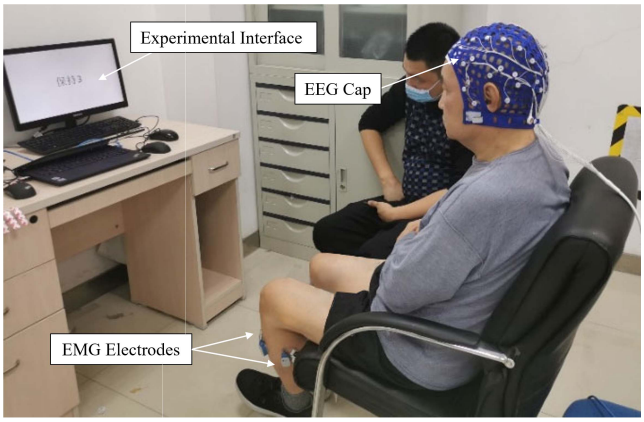

Fig. 1. Experimental scenario.

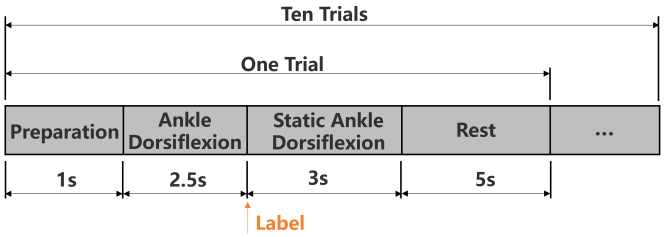

Fig. 2. Experimental procedure.

subtle movements except ankle dorsiflexion. In case of any discomfort during the experiment, the subject was allowed to immediately suspend the experiment. The experimental scenario was shown in Figure 1 (the person sitting against the wall was the patient's carer).

As CMC mainly appears during isometric contraction [29]. EEG and EMG signals were recorded at the same time during static ankle dorsiflexion. The experimental interface was designed by E-Prime 3.0 (Psychology software tools, Pittsburgh, PA, USA) which can communicate with EEG and EMG acquisition devices in real time. The experimental procedure was shown in Figure 2. Each trial consisted of four parts within 11.5 seconds: Preparation, Ankle Dorsiflexion, Static Ankle Dorsiflexion and Rest. "Preparation" was used to give subjects a prompt to keep their upper body and head stationary and avoid speaking, swallowing and excessive blinking during the next dorsiflexion part. When "Ankle Dorsiflexion" appeared on the screen, the patient had to lift his affected toes. As the reaction time and execution time varied between patients, the time was set to 2.5 seconds to give the patient sufficient time to lift his toes. When "Static Ankle Dorsiflexion" appeared on the screen, the patient was required to maintain static ankle dorsiflexion for 3 seconds. When "Rest" appeared on the screen, the patient needed to put down his toes, relaxed for 5 seconds and moved on to the next trial. There were 10 consecutive trials in each block and each subject had to complete a total of 5 blocks. In order to prevent muscle fatigue and ensure the accuracy of experimental data, blocks were separated by 10-minute rest.

C. Data Acquisition

1) EEG: In this experiment, the scalp EEG signals were acquired using an EEG recording system (Grael, Compumedics Ltd., Australia). The electrode placement adopted the international standard 10-20 system, as shown in Figure 3. The GND was the grounding electrode and the reference electrode was located between FCz and Cz. When collecting EEG signals, the sampling rate was set to 1024 Hz and the filtering range was 0.5-60 Hz. A notch filter was used to filter out 50 Hz power-line interference and conductive paste was injected into the electrodes to keep the impedance below 10KΩ. Finally, a total of 24 channels of EEG signals were acquired, namely F7, F8, F3, Fz, F4, FC3, FCz, FC4, T7, C3, Cz, C4, T8, CP3, CPz, CP4, P7, P3, Pz, P4, P8, O1, Oz and O2.

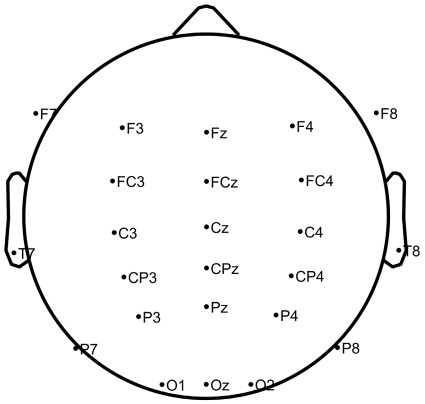

Fig. 3. EEG cap electrode distribution.

2) EMG: The EMG was collected from the Tibialis Anterior (TA), Lateral Gastrocnemius (LG) and Medial Gastrocnemius (MG) muscles with a wireless surface EMG acquisition device (Noraxon USA Inc., Scottsdale, AZ, USA). The sampling frequency was 2000Hz. Before the procedure, the skin was cleaned by 75% alcohol to reduce the surface impedance. Leaving the alcohol evaporated, we pasted the disposable EMG electrode sheet at the target muscles, including TA, LG and MG.

D. Data Processing and Analysis

1) Preprocessing: Data were pre-processed and analyzed using MATLAB 2021a (MathWorks, Inc., Natick, MA, United States) and the open-source toolbox EEGLAB (version 2021.1). Firstly, band-pass filtering was carried out at the frequency of 0.5-45 Hz to remove some high frequency noise interference in EEG signal. After that, the data were downsampled to 512 Hz [30], [31], [32], and a visual inspection was undertaken to reject any segments or channels with large motion artifacts. Finally, independent component analysis (ICA) was used to remove the electromyogram and electro-oculogram components from the signals after the above steps of processing. After pre-processing, the EEG data were processed with spatial filtering in MATLAB. This method can improve the signal-to-noise ratio of EEG signal [33]. The final potential S'_h was obtained through potential S_h of the electrode at the center minusing the weighted average potential S_i of four adjacent orthogonal electrodes. This study used the simplest method, assuming the four adjacent electrodes

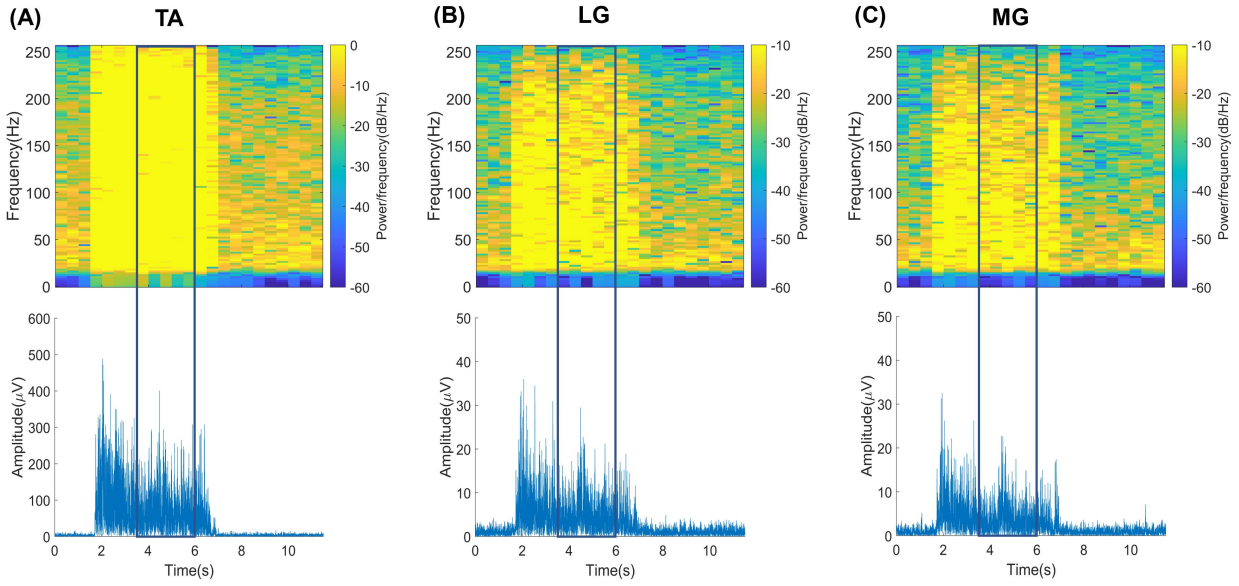

Fig. 4. EMG power and rectification of TA (A), LG (B) and MG (C) in an ankle dorsiflexion trial after pretreatment. The blue boxes showed the EMG for coherence analysis. TA = Tibialis Anterior, LG = Lateral Gastrocnemius, MG = Medial Gastrocnemius.

contribute equally to the intermediate electrode potential, i.e. the weight of each adjacent electrode was 0.25. The specific formula was as follows:

$$S'_h = S_h(t) - \sum_{i=1}^{4} \frac{1}{4} \cdot S_i(t) \tag{1}$$

All raw EMG data were filtered using a fourth-order Butterworth bandpass filter within 20-250 Hz. The mean baseline of each channel was subtracted from EMG data. The power frequency interference was removed by a notch filter. Finally, the EMG was resampled to 512 Hz, in accordance with EEG. The EMG power and rectification of TA, LG and MG after preprocessing in an ankle dorsiflexion trial were shown in Figure 4. During the experiment, the EMG amplitude increased sharply under the hint of "Ankle Dorsiflexion" (t = 1s in Figure 4), then the muscles began to contract dynamically. When "Static Ankle Dorsiflexion" was prompted, the dynamic contraction changed to static contraction, which lasted for 3 seconds (t = 3.5s-6.5s in Figure 4). In order to ensure that the data used for coherence analysis were collected during static contraction, the data of the former 2.5 seconds during static dorsiflexion were intercepted. The blue boxes in Figure 4 indicated the EMG used for coherence analysis.

2) Corticomuscular Coherence: Coherence was obtained from the normalization of cross-spectrum. The calculation formula was as follows [34]:

$$Coh_{xy}(f) = \frac{\left|P_{xy}(f)\right|^2}{|P_{xx}(f)| \cdot \left|P_{yy}(f)\right|} \tag{2}$$

where $P_{xy}(f)$ was the cross-spectral density of the signal, obtained as $P_{xy}(f) = \sum_{m=0}^{N-1} R_{xy}(m) e^{-jfm}$, where $R_{xy}(m)$ was the intercorrelation sequence of x, y, and m was the m-th sampling point. P_{xy} was the Fourier coefficient of R_{xy}. $P_{xx}(f)$ and $P_{yy}(f)$ were the self-spectra densities of signal x and y at frequency f, respectively. In order to calculate the Fourier transform, the data were divided into non-overlapping segments of 500 ms with a Hanning window. The coherence value was always between 0 and 1, where 1 represented that the two signals were completely coherent in an ideal state, and 0 represented that the two signals were completely incoherent. EEG-EMG coherence referred to the linear coupling between the EEG generated by brain activities and the EMG generated by muscle contraction in motor tasks.

Before calculating CMC, all EEG and EMG data segments were spliced to a time sequence respectively. Then the coherence was calculated by the mscohere function in MATLAB. The window length was 256, the number of overlapping points was 0, the sampling rate was 512 Hz and therefore the frequency resolution was 2 Hz. Finally, the CMC in alpha (8-12 Hz, point 5-7), beta (14-30 Hz, point 8-16) and gamma (32-44 Hz, point 17-23) bands were analyzed. The mean CMC was obtained by calculating the mean of all frequency points within each band. We calculated the coherence of EMG and EEG in all channels, and then used the topoplot function in EEGLAB to plot the brain map.

3) Confidence Level: The confidence level (CL) of coherence was calculated as follows [35]:

$$CL(\alpha) = 1 - (1-\alpha)^{\frac{1}{N-1}} \tag{3}$$

where N represented the number of data segments, α was the significant level. When CMC was higher than the 95% of significant limit, it indicated that there existed a significant coherence between the cortical and muscular activities ($\alpha = 0.95$). In this study, the total number of data segments N was 250, so the confidence level was 0.012.

E. Statistical Analysis

Statistical analysis of CMC was performed in MATLAB and SPSS (Statistical Package for the Social Science,

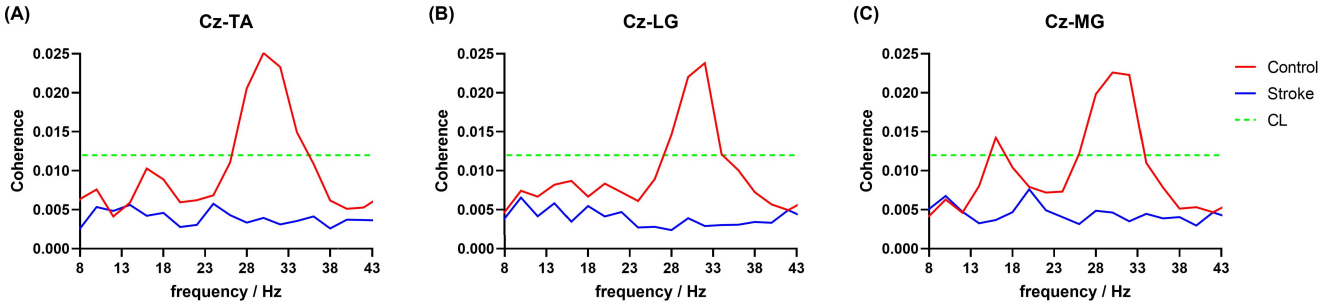

Fig. 5. The mean CMC in stroke patients and healthy controls. TA = Tibialis Anterior, LG = Lateral Gastrocnemius, MG = Medial Gastrocnemius.

version 26, IBM). Some studies pointed out that the middle part of the sensorimotor cortex (corresponding to Cz) was activated during lower limb movement [36], [37], which hinted the existence of coherence between the EEG at Cz electrode and the EMG of lower-limb muscles during a postural task. To assess differences in CMC between Cz-EEG and EMG of different muscles (TA, LG and MG), a three-way repeated measures ANOVA with the factors of "group" (healthy controls vs. stroke patients), "muscle" (TA vs. LG vs. MG) and "frequency" (alpha vs. beta vs. gamma) was used. The data were tested by Mauchly's Test of Sphericity. If sphericity was violated (i.e., Mauchly's test < 0.05), the Greenhouse-Geisse correction was used. After that, to investigate whether the mean CMC was significantly different between healthy controls and stroke patients, the Wilcoxon rank sum test was used. In order to analyze whether CMC can be used as an index to evaluate patients' lower limb motor function, the mean CMC of all trials of each patient was extracted to analyze the correlation between CMC and clinical scales. For the indexes with significant differences between groups, we used Pearson correlation analysis to test the correlation between CMC and NIHSS, FMA or BBS. In addition, the multiple linear regression analysis was used to explore the correlation between the above indexes and the clinical scales. The significance level of this study was set as $p < 0.05$.

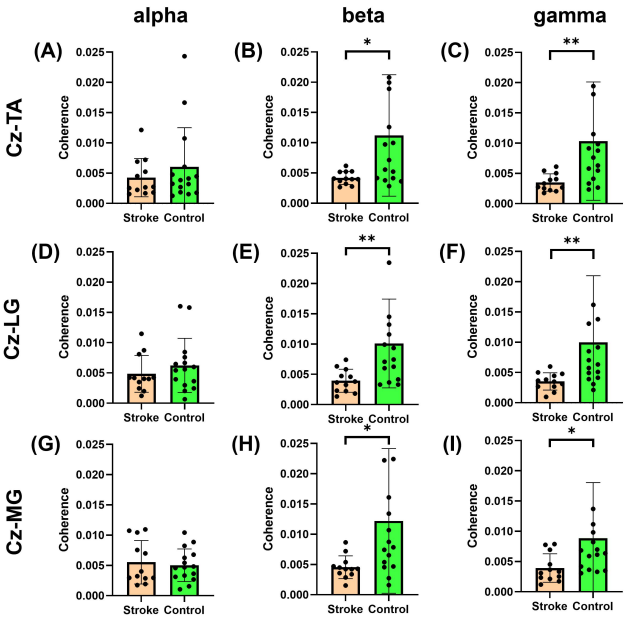

Fig. 6. Results of comparison of the mean CMC in alpha band (A, D, G), beta band (B, E, H), gamma band (C, F, I) for Cz-TA (A, B, C), Cz-LG (D, E, F), Cz-MG (G, H, I). TA = Tibialis Anterior, LG = Lateral Gastrocnemius, MG = Medial Gastrocnemius. * indicates $p < 0.05$, ** indicates $p < 0.01$.

III. Results

In order to better present the patients' characteristics of neural response, the brain topography of the patients with the left hemisphere affected was flipped left-right symmetrically. Thus, the left side of the brain topography referred to the unaffected hemisphere, and the right side referred to the affected hemisphere.

A. Difference of CMC Between Stroke Patients and Healthy Controls

1) CMC Spectra: In order to clearly show the coherence between stroke patients and healthy controls at different frequencies, the mean coherence between Cz and different muscles in 12 stroke patients and 15 healthy controls within 8 Hz-43 Hz was shown in Figure 5. It could be seen from the figures that the mean CMC in patients from alpha to gamma band was mostly lower than that in healthy controls, especially in beta and gamma bands. The beta CMC of healthy individuals exceeded the CL, indicating significant coherence.

2) CMC Comparison Between Stroke Patients and Healthy Controls: The coherence of stroke patients and healthy controls were compared in different frequency bands, i.e. alpha, beta and gamma bands. There was a significant main effect of the "group" factor ($F(1, 25) = 7.002$, $p = 0.0140$) and a significant interaction between "group" and "frequency" ($F(2, 24) = 3.935$, $p = 0.0330$). The CMC comparison between stroke patients and healthy controls was shown in Figure 6. The result of Wilcoxon rank sum test showed that mean CMC of Cz-TA, Cz-LG and Cz-MG in healthy controls was significantly higher than that in patients within beta and gamma bands (Cz-TA, beta: $p = 0.0120$; Cz-TA, gamma: $p = 0.0027$; Cz-LG, beta: $p = 0.0037$; Cz-LG, gamma: $p = 0.0037$; Cz-MG, beta: $p = 0.0104$; Cz-MG, gamma: $p = 0.0180$).

3) Brain Topography of CMC: The mean CMC was presented in the form of the brain topographic map. It was intuitively

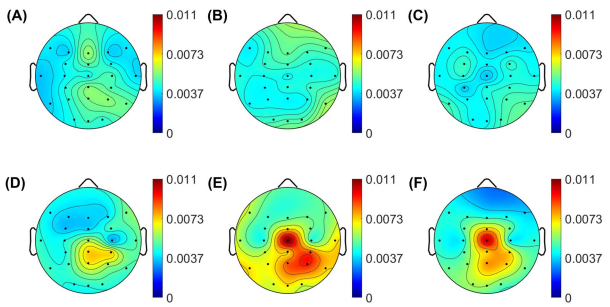

Fig. 7. Brain topography of EEG-TA CMC in alpha (A, D), beta (B, E) and gamma (C, F) bands in stroke patients (A, B, C) and healthy controls (D, E, F). TA = Tibialis Anterior.

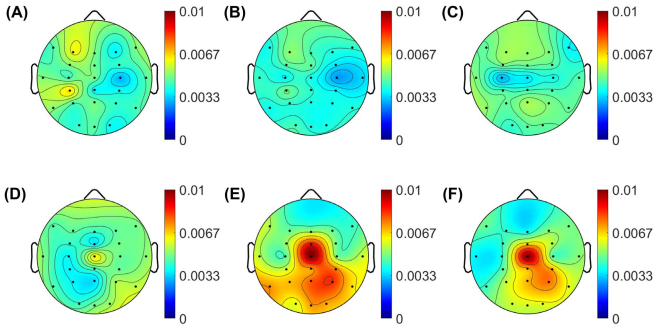

Fig. 8. Brain topography of EEG-LG CMC in alpha (A, D), beta (B, E) and gamma (C, F) bands in stroke patients (A, B, C) and healthy controls (D, E, F). LG = Lateral Gastrocnemius.

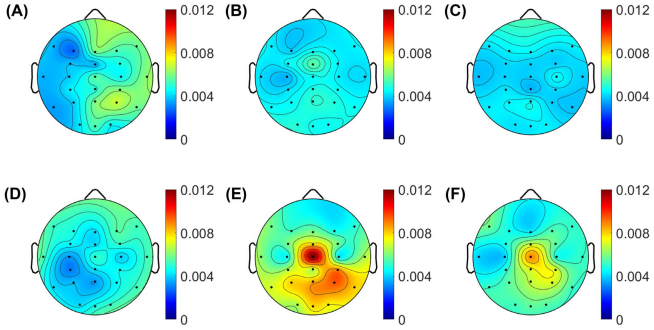

Fig. 9. Brain topography of EEG-MG CMC in alpha (A, D), beta (B, E) and gamma (C, F) bands in stroke patients (A, B, C) and healthy controls (D, E, F). MG = Medial Gastrocnemius.

observed whether there were differences in the activated cortical areas between stroke patients and healthy controls. The brain topographies of the mean CMC between EEG and TA, LG and MG EMG were shown in Figure 7, Figure 8 and Figure 9, respectively.

The CMC values of stroke patients were relatively lower than those of the healthy controls within the sensorimotor area, which was responsible for ankle movement. High CMC occurred mainly in the midline of the sensory motor area of the healthy controls within beta and gamma bands, and it also expanded to the right side of the brain corresponding to the movement of the left limbs.

B. Lower Limb Motor Assessment With CMC

The results of correlation analysis and multiple linear regression analysis were shown in Figure 10. The CMC of

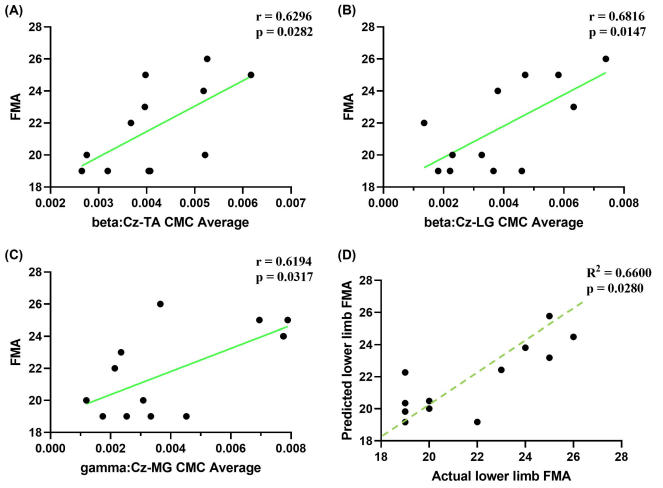

Fig. 10. Results of the correlation and regression analysis. (A) The correlation between Cz-TA CMC in beta band and lower limb FMA. (B) The correlation between Cz-LG CMC in beta band and lower limb FMA. (C) The correlation between Cz-MG CMC in gamma band and lower limb FMA. (D) The scatter chart of actual and predicted lower limb FMA obtained from regression. The green dotted line represented where actual and predicted lower limb FMA was equal. TA = Tibialis Anterior, LG = Lateral Gastrocnemius, MG = Medial Gastrocnemius. FMA = Fugl Meyer Assessment.

Cz-TA, Cz-LG and Cz-MG showed a significant positive correlation with lower limb FMA (beta CMC of Cz-TA: $p = 0.0282$, $r = 0.6296$; beta CMC of Cz-LG: $p = 0.0147$, $r = 0.6816$; and gamma CMC of Cz-MG: $p = 0.0317$, $r = 0.6194$). There was no significant correlation with NIHSS or BBS. It could be seen from the figure that the greater the CMC value, that is, the stronger the functional connection between the cerebral cortex and the corresponding muscles, and the higher the lower limb FMA score. The multiple linear regression model to describe the relationship between the CMC and lower limb FMA was established ($F(3, 8) = 5.177$, $p = 0.0280$, $R^2 = 0.6600$). The regression equation was as follows:

$$FMA = 14.937 + 717.113 \cdot Coh_{TA} + 597.816 \cdot Coh_{LG} + 371.780 \cdot Coh_{MG} \quad (4)$$

where Coh_{TA} represented the beta CMC of Cz-TA, Coh_{LG} represented the beta CMC of Cz-LG and Coh_{MG} represented the gamma CMC of Cz-MG.

IV. Discussion

The study aimed to investigate the coherence between motor cortex and lower limb muscles (TA, LG and MG) in stroke patients and to provide a new assessment method for stroke lower limb rehabilitation. Compared with the healthy controls, the CMC values between EEG of Cz and EMG of lower limb muscles (TA, LG and MG) in stroke patients were significantly lower in beta and gamma bands. CMC spectra and brain topography also reflected similar results. These results showed that stroke patients had abnormal corticomuscular coherence in the motor cortex. We also found that there was a significant correlation between the mean beta and gamma CMC and the

clinical scales. The biomarker could be used as an indicator for stroke assessment.

A. Changes of the CMC Mean After Stroke

Compared with the healthy controls, the mean values of CMC between EEG of Cz and EMG of lower limb muscles (TA, LG and MG) in stroke patients were significantly lower, as shown in Figure 6. The CMC spectra also reflected similar results, as shown in Figure 5. Some studies had reported that the values of CMC varied among individuals in healthy subjects [38], [39]. In our research, although the mean values were close, the distribution was quite different, which resulted in the difference of p values. Some studies pointed out that the decrease of CMC was related to age [40], [41]. However, there was no significant difference in age between stroke patients and healthy controls in this study. Some studies pointed out that compared with the healthy controls, the corticomuscular coherence of the anterior deltoid and brachial muscles in beta bands (20-30 Hz) and lower gamma bands (30-40 Hz) of stroke patients during exercise was significantly lower, indicating that the lower EEG and EMG coherence in the higher frequency band might reflect the potential mechanism of motion defects after stroke. It might be due to the injury of neural pathways caused by stroke, which resulted in the weakening of brain control over muscles [13]. Mima et al. also pointed out that the functional coupling between cortex and corresponding muscle activities in stroke patients was weaker than that in healthy subjects [19]. Our study also showed that the coherence in beta and lower gamma bands was very low after stroke. Therefore, the low coherence of stroke patients might be caused by structural damage to cerebral cortex. Partial interruption of corticospinal neurons might affect the excitability and discharge characteristics of local inhibitory and excitatory interneurons in the motor cortex [42]. The structural damaged brain could not control the abduction of the ankle joint well [43]. In addition, the lack of ankle dorsiflexion muscle strength in patients was another reason for the difference [44].

In addition, the results of EEG topography in this study showed that in beta and gamma frequency bands in the healthy controls, EEG of Cz had a high coherence with all muscles (TA, LG and MG), and the coherence of midline sensorimotor area was relatively high, as shown in Figure 7, Figure 8 and Figure 9. Omlor et al. observed that under the condition of isometric muscle contraction of static output force, the significant corticomuscular coherence was limited to the beta range, the most prominent coherence occurred in the gamma range in the dynamic condition [45]. Kristeva et al. found that increased corticospinal coherence in the beta range improved motor performance during steady-state motor output [46]. Beta oscillation mainly originated from the primary motor cortex and was related to the control and maintenance of steady-state forces [47], [48], [49].

Otherwise, some studies found that stroke patients showed more task-related brain activation in the affected and unaffected hemispheres [50]. For example, Belardinelli et al. found that CMC appeared on both sides of the brain in patients with severely damaged hand movement. We also found that the distribution of CMC in patients was scattered. This result may be caused by the defect of the patient's lower limb motor function, which may cause the patient to mobilize other parts of his body for compensation during static ankle dorsiflexion. However, Rossiter et al. included patients with various injuries and found no correlation between injury location and CMC [51]. Further investigation is required to deeply understand the relationship between functional recovery and CMC.

B. Relationship Between CMC and Clinical Scales

CMC was a marker of the corticospinal pathway based on the functional coupling between oscillatory signals from the brain and active muscles [17]. This study explored the correlation between the clinical scales and the corticomuscular coherence for different muscles in different frequency bands. As shown in Figure 10, the study found that the coherence was only significantly correlated with the lower limb FMA scale. The coherence increased with the increase of the lower limb FMA score, which meant that the patient with a higher CMC had better motor function. In addition, some studies have pointed out that joint movement requires not only the contraction of the agonistic muscles, but also the antagonistic muscles. Antagonistic muscles can generate torque around the joint, which increased the stiffness of the joint and improved the accuracy of movement [52], [53], [54]. Therefore, the agonistic (TA) and antagonistic (LG and MG) muscles contracted simultaneously during dorsiflexion in this experiment, showing similarities in shape. Dal Maso et al. measured the functional coupling between the motor cortex and muscle activity during knee movement, and the results showed that the motor cortex was directly involved in the regulation of both agonist and antagonist muscles [55], which was consistent with the result of our study. Our results also showed that the motor function of stroke patients can be assessed by the coupling between the brain and both agonistic and antagonistic muscles, which were activated in the movement. However, some studies found that there was no correlation between CMC and motor performance in patients with chronic stroke [51]. The reasons for this difference might be diverse, including but not limited to the differences in the rehabilitation status of patients, the time after stroke and the experimental paradigms used by different researchers [23]. We also used CMC of all muscles to predict lower limb FMA. The multiple linear regression model between CMC and lower limb FMA was significant indicating that CMC could be used to predict lower limb FMA. Besides, the R-Squared of multiple muscles ($R^2 = 0.6600$) was higher than single muscle ($R^2_{TA} = 0.3964$, $R^2_{LG} = 0.4645$, $R^2_{MG} = 0.3837$) for regression evaluation. Therefore, multiple muscles could assess the motor function of stroke better. The above results showed that CMC between the cerebral cortex and lower limb muscles could be used as indexes for stroke motor assessment.

C. Limitation

There were still some limitations in this study. Firstly, in terms of the number of subjects, this study belonged to

a small group study. In order to make the analysis results more convincing, more stroke patients should be recruited in future studies. Secondly, this experiment required stroke patients to have a certain level of residual motor function and be able to complete simple ankle dorsiflexion. Therefore, most of the recruited patients had mild and moderate damage. Correspondingly, the lower limb FMA scale score was more than 18, and there was a lack of experimental data on patients with severe damage. Thirdly, this study did not set certain requirements for the degree of ankle dorsiflexion. Different subjects may have different ankle dorsiflexion angles, which is also an important reason for the result variation.

V. Conclusion

To the best of our knowledge, this is the first study on the corticomuscular coherence of lower limbs after stroke. In this work, we analyzed the coherence between the motor cortex and lower limb muscles (TA, LG, MG) during static ankle dorsiflexion. The results demonstrated that the corticomuscular coherence at Cz of beta and gamma bands in stroke patients was significantly lower than that in healthy controls. The lower coherence might reflect the potential mechanism of functional impairment in stroke patients. In addition, there was a significant positive correlation between beta-band Cz-TA CMC ($p = 0.0282$, $r = 0.6296$), Cz-LG CMC ($p = 0.0147$, $r = 0.6816$), and gamma-band Cz-MG CMC ($p = 0.0317$, $r = 0.6194$) and lower limb FMA. We also established a multiple linear regression model to predict lower limb FMA with beta band Cz-TA, Cz-LG, and gamma band Cz-LG CMC ($p = 0.0280$, $R^2 = 0.6600$). In a word, these results demonstrated a significantly lower CMC of the lower limb for stroke patients, and the coherence between the cerebral cortex and lower limb muscles in stroke patients may be used as a new biomarker for rehabilitation evaluation.

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