

Correlation between EEG–EMG coherence during isometric contraction and its imaginary execution

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To assess the similarity between cortical activities observed during actual and imaginary motor tasks, we evaluated electroencephalography–electromyography (EEG–EMG) coherence during motor task execution (ME) and the same task-related EEG power increase (TRPI) during kinesthetic motor imagery (MI). EEGs recorded at the vertex and EMGs recorded at the right tibialis anterior muscle (TA) were analyzed in 13 healthy subjects. Subjects were requested to perform: (1) isometric TA contraction, (2) imagery of the same movement without overt motor behavior, and (3) rest without MI. The results show significant EEG–EMG coherence during ME, as well as TRPI during both ME and MI tasks within a similar 14–30 Hz band. The magnitude of EEG–EMG coherence and TRPI varied among the subjects. Intersubject analysis revealed a significant correlation between EEG–EMG coherence and TRPI. These results support the hypothesis that ME and MI tasks involve overlapping neural networks in the perirolandic cortical areas.

Key words: motor imagery, task-related EEG power, EEG–EMG coherence, isometric contraction, sensorimotor cortex

INTRODUCTION

It is broadly accepted that synchronized work of neurons results in the generation of oscillatory activities. The frequency of these activities may reflect both intrinsic membrane properties of single neurons and the organization and interconnectivity of neural networks (Lopes da Silva 1991). As revealed by Jasper and Penfield (1949), beta-range synchronization (18–30 Hz) is the most characteristic of the sensorimotor cortex during motor execution (ME). A similar beta-range (15–30 Hz) has been used since the early 1990s to investigate the coherence between neurons in the sensorimotor cortex and motor units in the effector muscle by electroencephalography (EEG) or magnetoencephalography (MEG), and electromyography (EMG) (Conway et al. 1995, Halliday et al. 1998, Mima and Hallett 1999). These researchers interpreted cortico-muscular coherence as evidence of the involvement of cortical neurons in motor unit synchronization.

Both actual ME and kinesthetic motor imagery (MI), a mental process by which an individual rehearses or simulates a movement, induce changes in cortical synchronization. Increased magnitude of beta-range synchronization in the EEG and MEG can be observed during MI by power spectral analysis (Schnitzler et al. 1997, Neuper et al. 2005, Pfurtscheller et al. 2005, Pfurtscheller and Solis-Escalante 2009). Although beta-range synchronization involving foot MI has been used successfully as a trigger for external devices (Pfurtscheller et al. 2003), large intersubject variability limits the practical application of this technology.

One of the possible reasons for this intersubject variability is the varied capability of neurons to work in synchrony in the sensorimotor cortex. Intersubject variability has been reported in previous studies of ME. Mima and coauthors (2000) reported that only four of nine subjects who participated in their experiments showed stable EEG–EMG coherence. Baker and Baker (2003) also reported a lack of coherence between EEG and EMG signals in the beta band of their subjects. GABA-mediated intracortical inhibition in the

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sensorimotor cortex may be the key mechanism supporting the beta-range synchronization during ME (Jackson et al. 2002, Baker and Baker 2003).

Several functional magnetic resonance imaging (fMRI) studies have pointed out a similarity between ME and MI in the neural process (Porro et al. 1996, Roth et al. 1996, Erslund et al. 1996). Therefore, we hypothesized that the magnitude of the beta-range synchronization during MI also shows a subject-dependent tendency for corticomuscular coherence during ME. The magnitude of synchronization in the two conditions may be determined by the capability of neuronal networks to work in synchrony.

To assess the intersubject variability, we investigated isometric contraction of the tibialis anterior (TA) muscle focusing on the relationship between EEG–EMG coherence during ME and the same task-related EEG power increase (TRPI) during MI.

METHODS

Subjects

Thirteen healthy young subjects (2 females, 11 males: 21–30 years of age) participated in this study. No participant had a history of neuromuscular disorder. The study protocol was approved by the local ethics committee of the Faculty of Science and Technology, Keio University, Kanagawa, Japan. Informed consent was obtained from all subjects. The experiments were conducted in accordance with the Declaration of Helsinki.

Subjects were seated in an armchair. Their right leg was semi-flexed at the hip (120°), the knee was flexed to 40°, and the ankle in 10° dorsal flexion. The foot was mounted to a plate during all measurements. The plate was connected to a torque meter to measure dorsal flexion force.

Tasks

Subjects were requested to perform the following: (1) isometric contraction of the right TA muscle (“ME task”); (2) imagery of same movement without any overt motor behavior (“MI task”); and (3) rest without any MI (“REST task”). Each task lasted 28 s. During the ME task, the dorsal flexion force was displayed on the computer screen and the subjects were instructed to keep the force constant at 30% of maximum voluntary contraction (MVC). During the MI task, the sub-

jects were asked to generate a kinesthetic image, but not a visual image, of the ME task. One session consisted of one of these three tasks (ME, MI, and REST) executed in a random order, thus, a total of 4 sessions were conducted. The sessions were separated by 20–60 s breaks to prevent fatigue.

Data acquisition

Ag/AgCl electrodes (diameter 9 mm) were placed close to the foot representation area (FCz, C1, Cz, C2, and CPz) according to the guidelines for the standard electrode position nomenclature (Sharbrough et al. 1991) to record surface EEG. All 5 channels were referenced to the right ear lobe. The data were first converted to a reference-free form by a Laplacian algorithm (Hjorth 1975) that used the set of four nearest neighbor electrodes [for electrode Cz, these were FCz (anterior), C1 (left), C2 (right) and CPz (posterior)] at offline processing. The EEG was calculated by taking the difference between the potentials at the Cz electrode and the mean of the four nearest neighbor electrodes. EEG electrode impedances were kept below 5 k Ω during the experiment. EMG was simultaneously recorded from the right TA muscle by surface bipolar electrodes placed 2 cm apart and centered over the right TA muscle belly. By using a biosignal amplifier (NeuropackMEB-2200, Nihon-Koden, Japan), EEG and EMG were amplified and filtered (EEG: 5–100 Hz; EMG: 2–500 Hz), digitized at 1000 Hz (12-bit AD converter, PCI-6071E, National Instruments Corp., US), and stored on a personal computer for offline analysis.

Data analysis

Analysis of the EEG power spectrum and EEG–EMG coherence was conducted following segmentation of the data stream into 112 segments of 1-s duration (28-s data from 4 sessions). Because of the segment length of 1 s, frequency resolution was 1 Hz. The power of a signal as a function of frequency was calculated as:

$$P_{xx}(f) = \frac{1}{112} \sum_{j=1}^{112} X_j(f) X_j^*(f) \quad (1)$$

where $X_j(f)$ refers to the Fourier transform of the j th segment for EEG at a given frequency f . * indicates complex conjugation.

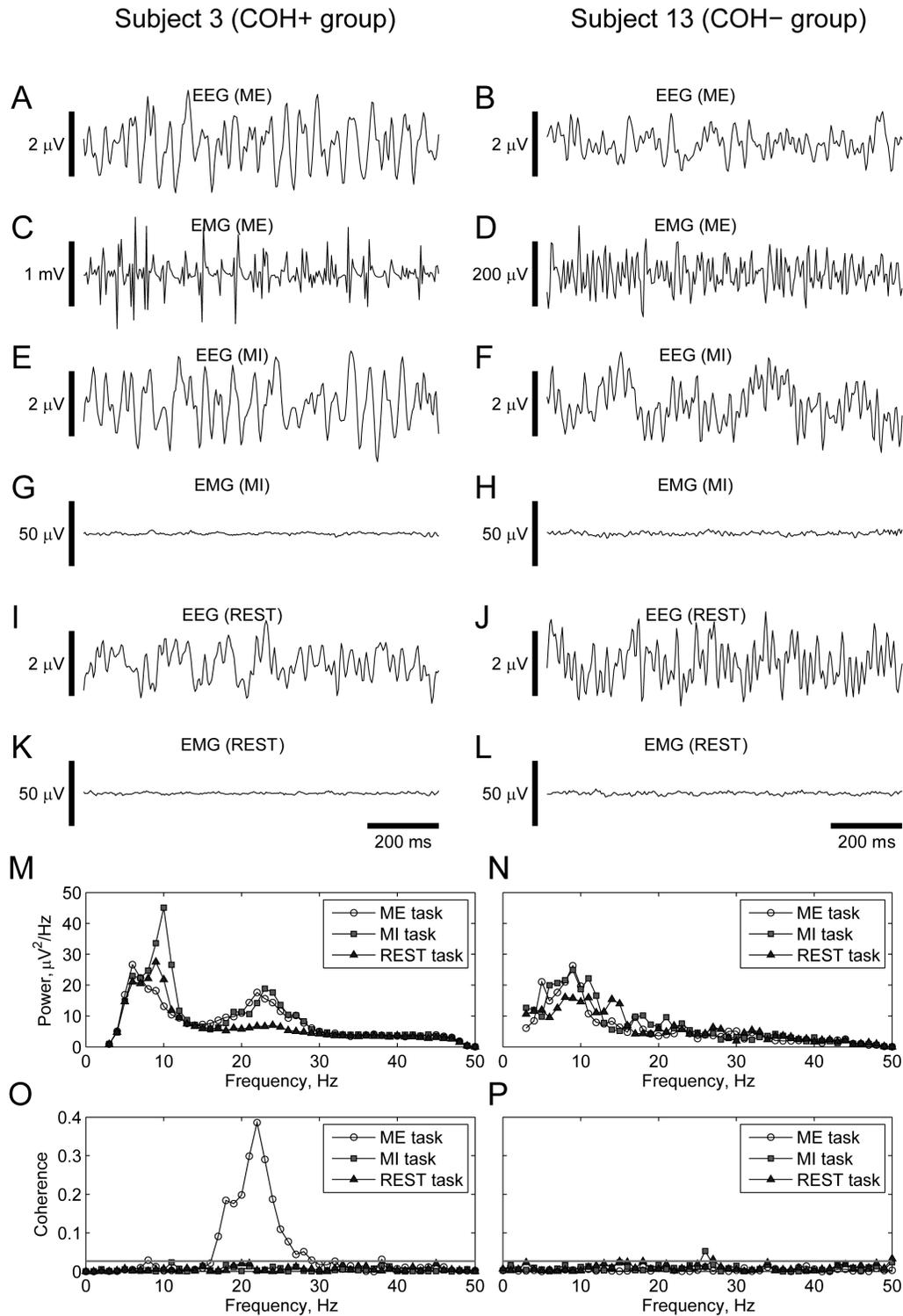


Fig. 1. Dataset obtained from two representative subjects, one with high and one with low EEG–EMG coherence. The panels in the left column are for a subject in the COH+ group, and those in the right column are for a subject in the COH- group. (A)–(L) shows 1-second EEG and EMG records. (M)–(N) shows power spectra of EEG during the ME, MI, and REST tasks. Subjects contracted the right tibialis anterior (TA) muscle during the ME task but only imagined contraction of the TA muscle during the MI task. (O)–(P) shows the coherence spectra between EEG and rectified TA muscle EMG. The horizontal line denotes the 99% confidence limit.

The coherence between EEG and EMG was calculated as:

$$C_{xy}(f) = \frac{\frac{1}{112} \sum_{j=1}^{112} X_j(f) Y_j^*(f)}{P_{xx}(f) \cdot P_{yy}(f)} \quad (2)$$

where $Y_j(f)$ refers to the Fourier transform of the j th segment for rectified EMG recorded from the TA muscle, and $P_{yy}(f)$ is its power calculated in the same manner as Equation 1. EMG was rectified before

coherence analysis but not integrated. Coherence was considered to be significant if the value exceeded the α % confidence limit, which can be calculated by the following equation (Rosenberg et al. 1989):

$$CL(\alpha) = 1 - \left(1 - \frac{\alpha}{100}\right)^{\frac{1}{112-1}} \quad (3)$$

with α of 99% and 99.9% corresponding in our experimental design to the confidence limits of 0.041 and 0.060, respectively.

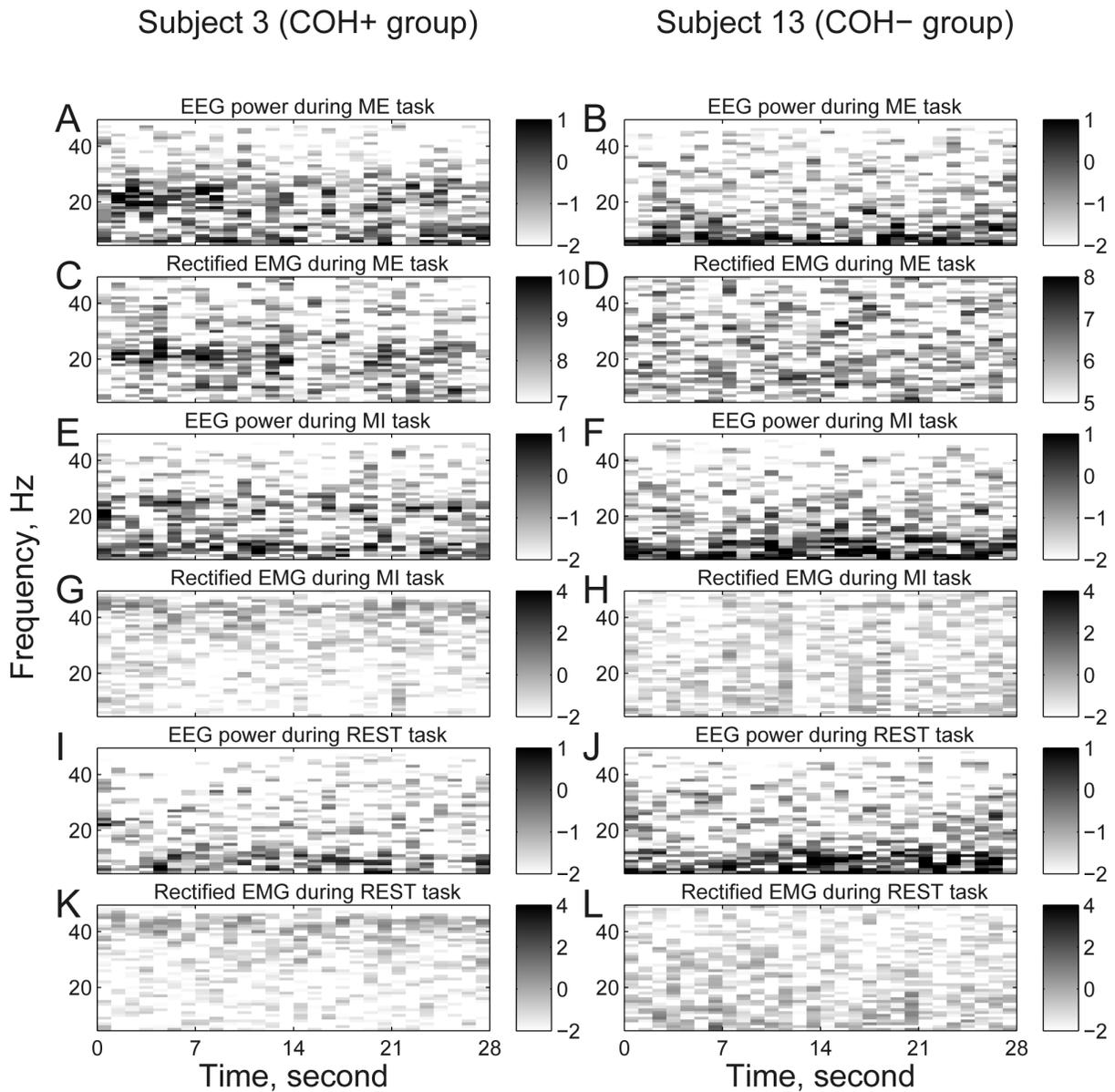


Fig. 2. The logarithm to base ten of the power of EEG and rectified EMG in successive 1-s intervals for the entire 28 segments of the ME, MI, and REST tasks. The panels in the left column are for Subject 3 in the COH+ group, and those in the right column are for Subject 13 in the COH- group during the ME [(A)–(D)], MI [(E)–(H)], and REST tasks [(I)–(L)].

The percentage changes of EEG power during ME and MI tasks were calculated relative to EEG power during REST as follows:

$$\%TRPI(f) = \left(\frac{Pxx_{MT}(f) - Pxx_{REST}(f)}{Pxx_{REST}(f)} \right) \times 100 \quad (4)$$

where $Pxx_{REST}(f)$ and $Pxx_{MT}(f)$ are the power spectrum of a given frequency f obtained during REST and motor tasks (ME or MI), respectively. The same analysis was used in a previous study of TRPI (Manganotti et al. 1998) to investigate steady-state changes associated with tasks. We did not use the analysis for event-related synchronization/desynchronization (ERS/ERD), which is generally used to analyze phasic changes associated with events (Pfurtscheller and Lopes da Silva 1999). Statistical differences between $Pxx_{REST}(f)$ and $Pxx_{MT}(f)$ were calculated using a Wilcoxon rank sum test.

The individual maximum values of EEG–EMG coherence and TRPI within 14–30 Hz were plotted for assessing the correlation between them. We limited our analysis to one frequency range because significant EEG–EMG coherence and TRPI were observed in this range both in our records and in previous studies (Neuper and Pfurtscheller 1996, Baker et al. 1997, Pfurtscheller et al. 2003).

All analyses were carried out using MATLAB software (The MathWorks, US) with custom-developed programs.

RESULTS

We observed significant power increases in the beta band (14–30 Hz) during both ME and MI, and considerable intersubject variability, as reported previously (Doppelmayr et al. 1998, Neubauer et al. 2004, Pfurtscheller et al. 2005). EEG measurements during ME correlated with EMG activity. Our novel finding is that the degrees of TRPI during ME and MI are strongly associated with the magnitude of EEG–EMG coherence in ME.

During ME task, 8 of 13 subjects showed significant coherence between EEG and rectified EMG in the frequency range of 14–30 Hz. We categorize these subjects as COH+ group ($n=8$), and the remaining as COH- group ($n=5$). A dataset obtained from a representative subject in the COH+ group (Subject 3), i.e., raw EEG and EMG signals, and the power spectrum of EEG and EEG–EMG coherence are shown in the left column of

Fig. 1. For comparison, the same dataset obtained from a representative subject (Subject 13) in the COH- group is shown in the right column of Fig. 1. One-second EEG and EMG signals in an arbitrarily chosen segment are displayed in the top eight traces. In EEGs during ME (Fig. 1A,B) and MI (Fig. 1E,F), periodic EEGs were observed with a frequency of approximately 20 Hz in the COH+ group (Fig. 1A,E), whereas no clear irregularity was seen in the COH- group (Fig. 1B,F). EMG in the COH+ group tended to show recurrent bursts separated by silent periods (Fig. 1C). Significant EEG–EMG coherence in the beta band (Fig. 1O) suggested that the frequency of this EMG waning was correlated with EEG. On the other hand, EMG in the COH- group showed a stable pattern with mostly constant amplitude (Fig. 1D), and EEG–EMG coherence showed no obvious peaks (Fig. 1P). In the COH- group, EEG power during ME and MI tasks increased to the 14–30 Hz frequency range compared to that during rest (REST: Fig. 1M), whereas no obvious increase was observed in the COH- group (Fig. 1N). The frequency band of this TRPI was equivalent to that of significant peak in EEG–EMG coherence.

To obtain a better sense of the pattern of records over the entire duration of each task, Fig. 2 shows the power spectra using short-term Fourier transform of EEG and rectified EMG in successive 1-s intervals for the entire 28 segments of the three tasks performed by the same subject as those in Fig. 1. Figure 2 also shows that the pattern of records at Fig. 1 is uniformly apparent over the entire duration of the tasks.

To show whether there were differences at various percentage of MVC between the COH+ and COH- groups, two typical subjects (Subject 3 and Subject 13) participated in an additional experiment on a separate day. The subjects were instructed to keep the force constant at 5%, 10%, 20%, and 30% of MVC. Other conditions were same as that of the main experiment.

One-second EMG signals in an arbitrarily-chosen segment are displayed (Fig. 3). At 30% of MVC, Subject 3 (COH+) again showed the periodic EMG signal with a frequency of approximately 14–30 Hz. The amplitude of EMG gradually decreases as the contraction level decreases (30% > 20% > 10% > 5% of MVC); this was identical to Subject 13, classified as belonging to COH-. Identical single motor unit action potential was observed at 5% of MVC and was only 100 μ V from peak to peak. In contrast to Subject 3, Subject 13 showed no periodic EMG signal at 30% of

MVC. Identical single motor unit action potential was also observed at 5% of MVC same as in Subject 3.

Details of the association between EEG–EMG coherence and TRPI during ME and MI in both groups are summarized in Table I. In the COH+ group, peaks of EEG–EMG coherence were seen at 23 ± 3 Hz in ME, and the peak value varied among the subjects from 0.474 to <0.060 . The TRPI magnitude varied among the subjects from 22% to 184%, with a dominant frequency of 24 ± 4 Hz during ME. During MI, TRPI varied among the subjects from 16% to 208% with a dominant frequency of 24 ± 3 Hz. Although some subjects did not show any significant coherence or TRPI at most frequencies, data from such subjects were analyzed within the same beta frequency range where coherence and TRPI were highest in the COH+ group (14–30 Hz).

We observed significant TRPI in the beta band during both ME and MI tasks. The intra-subject comparison of TRPI during ME and MI is shown in Fig. 4 (the correlation was significant: $r=0.636$, $P<0.05$). Subjects had similar TRPI levels during both tasks. Figure 5

represents the correlation between peak values of EEG–EMG coherence and TRPI for all subjects. Strong correlations were observed both for ME ($r=0.934$, $P<0.001$) and for MI ($r=0.821$, $P<0.001$). The stronger the EEG–EMG coherence peaks a subject showed, the larger was the TRPI during ME and MI. Subjects in the COH- group did not show any obvious EEG–EMG coherence and had low TRPI values.

DISCUSSION

We found that beta-band TRPIs during both ME and MI correlated with EEG–EMG coherence during ME. This implies that activity of the sensorimotor cortex during MI involves neural assembly similar to that which is activated during actual ME.

Common features of ME and MI

The coherence peak value varied among the sub-

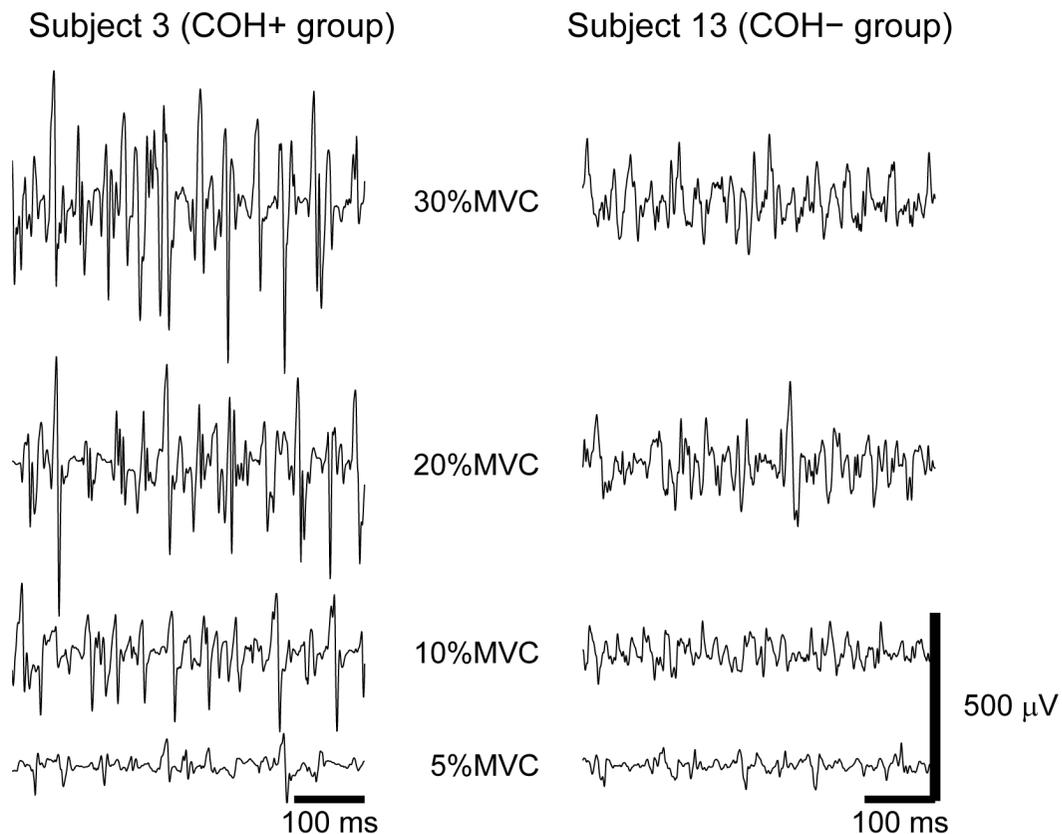


Fig. 3. Raw EMG signals (2–500 Hz) in Subject 3 (left column) and Subject 13 (right column) during 5%, 10%, 20%, and 30% of MVC. The data is showed in successive 1-s intervals.

jects from 0.01 to 0.47. Such a large intersubject variability was also reported in previous studies. Mima and colleagues (2000) reported that only four of their nine subjects showed stable EEG–EMG coherence. Baker and Baker (2003) also reported the existence of subjects who showed no EEG–EMG coherence in the

Table I

Summary of the maximum TRPI value and EEG-EMG coherence value in 14–30 Hz frequency range

Group	Subject number	EEG-EMG coherence		%TRP _{ow} (14–30 Hz)						
				ME task		MI task				
		Max.	Hz	Max.	Hz	Max.	Hz			
COH+	1	0.474	**	20	257	**	20	106	**	25
	2	0.456	**	15	132	**	19	302	**	16
	3	0.386	**	22	162	**	22	185	**	23
	4	0.192	**	28	104		27	123	*	22
	5	0.182	**	25	65	**	27	49	*	27
	6	0.165	**	25	50		26	101	**	25
	7	0.150	**	26	54	*	26	26	**	27
	8	0.060	*	23	–1		22	8		27
	Mean	0.258		23	103		24	112		24
	SD	0.157		4	81		3	96		4
COH–	9	0.039		28	13		28	47		28
	10	0.029		15	2		16	37		24
	11	0.023		18	12	*	23	15		24
	12	0.016		14	21		28	15		22
	13	0.012		26	12	*	15	35		27
	Mean	0.024		20	12		22	30		25
	SD	0.011		6	7		6	14		2

* $P < 0.01$; ** $P < 0.001$

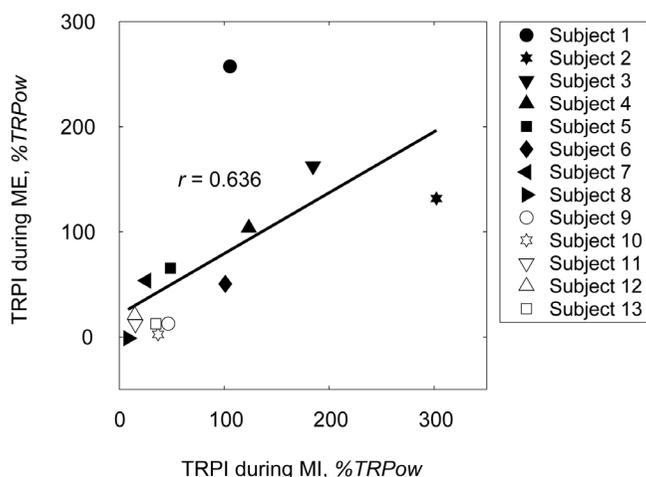


Fig. 4. The maximum TRPI value during the ME and MI tasks in 14–30 Hz frequency range. The solid line shows linear regression with a correlation coefficient (r) of 0.636 ($P < 0.05$).

beta band. This implies that the competence of neurons for synchronized interaction of distinct neural sites in the sensory-motor nervous system varies among individuals. Correlation of TRPI during MI in EEG–EMG coherence suggests that individual differences in the magnitude of TRPI during MI depend on the ability of neurons to work in synchrony.

Common features of ME and MI were also observed in fMRI studies. Primary motor cortex activity during MI was observed both in healthy subjects (Porro et al. 1996, Roth et al. 1996) and in a subject with a phantom limb (Erslund et al. 1996), with the weaker signal during MI rather than ME. The activity identified in the

primary motor cortex was similar to ME. These findings suggest involvement of the primary motor cortex in both ME and MI.

Not only motor output but also afferent input is important for enhancement of the degree of TRPI and corticomuscular coherence. A MI experiment employing tourniquet-induced ischemia showed that afferent input from the limbs is not required to dampen the beta rhythm in the motor cortex (Schnitzler et al. 1997). This result implies that sensory inputs influence TPRI magnitude. Another study revealed that coherence strength was also reduced during ischemia (Pohja and Salenius 2003). Although it is unclear whether the neural sites responsible for TRPI and corticomuscular coherence operate on the motor or sensory side, our results suggest a common neural mechanism for both ME and MI. Specifically, we believe that such a mechanism can synchronize the magnitude of EEG–EMG coherence during ME and the TRPI during MI.

Difference of EMG patterns between COH+ and COH- group

Subject 3 (COH+ group) showed the periodic EMG signal with a frequency of approximately 14–30 Hz. Such an EMG pattern with steep peaks resembles single motor unit action potentials, thus one may think that EMG was incorrectly recorded. However, the amplitude of EMG gradually decreases as the contraction level decreases, and identical single motor unit

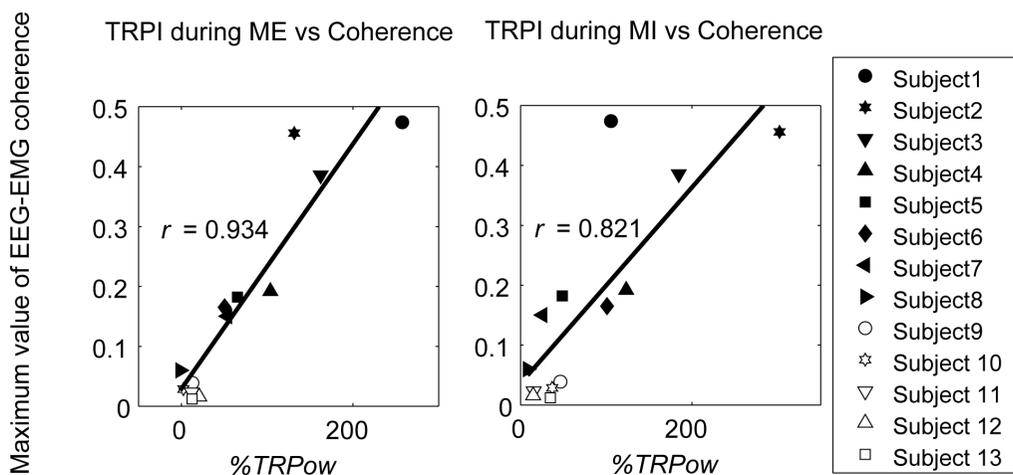


Fig. 5. The maximum TRPI value in the frequency range of 14–30 Hz versus the maximum EEG–EMG coherence value in the same frequency range. The left and right panels indicate intra-subject correlations between EEG–EMG coherence during the ME task and TRPI during the ME (left) and MI (right) tasks, respectively. The linear regressions (solid lines) were significant ($P < 0.001$ for both), and the correlation coefficients (r) were 0.934 for the ME task and 0.821 for the MI task.

action potential was finally observed at 5% MVC and was only 100 μV from peak to peak. Thus, spiky EMG observed during 30% MVC is a superimposed waveform of synchronized motor unit action potentials, not single motor unit action potentials. From this evidence, we believed that the recording was correctly done.

The recorded EMG activity seems different in amplitude between these two subjects. Subject 3 (COH+ group) showed much larger amplitude of EMG than Subject 13 (COH- group). We considered that the degree of synchrony among activated motor units results in such differences in amplitude in surface EMG. In 2000, Yao and coworkers suggested that motor unit synchronization increased the amplitude of EMG in their simulation study (Yao et al. 2000). They clearly showed how synchrony can minimize cancellation and generate differences in surface EMG. Our data agrees with their findings. In Subject 3, EMG showed larger amplitude of superimposed waveforms of synchronously activated motor units. The silent period in EMG also implied that activated motor units were strongly synchronized. In contrast, in Subject 13, EMG showed lower amplitude without a silent period, implying that activated motor units were out of phase.

The periodic EMG observed in the case of Subject 3 has also been reported in other papers in the field of corticomuscular coherence. For example, Salenius and others (1997) reported a subject who showed a periodic EMG signal and a high magnitude of MEG–EMG coherence. In addition, Gross and coauthors (2000) also reported similar EMG bursts with a frequency band centered at 20 Hz. These studies show that grouped discharges of EMG can be observed in a subject who shows strong corticomuscular coherence during isometric contraction.

CONCLUSIONS

We have further characterized the interaction between beta-band activity of the sensorimotor cortex and coherent activity between the sensorimotor cortex and EMG signal. Power spectrum and EEG–EMG coherence analysis revealed that the stronger the EEG–EMG coherence peaks shown by a subject were, the larger was the TRPI during ME and MI. The results support the hypothesis that the sensorimotor cortex is active during MI tasks and that ME and MI share common functional neural networks.

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