Chapter 6.6

EMG-EEG correlation

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Introduction

The study of relationship between EMG and EEG provides us with physiological information about how activities of the cerebral cortex, mainly those of the sensori-motor cortex, are related to the movement of interest, whether it is voluntary or involuntary. In case of voluntary movement, we study the EMG-EEG correlation mainly to investigate cortical mechanisms underlying the central motor control and its disorders. On the other hand, the use of EMG-EEG correlation for the study of involuntary movements serves as a diagnostic supplement as well as a method for clarifying the physiological mechanism underlying the generation of each involuntary movement. Since the movement-related cortical electric activities are usually small as compared to the background EEG activity, they cannot be identified by visual inspection of the raw record, even if they might occur in close time relation to the movement. Therefore, the method for averaging the EEGs with respect to the time of each movement is employed for increasing the signal-to-noise ratio. In the study of movement, in contrast to the sensori-perceptive mechanisms, an emphasis is often placed on the epoch before the movement onset, which makes it necessary to average the data backward with respect to the fiducial point.

Self-initiated movements

For investigating cortical electric potentials associated with self-initiated voluntary movements (movement-related cortical potentials, MRCPs), EEGs and EMGs are simultaneously recorded while the subject repeats a voluntary muscle contraction or relaxation at a self-paced rate of once every 3 s or longer (Shibasaki 1993). EEG electrodes should be placed at least over the precentral areas, approximately C3 and C4 for hand movement and Cz for foot movement according to the international 10–20 system. Use of additional electrodes, if possible, is helpful not only for studying the distribution of the potentials over the head but also, perhaps more importantly, for identifying each subcomponent of MRCPs. Usually linked ear electrodes are used as the reference for common referential derivation. Electrode impedance must be kept below 5 kΩ. It is important to use a long time constant for recording the slow components, and thus the filter setting of amplifiers commonly used is 0.05–500 Hz (−3 dB). EMG is recorded by

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a pair of disk electrodes placed over the contracting muscle with a filter setting of 30–1000 Hz, and rectified. A fiducial point for averaging is determined when the rectified EMG exceeds a preset threshold for an on-line analysis, but an off-line analysis by visually determining the precise onset of EMG discharge as well as by excluding the trials containing various artifacts gives rise to better waveforms. The analysis window should preferably cover 3.0 s before and 0.5 s after the movement onset. For monitoring blink artifacts, electro-oculograms (EOGs) should be simultaneously recorded and averaged by using the same method as the EEG. Two or more sessions should be repeated for each kind of movements, each session consisting of about 50 trials.

In normal subjects, self-paced finger movement is preceded by a slow negative potential shift starting at about 2.0 s before the movement onset (Fig. 1). This early component is called either Bereitschaftspotential (BP), or the early component of readiness potential (RP) or NS1. BP is maximal at the midline precentral region and distributed widely and symmetrically over the scalp, regardless of the site of movement. Approximately 400 ms before the movement onset, a sharper negative slope, called negative slope (NS1), the late component of RP or NS2, appears over the precentral region contralateral to the movement. Immediately before the movement, a small negative potential is identified in some subjects, which is localized to the contralateral precentral electrode (motor potential, MP). This potential continues to increase in negativity and moves anteriorly to the midline where it peaks about 100 ms after the movement onset. According to the results of subdural recording of these potentials, the BP mainly arises from activities in the primary sensori-motor cortex and the supplementary motor areas (SMAs), both bilaterally with somatotopic organization, and the NS1 reflects activities arising from the same areas but with much more contralateral predominance (Ikeda and Shibasaki 1992).

Voluntary inhibition of muscle contraction or self-paced muscle relaxation can be studied also by adopting the same principle. For this purpose, the time of muscle relaxation can be identified as the onset of the silent period of EMG discharges or by an accelerometer, which is used as a fiducial point for averaging. In the movement induced by muscle relaxation, BP/NS1, which is quite similar to the one preceding the muscle contraction, is recorded before the muscle relaxation. In the isometric muscle relaxation, however, smaller BP/NS1 was observed in the lateral central areas as compared with the muscle contraction (Rothwell et al. 1998).

MRCPs can be applied to the investigation of pathophysiology of primary as well as non-primary motor cortices in various kinds of movement disorders. For example, BP/NS1 is often absent in patients with cerebellar lesions, especially those involving the dentate nucleus and its efferent pathway. BP was reported to be smaller in patients with Parkinson’s disease than in age-matched control subjects while NS1 was normal or even larger in the patient group. NS1 was also reported to be smaller at the contralateral central region in patients with writer’s cramp; a kind of focal dystonia.
Cued movements

In a reaction time paradigm in which a pair of stimuli (S1 and S2) are presented with the interstimulus interval of, for example, 2.0 s, and the subject is asked to make a motor task soon after S2, we can record contingent negative variation (CNV) between the two stimuli, using otherwise the same method as used for recording MRCPs. Again by subdural recording, the late component of CNV was found to be generated, in addition to the SMAs and primary sensori-motor cortex, in wider cortical areas including prefrontal cortex. The late CNV was found to be abnormal especially in patients with basal ganglia disorders such as Parkinson’s disease and focal dystonia while it was well preserved in patients with cerebellar disorders.

EEG Correlates of involuntary movements

The most fundamental method for studying EMG-EEG correlation in spontaneous involuntary movements like myoclonus is a polygraphic recording of EMGs and EEGs. This can be done by using an electroencephalograph or a cathode ray oscillograph. Filter setting of amplifiers used for recording EEGs is usually 1–500 Hz, and that for EMGs is 30–1000 Hz. Large EEG waves like periodic synchronous discharges, which are seen in association with periodic myoclonus in patients with Creutzfeldt-Jakob disease or subacute sclerosing panencephalitis, are easily identified on the conventional polygraphic records.

On the conventional polygraph, however, it is usually difficult to study a precise relationship between the EMG and EEG activities. In such cases, EEGs can be back averaged with respect to the onset of the myoclonic EMG discharge by adopting the same principle as for recording the MRCPs (jerk-locked back averaging) (Shibasaki 1988, 1993). The analysis window is usually from 400 ms before to 200 ms after the myoclonus onset. By this method, the temporal and spatial relationship between myoclonus and the EEG activity can be studied more precisely. In cases of cortical reflex myoclonus, a myoclonic jerk of the hand is preceded by a positive-negative cortical spike by about 20 ms and the latter is localized to the central region contralateral to the myoclonus. As for the generator site of the myoclonus-related cortical activity, the recent application of the jerk-locked averaging to the magnetic fields, processed in the same way as the EEG, confirmed the source in the precentral gyrus contralateral to the involved muscle (Fig. 2) (Mima et al. 1998). Even in cases in whom

![Fig. 2. Myoclonus-related magnetic fields obtained by jerk-locked back averaging in a patient with cortical myoclonus. (a) Waveforms from two different blocks of averages are superimposed. The vertical line corresponds to the onset of myoclonic EMG discharge recorded from the left wrist extensor muscle. A biphasic activity preceding the myoclonus is localized to the right central area. (b) Magnified waveform from a selected channel. (c) Contour map of the earliest peak preceding the myoclonus onset. The white arrow indicates the site of the equivalent current dipole and its direction. (From Mima et al. 1998, with permission.)](image-url)
the conventional polygraph shows no EEG spikes in association with myoclonus, this technique may disclose a spike preceding the myoclonus if that is of cortical origin. If the aim of the study is to distinguish involuntary movements such as psychogenic myoclonus, tic and chorea from voluntary movements, the low frequency filter of approximately 0.05 Hz is necessary for recording slow EEG components.

Some involuntary movements, especially myoclonus, are often induced by external stimuli, thus called stimulus-sensitive or reflex myoclonus. The stimulus-sensitive myoclonus can be studied by simultaneous recording of cortical short latency evoked potentials and long latency muscle reflexes. Cortical reflex myoclonus is the best indication for this paradigm. The most appropriate modality of stimulus should be chosen depending on the specificity of stimulus sensitivity in each individual case, but the somatosensory evoked potential (SEP) following electric stimulation of the peripheral nerve is most commonly used for eliciting the cortical reflex myoclonus. EMG is recorded from a pair of electrodes placed over the corresponding muscles of the stimulated or other extremities, and rectified. The rectified EMGs and the simultaneously recorded EEGs are averaged by using the stimulus onset pulse as a trigger. In cortical reflex myoclonus, the EMG response of a hand muscle at a latency of about 45 ms is extremely enhanced (C reflex) which corresponds to an evoked myoclonic jerk. Cortical reflex myoclonus often shows a rapid spread of the reflex myoclonus from the proximal to distal muscles within the stimulated limb and also to the muscles of the contralateral limb most likely by transcallosal conduction.

Negative myoclonus (asterixis) of cortical origin can be studied also by using the back averaging method. In this case, the onset of the EMG silent period is used as the fiducial point (silent period-locked averaging). The precise origin of the cortical negative myoclonus has not been determined, but some of them are sensitive to external stimulus just like cortical reflex positive myoclonus (cortical reflex negative myoclonus) (Shibasaki et al. 1994).

**Rhythm correlation between EEG and EMG**

EEG and EMG signals are made up of activity in several frequency bands. As described in the sections above, in the EEG at least, different frequency bands are thought to be produced by activity in different physiological circuits within the brain. If this is correct, then it is meaningful to ask whether or not single events, or even specific frequency bands in the EMG are associated with changes in the amount of activity at particular frequencies in the EEG. In order to detect relations between discrete EMG events and frequency bands in the EEG, we use the technique of event-related synchronization/desynchronization. Detecting relations between frequency bands in EMG and EEG involves techniques of coherence analysis.

**Event-related desynchronization and synchronization**

EEG and EMG signals are usually recorded continuously and stored on a computer. Manual or automatic methods are then used to identify particular events in the EMG that can be used as fiducial points about which averaging can be performed. The computer calculates a power spectrum of the EEG over short, usually overlapping intervals around the fiducial point. This will then show how the power spectrum of the EEG signal changes with time relative to the EMG event. Because three measurements have to be plotted against each other, a two-dimensional color plot is often used to illustrate the results (e.g. Baker et al. 1997). Thus, time relative to the fiducial point is often plotted on the x-axis whilst frequency band in the EEG is plotted on the y-axis. A color scale is then used to indicate the amount of power at each frequency at different times. Changes in color indicate a change in the power at that frequency band.

A slightly different method can be used if there are a priori reasons for examining particular frequency bands of EEG activity (for example around 10, 20 and 40 Hz) (Pfurtscheller and Klimesch 1991). Digital methods are used to filter the EEG to select the frequency band of interest. The resulting signal is then squared in order to give a value related to the power within the frequency...
band of interest. This can then be averaged with respect to the fiducial point in the EMG trace. A typical use of such techniques is to record the attenuation of the mu rhythm preceding and accompanying voluntary movement. Power in the EEG at around 10 Hz over the contralateral motor cortex decreases 1 to 2 s before movement onset.

Coherence analysis

The above methods in which averages of EEG activity are produced relative to a selected fiducial point in the EMG trace are often referred to as time-domain analysis. In essence, they detect activity in the EEG which is correlated with an identified event in the EMG. A rather different way of inferring the relationship between EEG and EMG events is to use frequency domain analysis (see Farmer 1998). The EEG and EMG signals are first broken down into their individual frequency components. Then, the analysis tests whether any of these components are coherent in the two sets of signals. For example, there may be a 20 Hz frequency band in both EEG and EMG traces. Coherence analysis tells us whether or not these frequencies have a constant temporal relationship with each other. The values of coherence lie between 0 and 1 where the value of 1 means that the signals are perfectly in step one with the other. The analysis also gives a phase value for the coherence that indicates the temporal relationship of the two frequency bands.

Coherence analysis does not rely on identifying specific events within the EMG or EEG. It is a continuous analysis of on-going data. There are two points to be borne in mind when using coherence analysis. First, measures of coherence do not give any idea of how much the coherent activity contributes to the total power of the two signals. To get some idea of this, it is also necessary to examine the power spectrum. For example, EEG and EMG could show strong coherence at 100 Hz, but the amount of power at 100 Hz could be extremely small in both of them. In such a case, it would be reasonable to ask how important this coherent signal was to performance of the task being studied. The second point relates to changes in coherence during performance of different tasks. For example, coherence at 20 Hz may be high during a constant contraction, but much lower during a phasic contraction, even at similar levels of EMG. The simplest explanation of this would be that whatever was causing the coherence between the signals was much less during phasic than during tonic contraction. However, this need not be the case. It is possible that the process producing coherence is still operating as normal during the phasic contraction, but that additional processes are recruited which produce activity at 20 Hz that is not coherent between the EEG and EMG. The presence of these additional uncorrelated processes means that the fraction of the signal explained by coherent activity would drop during phasic contraction even though the absolute amount of coherence would be the same.

Coherence analysis is widely available in several software packages. In general, the EEG is recorded with relatively open filters and the EMG signal is rectified before performing the analysis. In addition to providing measures of coherence at different frequency bands, analysis will also provide the phase of the coherent activity which gives some idea of the temporal relationship between the signals. However, phase plots always have the problem of knowing the direction of the correlation since a 100° phase lead is equal to a 260° phase lag, and therefore in most cases it is necessary to have additional data to support inferences about the time course of activation.

References

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