Functional significance of the mu rhythm of human cortex: an electrophysiologic study with subdural electrodes *

Santiago Arroyo a, Ronald P. Lesser a,b,c,d, Barry Gordon a,c,d, Sumio Uematsu b, Darryl Jackson a and Robert Webber a

The Johns Hopkins Epilepsy Center, a Department of Neurology, b Department of Neurosurgery, and c Cognitive Neurology Division, The Johns Hopkins Hospital, Baltimore, MD 21287-7247 (USA), and d The Zanevyl Krieger Mind / Brain Institute, The Johns Hopkins University School of Medicine, Baltimore, MD (USA)

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Summary

The existence of the mu rhythm and its general anatomical and physiological relationships are well known. There are few data, however, regarding the details of its anatomical and physiological specificity. We implanted fronto-temporal subdural electrode grids in 9 patients with intractable epilepsy to facilitate their surgical management. A 7–11 Hz cortical mu rhythm was observed in 5–16 electrodes located over the sensorimotor cortex as mapped by electrical stimulation. The mu rhythm was blocked by contralateral face and arm movements, passive movements of contralateral arm, and by ipsilateral arm movements. There was correspondence between the body area movement of which blocked the mu at a given site and the body region that was affected by stimulation at the same site. Power spectral analysis showed an overall decrease in power in all frequency bands. This was less prominent in the 14–100 Hz band resulting in a relative increase in high frequency power in association with movement.

We conclude that both the presence and blocking of mu rhythm are specific to the somatic representation of the cortex from which it is recorded. Its functional significance may be similar to other sensory rhythms like the occipital alpha rhythm.

Key words: Mu rhythm; EEG; Functional localization; Subdural electrodes; Cortical mapping; High frequency activity

It is well known that a characteristic EEG rhythm occurs over the central areas of the scalp and that it is reactive to sensorimotor stimuli. It is most widely known as the mu rhythm (Gastaut 1964; Gastaut et al. 1965; Schoppenhorst et al. 1977; Kuhlman 1978). Several studies have shown that the mu rhythm can be blocked by movement of the contralateral hand/arm (Magnus 1954; Kuhlman 1978), passive movements of the contralateral hand/arm (Gastaut 1952; Kuhlman 1978), and by attention to or preparation for activity (Magnus 1954; Chatrian et al. 1959; Schoppenhorst et al. 1980). Ipsilateral active or passive (Chatrian et al. 1958, 1959) movements of the arm also can block the mu rhythm, although this is usually less pronounced than when contralateral activity is performed. Mu also is reported to be blocked by contralateral body tactile stimulation (Magnus 1954; Chatrian et al. 1959), and the planning of voluntary movement (Chatrian et al. 1959; Pfurtscheller and Aranibar 1977). Interestingly, it is enhanced by intermittent photic stimulation (Brechet and Lecasble 1965) and patterned vision (Koshino and Niedermeyer 1975).

The prevalence of the mu rhythm in routine EEGs has been estimated to be from 2.8 to 16% (Klass and Bickford 1957; Chatrian et al. 1959; Bostem et al. 1964; Schnell and Klass 1966; Koshino and Niedermeyer 1975), and to be as high as 34% in children (Netchine et al. 1965). The presence of the mu rhythm once was related to neuropsychiatric and psychosomatic disease (Bostem et al. 1964, 1965), but it is now apparent that it is a normal cortical rhythm (Chatrian et al. 1959; Gastaut 1964; Kuhlman 1978). In fact, frequency analysis methods suggest that it can be present in 50–100% of healthy subjects (Schoppenhorst et al. 1977, 1980; Storm van Leeuwen et al. 1978; Pfurtscheller and Aranibar 1979).

Only a few case reports have described rhythmical activities in sensorimotor regions of the human cortex

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Correspondence to: Santiago Arroyo, M.D., Department of Neurology, Meyer Building 2-147, The Johns Hopkins Hospital, 600 North Wolfe Street, Baltimore, MD 21287-7247 (USA).
Tel.: (410) 955-1270; Fax: (410) 955-0751.

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CORTICAL MU RHYTHM

(Jasper and Penfield 1949; Gastaut 1952; Penfield 1954; Sem-Jacobsen et al. 1955; Graf et al. 1984; Lüders et al. 1987). In 4 reports, the rhythm was described to be at 11–12 Hz (Gastaut 1952; Sem-Jacobsen et al. 1955; Graf et al. 1984; Lüders et al. 1987) and, in the others, in the beta range (25 Hz) (Jasper and Penfield 1949; Penfield 1954).

We have studied the cortical rhythms over the sensorimotor cortex via large arrays of implanted subdural electrodes in patients who are candidates for epilepsy surgery to examine the following issues: First, what spatial relation has the mu rhythm with the sensorimotor cortex? Second, what functional somatotopisms exist for this rhythm? Third, why were the sensorimotor region rhythmical activities found in two different frequency ranges by different authors, as just described (Jasper and Penfield 1949; Gastaut 1952; Penfield 1954; Graf et al. 1984; Lüders et al. 1987)? Fourth, is there any other significant reactive activity present in the sensorimotor cortex?

Methods and materials

The mu rhythm was defined by the following features: it is a sinusoidal rhythm over the fronto-central cortex that occurs in the awake and relaxed state and that has a frequency range between 7 and 11 Hz. MU rhythm is not blocked by opening and closing of the eyes, but is blocked by a diversity of movements and sensory stimuli, from contralateral or ipsilateral limbs (Gastaut 1952, 1954; Magnus 1954; Chatrian et al. 1959; Cohen et al. 1964; Kuhlman 1978). The mu

TABLE I

Patient’s characteristics.

<table>
<thead>
<tr>
<th>No. 1</th>
<th>No. 2</th>
<th>No. 3</th>
<th>No. 4</th>
<th>No. 5</th>
<th>No. 6</th>
<th>No. 7</th>
<th>No. 8</th>
<th>No. 9</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>34</td>
<td>23</td>
<td>36</td>
<td>42</td>
<td>31</td>
<td>30</td>
<td>39</td>
<td>18</td>
</tr>
<tr>
<td>Sex</td>
<td>M</td>
<td>F</td>
<td>F</td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>M</td>
<td>M</td>
</tr>
<tr>
<td>Seizure onset (years)</td>
<td>10</td>
<td>19</td>
<td>15</td>
<td>23</td>
<td>28</td>
<td>10</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td>Types of seizures</td>
<td>CPS</td>
<td>CPS</td>
<td>Gelastic</td>
<td>CPS</td>
<td>CPS</td>
<td>CPS</td>
<td>CPS</td>
<td>CPS</td>
</tr>
<tr>
<td>MRI</td>
<td>Normal</td>
<td>Normal</td>
<td>Left mesial frontal vascular lesion</td>
<td>Left anterior inferior temporal encephalomalacia</td>
<td>Left frontal venous angioma</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Etiology of seizures</td>
<td>Unknown</td>
<td>Unknown</td>
<td>Cavernous angioma</td>
<td>Head trauma</td>
<td>Left temporal</td>
<td>Left mid and posterior temporal</td>
<td>Right temporal</td>
<td>Left temporal</td>
</tr>
<tr>
<td>Ictal scalp localization</td>
<td>Left fronto-temporal</td>
<td>Left fronto-temporal</td>
<td>Left and right temporal</td>
<td>Left temporal</td>
<td>Left F-T and occipital</td>
<td>Right F-T</td>
<td>Left F-T</td>
<td>Left T</td>
</tr>
<tr>
<td>Mu rhythm in scalp Grid placement</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Ictal grid localization</td>
<td>Left F-T</td>
<td>Left F-T</td>
<td>Left F and mesial frontal</td>
<td>Left F-T</td>
<td>Left F-T</td>
<td>Right F-T</td>
<td>Left F-T</td>
<td>Left T</td>
</tr>
<tr>
<td>Electrical stimulation</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes *</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Mu tested ***</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

* Patient no. 4 had a clot between the frontal grid and the cortex. Consequently stimulation procedures did not elicit any sensorimotor responses. EEG recording could be performed, however.

*** Stimulation procedures were not performed in the frontal lobe.

*** In addition to the bipolar recording, in those patients tested, mu rhythm was also recorded with referential montage. We chose as reference that subdural electrode over the frontal or temporal lobe that was most inactive during several days of recording. This electrode was 3–7 cm (mean 5.8 cm) from the electrodes displaying the mu rhythm and did not itself show this activity.

Abbreviations: F = lateral frontal and postcentral regions, T = lateral and basal temporal regions, CPS = complex partial seizures, M = male, F = female.
rhythm had to stand out from the background and had
to be clearly identifiable by simple record reading (not
by power spectral density measurement).

Unilateral lateral frontal-parietal-temporal grids of
subdural electrodes were implanted in 9 candidates for
epilepsy surgery (Table I). Most patients had complex
partial seizures of suspected fronto-temporal origin.
We routinely cover with subdural electrodes the basal
and lateral temporal regions and additional electrodes
are placed over the lateral frontal lobe depending of
the clinical situation of the patient. In most patients,
however, we only place 2 or 3 rows of subdural elec-

Fig. 1. Three-dimensional computerized tomography in patient no. 4 showing the subdural grid arrangement over the left temporal (basal and
lateral) and inferior lateral frontal lobes. A: lateral view. B: inferior view.


drodes over the lateral frontal regions (Fig. 1). This is
done in part to rule out frontal ictal onset, and in part
to define the location of the rolandic sulcus since one
traditional teaching holds that it is safe to resect tem-
poral lobe back directly inferior to the rolandic sulcus.
In all of the patients except in no. 3 we placed exten-
sive arrays of electrodes over the basal and lateral
temporal regions. Patients were continuously moni-
tored for 10–14 days, during which EEG-video record-
ing and functional mapping by means of cortical stimu-
lation were performed (Lesser et al. 1987; Lüders et al.

The subdural electrodes are composed of a plat-
inum-iridium alloy with electrode contacts of 5 mm
diameter with 2 mm exposed to the cortex and with
center to center distances of 1 cm between electrodes.
The electrodes are embedded in a thin transparent
silastic plate in which the electrodes are laid out in a
rectangular array (Lüders et al. 1987).

EEG was recorded with a customized EEG monitor-
ing system in which the information is digitized at 200
Hz (Nyquist frequency of 100 Hz) (Lesser et al. 1992).
We employed a 0.3 Hz high pass filter, a 60 Hz notch
filter and 100 Hz low pass filter. A 100 Hz filter may
have not been sufficient for preventing aliasing when
sampling data at 200 Hz. It is our experience, however,
that, when recording at a 400 Hz sampling rate, there
is no significant power above 50 Hz except during
seizures (Fisher et al. 1992). Concurrent EMG record-
ing was performed with surface electrodes attached
bilaterally to the face (buccinator muscle), arms
(supinator muscle) and legs (anterior tibialis muscle).
Also, an electrooculogram was obtained with skin sur-
face electrodes placed next to the superior and inferior
canthi of the eye.

Six patients were tested for approximately 45 min
during which reactivity (blocking) of the mu rhythm
was studied. Mu rhythm reactivity was assessed by
visual inspection. We defined a mu blocking as a
decrease of more than 90% of the baseline mu ampi-
utude. During the testing procedures the patient was
asked to perform the following tasks: relax with closed
eyes, unilateral (ipsi- and contralateral) and bilateral
sustained face contraction, tongue movement inside
the mouth, sustained fisting and opening and closing of
the hands (ipsilateral and contralateral), dorsal flexion
of the feet, mental calculation (serial 7 subtractions from 100), thinking about movements of the contralateral arm or face, closing and opening of the eyes and eye movements in all directions. We tested mu rhythm reactivity to sensory stimulation (touch) and passive arm movements. Finally, we asked patients to respond to 1 of 3 numbers by making 1 of 3 different movements. For example, the first number meant right hand fisting, the second right face contraction, and the third no movement. Those tasks in which the mu rhythm appeared to be blocked were repeated 3 times in order to assess reproducibility. EEG traces with interictal spikes over the fronto-temporal regions were disregarded for the study and for the power density analysis.

Electrical stimulation was performed for functional localization of the motor sensory and language areas following a standard protocol (Lesser et al. 1987). Informed consent were signed by all patients. Cortical mapping was performed with electrical stimulation delivered with a constant current patient-isolated stimulator. We used 0.3 msec pulses of alternating polarity in trains of 50 pulses/sec of 1–5 sec duration. We started at a low intensity, 0.5–1 mA, and increased stepwise at 0.5–1 mA intervals, until 1 of 3 endpoints

Fig. 2. A: in patient no. 1 the mu rhythm over electrodes 4, 5, 6, 7, 13, 14, 15, 16, 22, 23, 24, 30, 31, and 32 (left lateral frontal and postcentral grid) is blocked by face contraction. Although the patient was asked to move only the right side of his face, note the EMG artefact over both sides of the face. Abbreviations: EMG = electromyogram; RF = right face; LF = left face; RA = right arm; LA = left arm; RL = right leg; LE-RE = electrooculogram. B: a schematic layout of the placement of the subdural electrodes over the left lateral frontal, postcentral and temporal cortex in patient no. 1. Abbreviations: f = arrest of the face/tongue movements; a = arrest of the arm/finger movements; F = movements of the contralateral face/tongue; A = movement of the contralateral arm/fingers; sf = sensation over the face/tongue; sa = sensation over the arm/fingers; * = "body part specific" blocking of the mu; 0 = mu rhythm. Note: eye movements and language results are not represented.
was achieved: functional changes appeared, afterdischarges were recorded, or the intensity limits of the stimulators we use (15–17.5 mA) were reached (Lüders et al. 1987, 1988; Gordon et al. 1990). To define primary motor area we looked for movements of resting body parts in response to stimulation (e.g., twitching or sustained contractions of a finger or the tongue). Pre-motor area was defined as being localized beneath electrodes where stimulation altered continuation of an ongoing activity such as rapid alternating movements of the fingers, hands, tongue, etc.

The frequency spectra for the data from EEG channels that showed the mu rhythm were analyzed by performing a Fast Fourier Transformation of the data. Power spectra were divided in the following frequency bins: 0–6, 7–13, and 14–100 Hz. A 7–13 frequency bin was chosen to include all the frequencies at which alphoid central rhythmical activities have been described (Gastaut 1952; Magnus 1954; Chatrian et al. 1959; Netchine et al. 1965; Schoppenhorst et al. 1977). The other two bins were chosen to reflect frequencies above and below 7–13 Hz, respectively. We took two samples of 2.6 sec in one channel that displayed the mu rhythm: one while the patient was at rest and relaxed, and another while the patient was performing the action that blocked the mu rhythm. We also calculated the total power (TP) in the channel before and during activity and we compared the relative percentage of the power for each selected band in these situations. Statistical significance of the difference in

![Figure 3](image-url)

**Fig. 3.** A: in patient no. 2 the mu rhythm is blocked in electrodes P3-P8, P4-P9, P5-P10 when alternating right hand movements are performed. Those electrodes correspond to arm/hand functional areas as determined by electrical stimulation. Mu rhythm over face area (electrodes P5, P6) is not blocked by arm movement.
Fig. 3. B: in patient no. 2 right face contraction (note the EMG artifact at the onset) blocks mu rhythm over electrodes 4, 5, 6, 7, but the blockage is sustained in electrodes 5 and 6. These electrodes are over face areas as determined by electrical stimulation. Mu rhythm over arm/hand areas (P3-P8, P4-P9, P5-P10) is not blocked by face movement. C: a schematic layout of the placement of the subdural electrodes over the left lateral frontal, postcentral and temporal cortex in patient no. 2. Observe that 6 of the 8 electrodes presenting arm/fingers motor activity (F) have mu rhythm, and that 9 out 16 electrodes presenting face/tongue motor activity (F) have mu rhythm. Mu rhythm is present in 7 of the 8 electrodes that represent sensory areas (sf and sa). Abbreviations used are the same as in Fig. 1.
TABLE II

Somatotopism of mu rhythm.

<table>
<thead>
<tr>
<th>No. of electrodes over the lateral frontal and parietal regions</th>
<th>No. 1</th>
<th>No. 2</th>
<th>No. 3</th>
<th>No. 4</th>
<th>No. 5</th>
<th>No. 6</th>
<th>No. 7</th>
<th>No. 8</th>
<th>No. 9</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of electrodes with the mu rhythm</td>
<td>24</td>
<td>34</td>
<td>64</td>
<td>16</td>
<td>64</td>
<td>24</td>
<td>28</td>
<td>40</td>
<td>24</td>
</tr>
<tr>
<td>Functional (motor/sensory) areas mapped</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(no. of electrodes)</td>
<td>F (10)</td>
<td>F (18)</td>
<td>A (8)</td>
<td>F (16)</td>
<td>A (12)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Functional areas where the mu rhythm was present</td>
<td>F (7)</td>
<td>F (9)</td>
<td>A (6)</td>
<td>A (8)</td>
<td></td>
<td>A (6)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(no. of electrodes with both the mu rhythm and a primary function mapped)</td>
<td>5</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>13</td>
<td>13</td>
<td>6</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Movement which specifically blocked the mu rhythm ***</td>
<td>F</td>
<td>F/A</td>
<td>A</td>
<td>F</td>
<td>NT</td>
<td>F</td>
<td>F</td>
<td>NT</td>
<td>F</td>
</tr>
</tbody>
</table>

* Patient no. 4 had a clot between the frontal grid and the cortex. Consequently stimulation procedures did not elicit any sensorimotor responses.

** In the arm functional area mu rhythm was blocked by arm movement, and in the face area by face movement.

*** Multiple modalities were studied in the patients. Refer to Table III.

F = face/tongue, A = arm/hand, L = leg, NT = not tested.

Results

A 7–11 Hz cortical rhythm occurred over the rolandic cortex in all patients. Mu rhythm was present in a mean of 10 electrodes per patient (range 5–16 electrodes). Mu was recorded by 44 (55.7% of the total electrodes presenting mu rhythm) of the electrodes over areas that exhibited sensorimotor function as mapped by electrical stimulation and in 35 (44.3%) of the electrodes over areas without mapped function. (Patients no. 4 and 6 are not included in these calculations due to lack of stimulation results; see Table II). The mu rhythm was blocked by (Table III): movement of the contralateral face (in 5 patients), movement of the tongue inside the mouth (in 4), movement of the contralateral arm/hand (in 2), or passive movements of the contralateral arm (in 1).

In 4 of the 5 patients with mu rhythm blocked by contralateral face movement, the rhythm was observed over the cortex defined to be face/tongue area by cortical stimulation (Fig. 2A, B and Table II). In the fifth no stimulation testing could be performed over the frontal lobe due to the presence of a blood clot between the grid and the frontal cortex. Ipsilateral face
movements also blocked muscle activity in all frequency bands and decreased relative power in the 7-13 Hz and a significant decrease in relative power in the 14-100 Hz band. The mu rhythm was blocked by specific activities, as shown in Table IV. We observed a significant decrease in relative power in the 7-13 Hz band for movements that blocked the mu rhythm in the 14-100 Hz band. The mu rhythm was attenuated in all patients during sleep. The mu rhythm was not blocked in any patient by sensory (touch) stimulation, mental activation such as calculation, eye movements, opening or closing the eyes, or thinking about movements. Finally, sensory (touch) stimulation, mental activation such as calculation, eye movements, opening or closing the eyes, or thinking about movements.

In two cases the mu rhythm was seen over the arm/hand regions, and was blocked by active contralateral passive arm movements, in one patient this observed mu was blocked by contralateral passive arm movements. In one patient the mu rhythm over the arm/hand regions was blocked by active arm movement on the contralateral side. Therefore, it is impossible to know if truly ipsilateral face contraction had blocked the mu rhythm. In the 14-100 Hz band the mean percentage of the TP was 33.9% (+9.3) at rest and 98.4% (+5.6) during the activity that blocked the mu rhythm in that channel (P < 0.001).
Discussion

The present study shows the anatomical and functional characteristics of the mu rhythm in the human cortex. First, the cortically recorded mu rhythm was in the same frequency range as and had morphology similar to the mu rhythm recorded at the scalp. Despite its presence in cortical recordings, however, we observed this rhythm in the scalp recordings in only two of the patients. Second, the mu rhythm was blocked by contralateral face and arm movements, contralateral passive movements of the arms and, in some cases, by ipsilateral movement. Also, it was blocked in a non-specific way by attention and alerting, but this blockage was not sustained. Third, there was correspondence among areas with sensorimotor function as mapped with electrical stimulation and areas showing the presence of the mu rhythm. However, this correspondence was not complete, and mu rhythm was present in areas where there was no function mapped and there were functional areas without mu rhythm present. Fourth, in one patient the mu rhythm was present not only in the arm/hand cortical areas, but also in the face/tongue areas. The mu rhythm in either place was functionally independent and functionally specific with respect to its reactivity. There was correspondence between which body area movement blocked the mu at a given site and the body region that was affected by stimulation at the same site. Thus, both the presence and blocking of the mu rhythm corresponded to the specific somatic representation of the cortex from which it was recorded.

Although the presence of the mu rhythm in scalp EEG recordings is well known, to our knowledge recordings of the mu rhythm directly from the human somatosensory cortex have been reported in only a few patients using direct recording by electrocorticography during neurosurgical procedures (Jasper and Penfield 1949; Gastaut 1952; Penfield 1954; Graf et al. 1984), subdural electrode implantation (Lüders et al. 1987) and depth electrodes (Sem-Jacobsen et al. 1955). In two of these cases cortical functional mapping was done by means of electrical stimulation (Jasper and Penfield 1949; Gastaut 1952; Penfield 1954). Jasper and Penfield (1949) and Penfield (1954) described a patient with a cortical rhythm in the beta frequency (25 Hz) over the arm sensorimotor cortex that was blocked by contralateral arm movements. While the activity was blocked, the EEG displayed a “desynchronized” activity. However, the authors acknowledged that besides the beta blocking response, contralateral arm movement also blocked a less impressive 10–12 Hz activity over the same areas. In contrast, Gastaut (1952), Graf et al. (1984) and Lüders et al. (1987) reported cases of patients that had an 11–13 Hz rhythm over the sensorimotor cortex. In one case (Gastaut 1952), the rhythm was blocked by contralateral arm movements and there was a correspondence between the cortical area where the mu was present and the functional area determined by electrical stimulation. No report described a reactive face/tongue mu rhythm.

Gastaut (1952) explained the discrepancy in frequency between the reactive sensorimotor rhythm...
Fig. 5. Percentage of the total power at rest and during the activity that specifically blocked the mu rhythm in the 7-13 Hz and 14-100 Hz frequency bands. Note that during movement there is a relative decrease in the power of the 7-13 Hz band and an increase in the 14-100 Hz band in all patients (except for no. 9 in the 14-100 Hz band). (**)Power for patient no. 2 measured in a channel where mu rhythm was blocked by movement of contralateral arm or face (**).
al. 1991; Uematsu et al. 1992). The fact that the mu rhythm was responsive both to motor activation and to passive arm movements (proprioceptive input) is congruent with these notions.

In our study, when mu rhythm was blocked there was an overall decrease in power density and a relative increase of high frequency power appeared. This is consistent with previous work from Pfurtscheller et al. who showed, in scalp recorded EEG using event-related desynchronization, a decrease in the mu power spectra with simultaneous increase in the beta power in response to movement (Pfurtscheller and Aranibar 1977; Pfurtscheller 1989; Pfurtscheller and Klimesch 1992). It also has been reported by the same authors that preparation for movement produces attenuation of the 10–12 Hz activity and increase in high frequency power in the central region of the cortex (Pfurtscheller and Aranibar 1979; Pfurtscheller and Berghold 1989). This would agree with our finding that preparation for movement produced attenuation of mu rhythm.

Our results and previous conclusions support the idea that the mu rhythm in the human cortex may represent a neuronal oscillatory behavior while the subject is resting and awake. The thalamus probably is involved in this oscillatory behavior. However, alerting or specific stimuli (movement, proprioceptive change) produce a change in the oscillatory behavior with a shift to higher frequency activity (Bressler 1990). Higher frequencies may have a functional significance, for example, due to their specific frequency range, their distribution over the cortex, or their amplitudes.

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