Invited review

What is the Bereitschaftspotential?

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Abstract

Since discovery of the slow negative electroencephalographic (EEG) activity preceding self-initiated movement by Kornhuber and Deecke [Kornhuber HH, Deecke L. Hirnpotentialänderungen bei Willkurbewegungen und passiven Bewegungen des Menschen: Bereitschaftspotential und reafferente Potentiale. Pflugers Archiv 1965;284:1–17], various source localization techniques in normal subjects and epicortical recording in epilepsy patients have disclosed the generator mechanisms of each identifiable component of movement-related cortical potentials (MRCPs) to some extent. The initial slow segment of BP, called ‘early BP’ in this article, begins about 2 s before the movement onset in the pre-supplementary motor area (pre-SMA) with no site-specificity and in the SMA proper according to the somatotopic organization, and shortly thereafter in the lateral premotor cortex bilaterally with relatively clear somatotopy. About 400 ms before the movement onset, the steeper negative slope, called ‘late BP’ in this article (also referred to as NS^0), occurs in the contralateral primary motor cortex (M1) and lateral premotor cortex with precise somatotopy. These two phases of BP are differentially influenced by various factors, especially by complexity of the movement which enhances only the late BP. Event-related desynchronization (ERD) of beta frequency EEG band before self-initiated movements shows a different temporospatial pattern from that of the BP, suggesting different neuronal mechanisms for the two. BP has been applied for investigating pathophysiology of various movement disorders. Volitional motor inhibition or muscle relaxation is preceded by BP quite similar to that preceding voluntary muscle contraction. Since BP of typical waveforms and temporospatial pattern does not occur before organic involuntary movements, BP is used for detecting the participation of the ‘voluntary motor system’ in the generation of apparently involuntary movements in patients with psychogenic movement disorders. In view of Libet et al.’s report [Libet B, Gleason CA, Wright EW, Pearl DK. Time of conscious intention to act in relation to onset of cerebral activity (readiness-potential). The unconscious initiation of a freely voluntary act. Brain 1983;106:623–642] that the awareness of intention to move occurred much later than the onset of BP, the early BP might reflect, physiologically, slowly increasing cortical excitability and, behaviorally, subconscious readiness for the forthcoming movement. Whether the late BP reflects conscious preparation for intended movement or not remains to be clarified.

Keywords: BP, Bereitschaftspotential; Pre-movement slow negativity; Early BP; Late BP (NS^0); Conscious will to move

1. Introduction

Kornhuber and Deecke (1964) made the first report of electroencephalographic (EEG) activity preceding volitional movement in humans. Prior to that, Bates (1951) attempted to record the movement-related activity by photographic superimposition of multiple single-sweep EEG traces, but he could identify only the post-movement activity probably due to low signal-to-noise ratio. In the 1960’s, no computer software for making on-line back averaging was available. Therefore, Kornhuber and Deecke (1964) recorded EEG and electromyogram (EMG) simultaneously while the subjects were repeating movements at a self-paced rate, and stored all the data on magnetic tape. Then they made an off-line averaging of the EEG segment prior to the EMG onset by playing the tape backward. By using this chronologically reversed averaging

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technique, they successfully identified two components, one each before and after the EMG onset. Those were the Bereitschaftspotential (BP) or readiness potential (RP), and reafferente Potentiale (Kornhuber and Deecke, 1964, 1965). Later they found two more components just before the movement onset: pre-motion positivity (PMP) and motor potential (MP) (Deecke et al., 1969). Since then, a number of studies on the movement-related cortical potentials (MRCP) have been reported both in terms of physiological findings and clinical application, but the physiological significance of each identifiable component, among others that of BP, has not been fully clarified yet. Libet et al. (1983), by employing their novel technique in which the subjects were requested to remember the time of their actual awareness of intention to move by watching a clock, reported that the intention to move occurred much later than the onset of BP. Their report has brought up a continuing question as to the physiological implication of the BP (Klein, 2002; Eagleman, 2004). In 2003, a comprehensive book entirely devoted to “The Bereitschaftspotential” was published (Jahanshahi and Hallett, 2003). Since, in that book, each specific aspect of BP was discussed in detail, it seems now appropriate to integrate various aspects of the BP by further updating more recent findings with special emphasis placed on the information obtained by epicortical recording, the issue of voluntary motor inhibition, praxis movement, and the physiological implication of the BP.

2. Components of MRCP

Different terminologies have been proposed for identifiable components of MRCP (Table 1). Shibasaki et al. (1980a), based on the scalp distribution of averaged data across 14 subjects, identified 8 components, 4 each before and after the movement onset (BP, NS', P−50, N−10, N+50, P+90, N+C010, and P+300 for finger movements) (Fig. 1). In this terminology, each component, except for BP and NS', was named according to the surface polarity (P, positive; N, negative) and the mean time interval in ms between the peak of each component and the peak of the averaged, rectified EMG, the interval being designated negative if the peak occurred before the EMG peak, and positive if it occurred after the EMG peak (Table 1). The peak time of each component with respect to the movement onset obviously differs depending on how to define the movement onset. In case of finger movements, the onset of the mechanogram is about the same time as the EMG peak, but would lag the EMG onset by about 30 ms. These days it is most common to use EMG onset as the fiducial point.

BP starts about 2.0 s before the movement onset. It is maximal at the midline centro-parietal area, and symmetrically and widely distributed over the scalp regardless of the site of movement. The onset of BP with respect to the movement onset significantly differs among different conditions of movement and among subjects. For example, in the experimental setting in which the subject is requested to repeat the same movement at a self-paced rate of once every 5 s or longer, the BP commonly starts much earlier and is maximal over the contralateral central region (C2) (Fig. 1). Another negative peak occurring shortly after N−10 is localized over the midline frontal region and corresponds to N+50 or the frontal peak of motor potential (fpMP).

Table 1
Terminology of movement-related cortical potentials

<table>
<thead>
<tr>
<th>Pre-movement components</th>
<th>Post-movement components</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kornhuber and Deecke (1965)</td>
<td>BP</td>
</tr>
<tr>
<td>Vaughan et al. (1968)</td>
<td>N1</td>
</tr>
<tr>
<td>Shibasaki et al. (1980a)</td>
<td>BP</td>
</tr>
<tr>
<td>Dick et al. (1989)</td>
<td>NS1</td>
</tr>
<tr>
<td>Lang et al. (1991)</td>
<td>BP1</td>
</tr>
<tr>
<td>Tarkka and Hallett (1991)</td>
<td>BP</td>
</tr>
<tr>
<td>Kristeva et al. (1991)</td>
<td>RF</td>
</tr>
<tr>
<td>Cui and Deecke (1999)</td>
<td>BP1</td>
</tr>
</tbody>
</table>

| a | Peak of each component, except for BP and NS', was measured from the peak of averaged, rectified EMG. |
| b | Based on movement-related magnetic fields. |
earlier as compared to the movement executed in more natural conditions, because, in such experimental conditions, the subject has a much longer time to prepare for the movement. As pointed out originally by Kornhuber and Deecke (1964, 1965) and in their subsequent reports (Deecke et al., 1969, 1976), and later by Kutas and Donchin (1980), BP suddenly increases its gradient about 400 ms before the movement onset. Based on the clearly different scalp distribution of the late steeper slope from that of the early slow shift, Shibasaki et al. (1980a) designated the late segment as Negative Slope (NS'). The NS' was distinguished from the early BP based on abrupt increase of the gradient at the central electrode corresponding to the movement for each individual subject, instead of arbitrarily setting the time such as 500 ms before the movement onset for the distinction of the two slopes. In order to avoid confusion about the use of the term BP, however, in this review article we call the early segment 'early BP' and the late segment 'late BP', and just BP for the early BP and the late BP inclusive (Fig. 1). The late BP is maximal over the contralateral central area (approximately C1 or C2 of the International 10–20 System) for the hand movement and at the midline (approximately Cz) for the foot movement (Shibasaki et al., 1981). For the study of BP in individual subjects, therefore, it is important to record EEG from multiple electrodes, including C1 and C2 for the study of hand movement, for identifying the abrupt increase of the gradient. Later, the late steeper slope was called NS2 to contrast it with the earlier NS1 by Dick et al. (1989), and BP2 to differentiate it from the earlier BP1 by the group of Deecke (Lang et al., 1991; Cui and Deecke, 1999). Initially, the late BP was thought to be more specific for the site of movement while the early BP was thought to represent the more general preparation for the forthcoming movement because of its diffuse distribution. However, as will be discussed later, the early BP might be also movement site-specific at least within the supplementary motor area (SMA) and the lateral pre-motor cortex. Its midline maximal, symmetric distribution is most likely due to the summation of electrical fields generated from homologous areas of both hemispheres via volume conduction.

The asymmetric distribution of the late BP associated with unilateral hand movement has been studied as the lateralized readiness potential (LRP) (Coles et al., 1988). It is derived by subtracting the potential recorded at C4 from that at C3 for both the left-hand movement and the right-hand movement separately. In the field of behavioral psychology, LRP has been mainly obtained in a choice reaction time task, instead of a self-paced motor task, in order to investigate the time relationship between the stimulus evaluation system and the response activation system. For example, if the former can pass information to the latter before evaluation is completed, it is considered to suggest the evidence for ‘early communication’. Hence, the details of LRP will not be dealt with in this review article.

PMP or P−50 is predominant over the hemisphere ipsilateral to the moving hand (Deecke et al., 1969; Shibasaki et al., 1980a). Based on the fact that this component was not seen with bilateral simultaneous hand movements, Shibasaki and Kato (1975) proposed that it might be related to the suppression of movement of the opposite hand in intended unilateral hand movement (suppression of the physiological mirror movements). However, PMP might be just a trough formed between two successive negative potentials, and this peak itself might not have any physiological significance. In fact, P−50 has not been identified by epicortical recording.

As for MP or N−10, it is well localized to a small area of the contralateral central scalp, precisely corresponding to the movement site, and occurs immediately before the movement onset. N−10 in hand movement was named based on the mean time interval from its peak to the peak of the averaged, rectified EMG: the latter lagged the former by 10 ms. Thus, this component most likely represents the activity of pyramidal tract neurons in the primary motor cortex (M1). The isMP (initial slope of MP) as named by Tarkka and Hallett (1991) corresponds to the early part of MP (Table 1).

As for the post-movement components of MRCP associated with hand movement, Shibasaki et al. (1980a) named the four peaks based on the time interval measured from the peak of the averaged, rectified EMG to the peak of each identifiable peak (Table 1). The component N+50 is a prominent negative peak localized to the frontal region and corresponds to fpMP (frontal peak of motor potential) of Tarkka and Hallett (1991) (Fig. 1). The component P+90 is predominant over the parietal region, larger over the contralateral hemisphere. The peaks of N+50 and P+90 showed similar scalp distribution to those of N70 and P65, respectively, of the averaged EEG responses to the passive finger movements, suggesting that these peaks are related to kinesthetic feedback (Shibasaki et al., 1980b). However, due to the different peak time after the movement onset between N+50 and P+90, it is still undetermined whether or not these two peaks are derived from a tangentially oriented dipole sitting in the postcentral gyrus. The component N+160 is localized to the contralateral parietal area, thus forming a localized positive–negative complex with P+90. The component P+300 corresponds to reafferent Potentiale of Kornhuber and Deecke (1964).

Based on the recording of movement-related magnetic fields, BP, MP and four post-movement components were named readiness field (RF), motor field (MF) and movement-evoked field (MEF) I, II and III and post-movement field (PMF), respectively, by Kristeva et al. (1991) (Table 1).

This review article will focus on the pre-movement components, especially the slow negative shifts (early BP and late BP) recorded under the self-paced conditions, and those recorded under the cued condition will not be covered.
3. Factors influencing BP

The magnitude and time course of BP recorded in the self-paced condition are influenced by various factors such as level of intention, preparatory state, movement selection as to freely selected versus fixed, learning and skill acquisition, pace of movement repetition, praxis movement, perceived effort, force exerted, speed and precision of movement, discreteness and complexity of movement, and pathological lesions of various brain structures. Since this issue was extensively reviewed by Lang (2003), this review article refers to more recent findings and summarizes the current consensus on this issue in Table 2.

As regards the force exerted in isometric muscle contraction, Slobounov et al. (2004) found a larger amplitude of the last 100 ms segment of BP with greater perceived effort rather than the force itself, while other segments were not influenced. Since the exertion of stronger force is commonly associated with the greater intention, motivation and effort of the subject, the effects of these psychological and physical factors cannot be clearly distinguished from each other. Masaki et al. (1998), by comparing a target force production task with simple task, showed that the movement requiring precision in terms of force production was found to be preceded by a larger late BP than the simple task. The speed of movement also affects the onset and magnitude of BP; the faster the movement is executed, the later (closer to the movement onset) the BP begins. As for the site of movement, Jankelowitz and Colebatch (2002) found larger late BP in self-paced movement of the proximal than the distal part of upper extremity.

The effect of discreteness and complexity of the movement on BP is noteworthy. Kitamura et al. (1993a) compared isolated extension of the middle finger with simultaneous extension of the middle and index fingers, and found larger amplitude of late BP, but not early BP, in the isolated movement of the middle finger as compared with the simultaneous movement of the two fingers. In spite of the fact that a larger number of muscles were involved in the two finger movement than in the single finger movement, the late BP was larger in the latter, indicating the importance of discreteness of the movement. In this case, the amplitude difference of late BP was seen only over the central region contralateral to the movement, suggesting the important role of M1 (see Section 4). This finding is consistent with the clinical notion that a fundamental symptom of the pyramidal tract lesion is impairment of fine finger movement. In addition to the factor of discreteness, active inhibition of one finger while moving the other finger might add cortical activity related to active motor inhibition (Section 6). Another possibility is greater complexity of the isolated middle finger movement as compared with the simultaneous movement of two fingers. This is unlikely, however, because the amplitude difference was not seen over the midline central region where a complex movement would be expected to generate a larger amplitude of BP due to greater activation of SMA as compared to simple movement. The issue of complexity will be discussed further in the next paragraph.

As for the complexity of the movement, Benecke et al. (1985) found larger BP before the sequential or simultaneous performance of isotonic elbow flexion and isometric finger flexion than before either the isotonic elbow flexion or the isometric finger flexion alone. Simonetta et al. (1991) compared a simple movement with a motor sequence starting with the simple movement, and found earlier onset and larger amplitude of BP in the sequential movement than in the simple movement. In these two experiments, however, a larger number of muscles were involved in the complex movement than in the simple movement. In contrast, Kitamura et al. (1993b) compared the middle followed by index finger movement as a complex movement with the simultaneous movement of middle and index fingers as a simple movement, so that the muscles involved in the two conditions were matched. Furthermore, the subjects were trained so that the duration of EMG discharge was the same in the two conditions. As the result, they found larger amplitude of late BP, not of early BP, in the sequential movement as compared with the simultaneous movement. Since this amplitude difference was seen over the midline vertex as well as bilateral central regions, it was postulated that not only SMA but also bilateral sensorimotor cortices might play an important role in the preparation of the complex movement. This electrophysiological finding was later supported by the cerebral blood flow activation study with position emission tomography (PET) (Shibasaki et al., 1993). This observation suggests that, whenever BP is employed for studying motor control mechanisms or its abnormality in pathological conditions, the effect of task complexity or subjective difficulty to the subjects has to be taken into consideration as an important influencing factor.

### Table 2
Differential influence of various factors on early and late BP in normal and pathological conditions

<table>
<thead>
<tr>
<th>Factors</th>
<th>Early BP</th>
<th>Late BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of intention</td>
<td>Larger*</td>
<td></td>
</tr>
<tr>
<td>Preparatory state</td>
<td>Earlier onset*</td>
<td></td>
</tr>
<tr>
<td>Movement selection</td>
<td>Larger</td>
<td>No effect</td>
</tr>
<tr>
<td>Learning</td>
<td>Larger during learning*</td>
<td></td>
</tr>
<tr>
<td>Praxis movement</td>
<td>Start parietally*</td>
<td></td>
</tr>
<tr>
<td>Force</td>
<td>Larger*</td>
<td></td>
</tr>
<tr>
<td>Speed</td>
<td>Later onset*</td>
<td></td>
</tr>
<tr>
<td>Precision</td>
<td>No effect</td>
<td>Larger</td>
</tr>
<tr>
<td>Discreteness</td>
<td>No effect</td>
<td>Larger</td>
</tr>
<tr>
<td>Complexity</td>
<td>No effect</td>
<td>Larger</td>
</tr>
<tr>
<td>Parkinsonism</td>
<td>Small</td>
<td>No change</td>
</tr>
<tr>
<td>Cerebellar lesion</td>
<td>Small</td>
<td>Small</td>
</tr>
<tr>
<td>Dystonia</td>
<td>No change</td>
<td>Small</td>
</tr>
<tr>
<td>Hemiparesis recovery</td>
<td>No change</td>
<td>Involved</td>
</tr>
<tr>
<td>Mirror movement</td>
<td>No change</td>
<td>Involved</td>
</tr>
</tbody>
</table>

As for the factors in normal conditions, the effect is shown in a comparative form; the greater the factors, the larger or smaller the BP.

* Late BP not clearly distinguished.
So far, most studies of MRCPs have employed simple motor tasks such as finger extension, wrist extension, fist clenching, elbow movements, foot extension and tongue protrusion, which are quite different from the practical movements performed under more natural conditions. Wheaton et al. (2005a) recently studied movements employed in daily life, or the so-called praxis movements, by recording MRCPs associated with pantomiming of common gestures or tool use in normal right-handed subjects. Under these conditions, BP was found to start at the parietal region, larger over the left, and then followed by the BP over the midline frontal region and bilateral central regions (Fig. 2). The initial activation of the left parietal cortex during preparation for praxis movements in the right-handed subjects is consistent with the clinical notion that ideomotor apraxia is caused by the lesion involving the left parietal lobe; in particular, the supramarginal gyrus and its projection fibers to the frontal lobe.

4. Generator sources of MRCP

Various dipole source localization techniques have been applied to estimate the generator sources of MRCPs (Deecke and Kornhuber, 2003). In the case of hand movements, SMA and lateral precentral gyrus, both bilaterally, were estimated to be the main generator sources for early BP. Praamstra et al. (1996) estimated three dipole sources for explaining the early BP; one in the SMA and two others in bilateral M1. They further showed that only the current source identified in the SMA was affected by the mode of movement selection; larger before freely selected than fixed movement. Cui and Deecke (1999), based on the high-resolution DC-EEG analysis, demonstrated that BP occurs earliest in the medial wall motor areas (SMA and cingulate motor areas), then in the contralateral M1, and lastly in the ipsilateral M1. Toma et al. (2002), by using principal component analysis and functional magnetic resonance imaging (fMRI)-constrained EEG dipole source analysis, determined the crown of the precentral gyrus bilaterally, specifically the hand area of area 6, as the main source of early BP, area 4 in addition to area 6 as the source of late BP, and area 3 as the source of fpMP or N+50. Most studies, including that of Toma et al., have localized the source of MP or N−10 in the M1 hand area.

Deecke et al. (1982) reported the first MEG correlates of BP. They attributed BP to two current dipoles located in the SMA and the contralateral M1. Nagamine et al. (1996), by using the whole head MEG system, reported that the scalp distribution of magnetic field corresponding temporally to EEG BP is different from that of EEG BP, most likely due to the fact that MEG picks up only the dipole sources tangentially oriented to the head surface while EEG records both the radially and tangentially oriented dipole sources. The slow pre-movement shift on MEG (RF) begins much later than that of EEG and is distributed almost exclusively over the contralateral central region (Fig. 3). This finding can be explained by postulating that, at least as far as the lateral hemispheric convexity is concerned, early BP arises in the lateral premotor cortices (area 6) bilaterally, which is recorded by EEG but not by MEG because of its radial orientation, and late BP occurs in the contralateral M1 (area 4), which is recorded not only by EEG but also by MEG due to its tangential orientation (Nagamine et al., 1996). Furthermore, MEG
has some disadvantage in recording the activities arising from SMA especially when SMA is bilaterally activated, even though the dipoles are supposed to be tangentially oriented. However, Erdler et al. (2000), by using a whole head MEG system, clearly demonstrated that the SMA could contribute to the origin of the early BP. In particular, the SMA participation was shown to be, although clearly bilateral, contralaterally predominant, thus giving some solution to the cancellation problem (Lang et al., 1991; Erdler et al., 2000; Deecke and Kornhuber, 2003).

As for the electrical fields, it is not certain how much activity arising from SMA can be actually recorded from the scalp electrodes. There seems to be anatomical variability of SMA among normal subjects; in some subjects the SMA extends over to even the crown of the superior frontal gyrus. Lim et al. (1994), in an electrical stimulation study, observed tonic motor phenomena similar to the SMA motor symptoms by stimulating the lateral convexity of the superior frontal gyrus. Under this condition, it is expected that the radially oriented dipole can be easily recorded over the midline by EEG. Thus, in regard to the early BP, the midline maximal and symmetrical distribution is mainly due to the summation of electrical fields arising from bilateral premotor cortices over the lateral frontal convexity as the result of volume conduction, and partly due to the contribution of electrical fields arising from bilateral SMA at least for the midline component, although to different degrees among subjects.

MEG has special advantage in recording the components immediately preceding and following the movement onset, because both activities are tangentially oriented; one in the anterior bank and the other in the posterior bank of the central sulcus. Gerloff et al. (1998a,b) employed the steady state motor paradigm in which the subject repeated finger movement once every 500 ms paced by auditory cue. They identified the component corresponding to MP or N/C0 in M1 and the peak immediately after the movement onset in the postcentral gyrus. This technique is useful, along with an fMRI study, for the non-invasive study of somatotopic organization in M1.

Ikeda and colleagues have collaborated with Luders and his group in recording MRCP from chronically implanted subdural electrodes in patients with medically intractable partial epilepsy as a part of presurgical evaluation (Ikeda and Shibasaki, 2003). From extensive studies, it is the current consensus that early BP starts first in the SMA including pre-SMA and SMA proper, and then shortly thereafter in the lateral premotor cortices bilaterally, and about 400 ms prior to the movement onset the late BP (NS') starts in the M1 and premotor cortex mainly contralaterally (Ikeda et al., 1992; Yazawa et al., 2000). In M1, late BP culminates in MP (N/C0) just before the movement onset. According to stimulation studies which are carried out by delivering 50 Hz electrical pulses for 5 s in order to identify the cortical function beneath the electrodes, there is a somatotopic organization within the SMA proper, with the face area located rostrally and the leg area caudally, although not as discrete as in the M1 (Lim et al., 1994). Early BP is bilaterally generated from the localized area of the SMA proper following its somatotopic organization. Late BP is well localized to the part of M1 and lateral premotor cortex clearly following the somatotopic distribution as determined by the stimulation study. MP (N−10) is generated from a small area of M1 precisely corresponding to the somatotopic organization (Neshige et al., 1988). The generator sources of post-movement components have

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**Fig. 3.** Representative waveforms of MRCP and movement-related magnetic field (MRMF) in association with self-paced right finger movements in a right-handed normal subject. Early BP starts bilaterally about 3 s before the movement onset, and late BP (NS') is seen over the contralateral hemisphere starting about 500 ms before the movement onset. On MEG, a sharp slope is seen only over the contralateral hemisphere starting about 800 ms before the movement onset (By courtesy of Dr. Takashi Nagamine).
not been clearly identified, partly because it is difficult to correlate the scalp-recorded peaks to the peaks identified by epicortical recording (Neshige et al., 1988).

Pre-SMA in humans is defined anatomically as the area just rostral to the vertical anterior commissural (VAC) line and functionally as the area around the VAC line where electrical stimulation causes inhibition of ongoing or repetitive movements, the so-called supplementary negative motor area (SNMA) as described by Luders et al. (1995). In the pre-SMA thus defined, no somatotopic organization is recognized based on either the stimulation effect or the distribution of early BP (Yazawa et al., 2000). Furthermore, pre-SMA is sensitive to the rate of movement repetition; the late part of BP is seen in the pre-SMA only with slow rate of movement (0.2 Hz) whereas in M1 it is seen irrespective of the movement rate (as fast as 2 Hz) (Kunieda et al., 2000). The SMA proper is in between the pre-SMA and M1 in terms of the effect of movement rate. Often in the SNMA, a surface-positive BP is recorded regardless of the site of movement. Similarly, a surface-positive BP is recorded, again irrespective of the site of movement, over a part of the lateral precentral area which is defined as the primary negative motor area (PNMA) by Luders et al. (1995) (Kunieda et al., 2004). This area is usually located just rostral to the face or hand area of M1. The reason as to why BP is surface-positive in these negative motor areas has not been clarified.

Automatic movements such as eyelid blinks, spontaneous eye movements, chewing, swallowing and respiration are also controlled by volitional factors to a certain degree. Thus, BP is recorded when these movements are repeated at a self-paced rate. However, it is not known whether these movements when occurring under automatic and more natural conditions are associated with BP or not. Since most of these movements involve contraction of cranial muscles or ocular movements, recording of BP in association with these movements is faced with contamination by conspicuous artifacts such as electrooculogram (EOG), glossokinetic potential and respiration-associated potential (R-wave) (Deecke and Kornhuber, 2003). As first reported by Becker et al. (1971), self-paced ocular saccades are preceded by a slow surface-negative potential. By epicortical recording, Sakamoto et al. (1991) showed a localized BP in the SMA and the lateral frontal convexity before self-initiated saccade. Likewise, showed that BP first starts in the supplementary eye field which is located at the rostral edge of the SMA proper or between the SMA proper and the pre-SMA on the mesial frontal cortex, and then BP is seen in the frontal eye field, which is usually identified just rostral to the face or hand motor area, in close proximity to the PNMA as described above (Fig. 4).

BP associated with self-initiated swallowing was studied by Huckabee et al. (2003). They found the main generator of swallowing BP in SMA, but not much in M1. By
Table 3
Generator sources of each component of movement-related cortical potentials (MRCP)

<table>
<thead>
<tr>
<th>Component</th>
<th>Generator sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early BP</td>
<td>Pre-SMA (bilateral)</td>
</tr>
<tr>
<td>Earliest</td>
<td>SMA proper (bilateral)*</td>
</tr>
<tr>
<td>Next earliest</td>
<td>Area 6 (bilateral)*</td>
</tr>
<tr>
<td>Late BP (NS)</td>
<td>Area 6 (mainly contralateral)*</td>
</tr>
<tr>
<td>MP (N−10)</td>
<td>Area 4 (contralateral)*</td>
</tr>
<tr>
<td>fpMP (N+50)</td>
<td>Area 3 (contralateral)*</td>
</tr>
</tbody>
</table>

* Somatotopically organized to some degree.
* Somatotopically organized precisely.

epicortical recording of BP associated with self-paced swallowing in comparison with tongue movement, Satow et al. (2004) showed the main source of early BP and late BP in the same areas of SMA and M1 as found in self-paced tongue movements.

Several investigators reported that BP could be recorded from subcortical structures such as thalamus and basal ganglia (Rektor et al., 2001, 2003; Paradiso et al., 2004). However, some of the reported data based on the depth recording show BP of exactly the same waveform, except for the opposite polarity, and of exactly the same time course as the scalp-recorded BP, suggesting possible volume conduction from the cortical sources. Even if there might be some BP-like activities in the deep structures, their physiological meaning still remains to be elucidated.

The current consensus on the generator source of each MRCP component is summarized in Table 3. In self-paced repetition of simple movements at slow rate, the early slope (early BP) begins about 2 s before the movement onset in the pre-SMA with no site-specificity and in the SMA proper according to the somatotopic organization, and shortly thereafter in the lateral premotor cortex bilaterally again with relatively clear somatotopy. About 400 ms before the movement onset, another steeper negative slope (late BP or NS) occurs in the contralateral M1 and lateral premotor cortex with precise somatotopy. This sequence of activation within motor areas was demonstrated also by event-related fMRI (Cunnington et al., 2002, 2003), although not with the precise timing of EEG. For the praxis movement, parietal cortex generates BP in the early stage of preparation (Wheaton et al., 2005a).

5. Movement-related desynchronization/synchronization in relation to BP

Pfurtscheller and his group pioneered the study of the power changes of EEG oscillatory activity of various frequency bands associated with various tasks including voluntary movement. They showed that a power decrease in alpha or beta bands time-locked to an event or a task, event-related desynchronization (ERD), represents increased activation of the corresponding cortical area while a power increase, event-related synchronization (ERS), is associated with return to the resting condition or even decreased activation (Pfurtscheller and Aranibar, 1977; Pfurtscheller and Lopes da Silva, 1999; Pfurtscheller and Neuper, 2003). As far as the scalp-recorded EEG is concerned, the evolution of ERD along with the self-paced hand movement is different from that of BP. As described above, BP starts bilaterally (early BP) and becomes larger over the contralateral central region toward the movement onset (late BP). In contrast, at least for the right-hand movement in the right-handed subjects (see below), ERD starts over the left hemisphere and then spreads bilaterally (Babiloni et al., 1999). From these findings, it is postulated that movement-related electrical fields and ERD are generated through different neuronal mechanisms.

Recently Bai et al. (2005) showed different evolutions of ERD depending on the side of hand movement. When right-handed subjects move the right-hand, ERD of beta band starts over the left hemisphere and becomes more bilateral toward and during the movement. By contrast, for left-hand movement of right-handed subjects, the ERD starts bilaterally and remains bilateral throughout (Fig. 5). This finding is in conformity with the clinical notion that, for the right-handed subjects, the movement of the left-hand is associated with bilateral activation of the lateral premotor cortex while the movement of the right-hand is associated with exclusive activation of the left premotor cortex (Heilman et al., 2000).

It is important to note that ERD behaves differently depending on the frequency band. In self-initiated hand movements, ERD around 10 Hz starts about 2.0 s before the movement onset bilaterally at the sensorimotor areas, and 20 Hz ERD appears later and is localized more anteriorly with contralateral predominance (Nagamine et al., 1996). This finding is in agreement with the notion that alpha activity is predominant in the sensory cortex while beta activity in the motor cortex. After the termination of movement, a rebound of power or ERS occurs for the beta and/or gamma band. According to the analysis of ERD/ERS based on the epicortically recorded data, ERD for beta frequency band starts in the SMA bilaterally as early as 4 s before the movement onset, and then the precentral gyrus (the crown corresponding to area 6) shows ERD for alpha and beta frequency bands, again bilaterally with contralateral predominance (Fig. 6) (Ohara et al., 2000a). By strong contrast, the post-motion ERS for high frequency band is seen exclusively in the contralateral postcentral gyrus (Ohara et al., 2000a) (Fig. 6).

Given that the temporospatial pattern of the ERD prior to movement is different from that of the BP, it is clear that these two signals are providing different information. The explanation of the difference has been obscure, however. Recently Bai et al. (2006) have studied both the BP and ERD in patients with primary lateral sclerosis, where the primary pathology is in the large corticospinal pyramidal neurons. The BP was markedly diminished while the ERD was normal. Since the BP is likely mostly generated by postsynaptic potentials on the pyramidal cell apical dendrites,
it is not surprising that it is diminished. The normal ERD suggests that its cells of origin differ and might be the smaller neurons involved in cortico-thalamic oscillations. Functional coupling among different cortical areas can be studied by calculating the coherence of oscillatory activities in specific frequency bands among those areas.

Fig. 5. Scalp distribution of event-related desynchronization (ERD) of beta frequency bands in association with self-paced sequential movements of the right and left-hand across 9 right-handed normal subjects. In the right-hand movement, ERD starts over the left central region about 1.5 s before the movement onset and becomes bilateral shortly before and during the movement. In the left-hand movement, ERD is seen bilaterally from about 1.5 s before the movement onset and increases toward the movement onset [cited from Bai et al. (2005) with permission].

Fig. 6. Movement-related power change of electrocorticogram for various frequency bands, simultaneously recorded from the SMA proper, M1 hand area and SI hand area which were determined by electrical stimulation study in a patient with medically intractable partial epilepsy. The data for the contralateral hand movement are shown in thick black line, and those for the ipsilateral hand movement are shown in thin grey line. ERD for beta frequency band starts in the SMA proper about 3.5 s before the movement onset bilaterally. About 2 s prior to the movement onset, ERD for high alpha and beta frequency bands is seen in the M1 hand area bilaterally, more contralaterally, followed by ERD in the SI hand area. After the movement, increase of gamma frequency band (ERS) is seen exclusively in the contralateral SI hand area [cited from Ohara et al. (2000a) with permission].
Toward the movement onset, coherence between the SMA and the contralateral sensorimotor cortex as well as between the left and right sensorimotor areas increases (Gerloff et al., 1998c). A coherence study based on the epicortically recorded EEG data showed an increase in coherence for alpha frequency band toward the movement onset in the M1 hand area and the SI hand area each with respect to the hand area of SMA proper (Ohara et al., 2001). Coherence studies are difficult, in part because of possible artifacts from volume conduction. This can be overcome to some degree using partial coherence, where sources of volume conducted activity can be subtracted (Mima et al., 2000). For the praxis movements, by coherence analysis of EEG beta band (18–22 Hz), Wheaton et al. (2005b) showed increased functional coupling between parietal and premotor areas during preparation and execution of both transitive and intransitive movements. Using event-related fMRI, Fridman et al. (2006) explored activity during instructed-delay transitive and intransitive hand gestures. The comparison between planning–preparation and execution of gestures demonstrated a temporal rostral to caudal gradient of activation in the ventral premotor cortex and inferior to superior gradient of activation in the posterior parietal cortex.

Coherence between cortical oscillatory activities arising from M1 and EMG discharges associated with the sustained muscle contraction can be calculated as an index of motor cortical drive of the corresponding peripheral muscles. During hand muscle contraction, the EEG–EMG or cortico-muscular coherence is usually seen at around 20 Hz. Coherence is also seen at the same frequency between the left and right sensorimotor areas increases (Gerloff et al., 1998c). A coherence study based on the epicortically recorded EEG data showed an increase in coherence for alpha frequency band toward the movement onset in the M1 hand area and the SI hand area each with respect to the hand area of SMA proper (Ohara et al., 2001). Coherence studies are difficult, in part because of possible artifacts from volume conduction. This can be overcome to some degree using partial coherence, where sources of volume conducted activity can be subtracted (Mima et al., 2000). For the praxis movements, by coherence analysis of EEG beta band (18–22 Hz), Wheaton et al. (2005b) showed increased functional coupling between parietal and premotor areas during preparation and execution of both transitive and intransitive movements. Using event-related fMRI, Fridman et al. (2006) explored activity during instructed-delay transitive and intransitive hand gestures. The comparison between planning–preparation and execution of gestures demonstrated a temporal rostral to caudal gradient of activation in the ventral premotor cortex and inferior to superior gradient of activation in the posterior parietal cortex.

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The relationship of epicortical distribution of BP, ERD/ERS, cortico-cortical coherence and EEG–EMG coherence with that of functional cortical areas determined by stimulation studies is important from a practical point of view. It is especially true for presurgical study of patients in which the precise somatotopy within the motor cortex has to be determined. Generally speaking, BP, especially late BP, is relatively well localized, sometimes localized to even a smaller area than the areas determined by electrical cortical stimulation. In contrast, ERD, cortico-cortical coherence, and EEG–EMG coherence tend to be distributed in a wider area as compared with the motor areas as determined by electrical stimulation (Ohara et al., 2000b).

In summary, BP and ERD show different topographical evolution over the course of pre-movement period, suggesting different neuronal mechanisms between generation of the two phenomena. Both show somatotopic organization, but as far as electrocorticographic (ECoG) data are concerned, BP is localized to a smaller area than ERD. Since it is possible to analyze ERD on a single trial basis, it is being tested for utility for a brain–computer interface. Furthermore, coherence of rhythmic oscillations of various frequency bands among different cortical areas is a useful tool to analyze inter-areal functional coupling.

6. BP preceding voluntary motor inhibition

Studies of patients with epileptic negative myoclonus suggest the existence of a negative motor area in the sensorimotor cortex. Involvement of that area by epileptogenic discharges causes epileptic negative myoclonus associated with the EMG silent period in the contralateral limb in accordance with the somatotopy (Noachtar et al., 1997). Single electrical shock given to some parts of sensorimotor cortex elicits a pure silent period in the EMG without any preceding motor evoked potential (MEP) (Ikeda et al., 2000). Thus, the question arises as to whether voluntary muscle relaxation or voluntary termination of muscle contraction is associated with BP-like activity just like the case of voluntary muscle contraction. Terada et al. (1995a) trained normal subjects to be able to abruptly terminate the contraction of wrist extensor muscles without contracting any other muscles including the antagonist muscle, the wrist flexor in this case, and used the beginning of muscle relaxation as a fiducial point for back averaging the simultaneously recorded EEG. As the result, they found surface-negative slow shifts before the voluntary muscle relaxation, which were similar to BP associated with muscle contraction. Similar findings were reported regarding voluntary relaxation of foot extensor muscles (Terada et al., 1999). Rothwell et al. (1998) studied BP associated with isometric muscle relaxation, and found smaller BP over the lateral central region in the relaxation than in the contraction while they found equal activity on the midline frontal region in the two tasks. They interpreted this finding by postulating that the motor cortex activity related to the onset task is lacking in the offset task.

The findings of the muscle relaxation BP by Terada et al. (1995a, 1999) were supported by fMRI study (Toma et al., 1999) as well as by ERD/ERS based on MEG recording (Toma et al., 2000). The fMRI study of voluntary muscle relaxation showed activation in the pre-SMA, SMA proper and the contralateral sensorimotor cortex just like in case of muscle contraction. The activation of the mesial frontal region (pre-SMA and SMA proper) was greater in the muscle relaxation task than in the muscle contraction. Thus, it is postulated that activation of some of the pyramidal tract neurons in SMA, lateral premotor cortex and M1 causes the EMG silent period probably via the inhibitory interneurons at the spinal cord level.

7. BP in movement disorders

In view of the fact that at least some part of the scalp-recorded BP originates from SMA which receives main dopaminergic input from the basal ganglia via thalamus, several investigators have studied BP in patients with Parkinson’s disease (PD). It has been reported that early BP is smaller in PD than in controls whereas late BP is not different (Dick et al., 1989). Furthermore, early BP was shown to be smaller in off-phase of DOPA medication and returned to normal in on-phase (Dick et al., 1989). In
contrast, the amplitude of late BP in patients with PD increased after pallidotomy (Gironell et al., 2002). However, when a single case is studied by MRCP, the size of early BP or late BP may not be very helpful for the diagnostic purposes or evaluation of the severity, because the magnitude of BP is significantly influenced by intention, motivation, effort, and various physical features of movement (see Section 3), thus giving rise to significant inter-trial as well as inter-individual variability.

Shibasaki et al. (1986) have been impressed by clear abnormality of BP in patients with cerebellar lesions; among others, the lesions involving the dentato-thalamic, cerebellar efferent pathway. BP is almost absent in patients with degenerative diseases of dentate nucleus such as progressive myoclonic ataxia and lipidosis. Ikeda et al. (1994) reported a patient with an infarction in the midbrain involving the cerebellar efferent pathway, who showed no BP but normal contingent negative variation (CNV), a similar slow negativity prior to movements that are triggered by the imperative sensory stimuli in a reaction time task rather than self-paced. BP is much smaller or even absent when the patients with cerebellar hemispheric lesions repeat self-paced movements of the hand ipsilateral to the cerebellar lesion (Kitamura et al., 1999). This is considered to be the results of deficiency of facilitatory input from cerebellum, which is known to project primarily to the lateral premotor cortex and M1. This is in strong contrast with CNV (see above). CNV is often diminished in PD but not affected in patients with cerebellar lesions (Ikeda et al., 1997).

Mirror movements are a good indication for the clinical application of BP. Some mirror movements are associated with bilateral distribution of late BP instead of the contralateral predominance with respect to the side of intended movement (Shibasaki and Nagae, 1984). Mayer et al. (1995) studied MRCP in six patients with congenital mirror movements. They found that the scalp distribution was different from that of normal subjects only for the period from 50 ms before to 50 ms after the EMG onset, and no difference was found in BP. In their record, however, the analysis window covered only 1.2 s before the movement onset, and thus late BP was not clearly distinguished from early BP. If mirror movements result from activation of unilateral motor cortex via uncrossed pyramidal tracts, then late BP is expected to be localized to the central region contralateral to the side of intended movement. Thus, bilateral distribution of late BP in intended unilateral movement indicates activation of bilateral motor cortices, probably due to loss of transcortical inhibition for the movement of the opposite (unintended) hand. This was supported by an fMRI study (Maegaki et al., 2002), but studies using transcranial magnetic stimulation (TMS) showed bilateral representation of hands in M1 with stronger ipsilateral corticospinal projection (Cohen et al., 1991; Maegaki et al., 2002; Ueki et al., 2005). The discrepant results between the MRCP or fMRI studies and the TMS studies might be partly due to the entirely different nature of movement studied; voluntary condition in the former and stimulus-induced condition in the latter. Furthermore, mirror movements may not be homogeneous depending on the etiologies (congenital vs. acquired), stage of illness, and age of patients.

BP recording has been also applied to the study of mechanisms underlying recovery of paretic hand in stroke patients. During the motor recovery in some patients with hemiparesis due to stroke, late BP preceding the movement of the paretic hand is larger over the central region ipsilateral than contralateral to the affected hand, suggesting that the motor cortex of the intact hemisphere is driving the movement of the paretic hand during motor recovery (Honda et al., 1997). This finding was also supported by fMRI and TMS studies. By applying integrated multimodal approach including EEG spectral analysis and PET blood flow activation study during movements, EEG coherence analysis, and TMS to patients who well recovered from chronic capsular stroke, Gerloff et al. (2006) showed that (i) effective recovery is based on enhanced utilization of ipsi- and contra-lesional resources, (ii) basic corticospinal commands arise from the lesioned hemisphere without recruitment of (‘latent’) uncrossed corticospinal tract fibers, and (iii) increased contralesional activity probably facilitates control of recovered motor function by operating at a higher-order processing level.

BP has been also applied to the study of focal dystonia, because functional abnormality of sensorimotor cortex has been proposed to play an important role in its pathogenesis. Deuschl et al. (1995) reported lower amplitude of late BP over the contralateral hemisphere when the patients with focal hand dystonia made simple motor task by the affected hand as compared with normal control subjects. Toro et al. (2000) found reduced ERD in the 20–30 Hz band in association with the movement of both affected and unaffected hands in patients with focal hand dystonia as compared with age-matched normal subjects, suggesting abnormal motor command in this condition. Based on the hypothesis of impaired inhibitory mechanism before and during the motor execution, Yazawa et al. (1999) studied BP associated with self-initiated muscle relaxation (see Section 6) in patients with writer’s cramp, and found reduced amplitude of BP associated with muscle relaxation of the affected hand over the contralateral hemisphere (Fig. 7). Muscle contraction which was studied as a control task showed no difference in BP amplitude over the contralateral hemisphere, but it showed relatively increased amplitude over the ipsilateral hemisphere in the patient group (Fig. 7). Assuming that the muscle contraction task was subjectively more difficult for the patients to execute than for the normal subjects, it might have activated bilateral sensorimotor cortices, as has been discussed in relation to the effect of the movement complexity (see Section 3). In fact, most patients with focal hand dystonia who were recruited for this study confessed that they found it more difficult to contract than to relax the muscle. Oga et al. (2002) applied fMRI to this voluntary motor inhibition study in patients with writer’s
cramp, and found smaller area of activation both in the SMA proper and the contralateral sensorimotor cortex as compared to normal subjects. In contrast to the BP study, this abnormality of BOLD effect was found not only for muscle relaxation but also for muscle contraction. These findings suggest functional abnormality of motor cortices not only for voluntary positive movement but also for voluntary motor inhibition in focal dystonia.

One of the most useful clinical application of BP is its use for detecting the participation of the voluntary motor system in the generation of ‘psychogenic involuntary movements’. Here the technique is based on the principle of jerk-locked back averaging, the technique which was initially developed to study cortical myoclonus (Shibasaki and Kuroiwa, 1975). While psychogenic movements look voluntary, patients say they are involuntary. This is believed to be the case for most such patients who have a conversion disorder, but is not the case for those patients with a factitious disorder or who are malingering. Obeso et al. (1981) studied patients with Tourette syndrome by using the EMG onset of tics as a fiducial point for back averaging EEG, but they did not find any activity before the tics, although they found normal BP before the movements mimicking their tics. Karp et al. (1996) observed an activity like late BP before simple tics in 2 out of 5 patients. Terada et al. (1995b) applied this principle to patients with clinical diagnosis of psychogenic myoclonus, and found a BP-like slow EEG shift before the psychogenic myoclonus, which was similar to the BP before the mimicking movements. Since a typical complex of early BP and late BP is never recorded before organic involuntary movements, the demonstration of early BP and late BP before the movement in question strongly suggests its psychogenic origin, and we use this routinely in clinical practice. However, the absence of BP does not always exclude movement generated by the voluntary motor system, because BP may fail to be recorded even in normal subjects.

Recently Sirigu et al. (2004) recorded BP in patients with selective lesions in the parietal cortex, and compared the results to those from patients with cerebellar lesions and normal subjects. Within each group, they further compared the task when the subjects were paying attention to the time of button press (M-judgment) and the task when the subjects were paying attention to the time of becoming aware of the intention to move (W-judgment). Behaviorally, there was no difference in the M-judgment among three groups, but in the patients with parietal lesions, the time of W-judgment was significantly delayed as compared with two other groups. Furthermore, they reported that, in the parietal group, the BP in the W-judgment was poorly formed while it was normal in the M-judgment. As admitted by these authors themselves, however, the waveforms
of BP presented in their paper appear to contain artifacts, indicating how difficult it is to obtain satisfactory waveforms especially in patients with neurological diseases.

8. Physiological implication of BP

The pre-movement slow negativity is clearly distinguishable into at least two components; early BP and late BP (NS'). It is certain that both components are related to preparation and/or execution of voluntary movement, because neither of them is associated with involuntary movements although rare exceptional cases have been reported (see below). As has been discussed in the section on the factors influencing BP (Table 2), early BP and late BP are differentially influenced by various factors. Early BP is influenced by cognitive functions such as level of intention, preparatory state and movement selection whereas late BP is influenced by features of the movement itself such as precision, discreteness and complexity. In pathological conditions, early BP is abnormal in PD, while late BP is abnormal in hemiparesis, dystonia and mirror movement. Both are abnormal in cerebellar lesions. This is consistent with the generator sources of each component; pre-SMA, SMA proper and lateral premotor area (area 6) bilaterally for early BP and contralateral precentral cortex including area 6 and area 4 for late BP. However, the precise relationship of each component to the intention to move is not understood.

If the timing of awareness of the conscious will to move occurs much later than the BP onset as suggested by Libet et al. (1983) and subsequent investigators, then a big question arises as to what the physiological significance of BP might be (Klein, 2002; Eagleman, 2004). Deecke and Kornhuber (2003) in their review article pointed out the complexity of interpretation of the Libet's experiment and gave a general explanation. First, there is a general decision to move periodically in the beginning of the whole experiment. Second, it is then possible that the decision for each individual movement is made subconsciously and that consciousness is “switched on” about 200 ms before movement onset. Third, this 200 ms interval gives time for consciousness to veto the movement.

In relation to this discussion, Oga et al. (2000) made an interesting observation on EEG in a patient with subacute sclerosing panencephalitis (SSPE). The patient showed typical periodic synchronous discharges (PSDs) on EEG which were time-locked to periodic dystonic movements of extremities and trunk occurring once every 6–7 s. Between the successive periodic phenomena, a slowly rising, surface-negative shift was observed when EEG was recorded with long time constant (Fig. 8). The slow negativity was maximal at the parietal midline electrode and was accompanied by low amplitude surface-positivity at anterior electrodes. Furthermore, the periodicity of EEG and abnormal movements was disturbed by either various stimulation nor volitional movements, but it was clearly disturbed when the patient was requested to make a motor task in response to external stimulus (simple reaction time paradigm). Moreover, when the stimulus was presented within 1.7 s after the spontaneous PSD or dystonic movements, the patient responded to the stimulus by motor task with normal reaction time. However, if the stimulus was given 1.7 s or longer after the spontaneous phenomena, the stimulus elicited the EEG discharge as well as the dystonic movements which were exactly the same as the spontaneous ones, causing a significant delay in reaction time. Taken together with the slow surface-negative EEG shift rising toward the next involuntary phenomenon, it is postulated that, in this particular patient with SSPE, the spontaneous discharges occur when the cortical excitability level reaches a certain threshold causing the periodicity. Furthermore, if the subject is requested to respond to a stimulus by motor task (sensorimotor integration) after the cortical excitability rises to a certain level, then it suddenly increases the cortical excitability level up to the threshold, causing the premature occurrence of PSD and dystonic movements. Since these periodic events are undoubtedly involuntary phenomena, this is perhaps the only organic involuntary condition in which a slow, surface-negative shift has been clearly recorded.

Assuming that the slow surface-negativity arises from increasing depolarization of the superficial layer of the apical dendrites of pyramidal neurons, this might reflect subconscious readiness for the forthcoming movement. Taken together with the finding of Libet’s paradigm, it is postulated that the early BP might reflect subconscious readiness for the forthcoming movement while the late BP might be related to the conscious will to move. In this regard, the study of the time relationship between the late BP and conscious will to move is warranted.


