Rapid Communication

Is the movement-evoked potential mandatory for movement execution? A high-resolution EEG study in a deafferented patient

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During simple self-paced index finger flexion with and without visual feedback of the finger, we compared the movement-evoked potentials of the completely deafferented patient GL with those of 7 age-matched healthy subjects. EEG was recorded from 58 scalp positions, together with the electromyogram (EMG) from the first dorsal interosseous muscle and the movement trace. We analyzed the movement parameters and the contralateral movement-evoked potential and its source. The patient performed the voluntary movements almost as well as the controls in spite of her lack of sensory information from the periphery. In contrast, the movement-evoked potential was observed only in the controls and not in the patient. These findings clearly demonstrate that the movement-evoked potential reflects cutaneous and proprioceptive feedback from the moving part of the body. They also indicate that in absence of sensory peripheral input the motor control switches from an internal "sensory feedback-driven" to a "feedforward" mode. The role of the sensory feedback in updating the internal models and of the movement-evoked potential as a possible cortical correlate of motor awareness is discussed.
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Introduction

There is an increasing body of experimental evidence, coming from various approaches, favoring the hypothesis that the cortical activity between 90 and 120 ms after movement onset reflects sensory feedback from the moving part of the body. After the movement onset, an increase in neuronal activity in somatosensory cortex has been recorded in monkeys (Evarts and Fromm,

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1977; Wannier et al., 1991) and a negative peak called the "hand somatosensory potential" reported in humans (Neshige et al., 1988). This activity, manifested as movement-evoked potential (MEP), was considered by most of the electrical macropotential studies, including the pioneering work of Kornhuber and Deecke (1965) who introduced the Bereitschaftspotential paradigm (BP) as a typical paradigm to study voluntary movement-related activity, as reflecting proprioceptive input from the periphery (Kristeva et al., 1979; Tarkka and Hallett, 1991; Kornhuber and Deecke, 1965). These EEG studies provided consistent evidence that the topographic distribution of the MEP is characterized by a bipolar pattern with a maximum of negativity over the fronto-central midline and a positivity above the contralateral parietal areas. This pattern is caused by the activation of a network in the contralateral somatosensory area as shown by electric and magnetic source reconstruction (Toro et al., 1993; Ball et al., 1999; Toma et al., 2002; Toma and Hallett, 2003; Kristeva-Feige et al., 1994, 1995; Cheyne and Weinberg, 1989; Kristeva et al., 1991). Further arguments favoring the peripheral origin of the MEP came from its somatotopic organization (Walter et al., 1992) and from the finding that it had a longer latency for toe than for finger movements.

Cheyne et al. (1997) using MEG showed that cooling the subject's arm resulted in delays of about 8 ms in the MEP latency. This delay was attributed to an increase in conduction time in the afferent pathways as confirmed by electrically evoked somatosensory responses and thus, strongly suggested a peripheral origin for the movement-evoked field. In a previous study of ours (Kristeva-Feige et al., 1996), we demonstrated that elimination of cutaneous inputs by anesthetic nerve block modulated the MEP amplitude, suggesting that this potential reflects not only proprioceptive but also cutaneous input from the periphery. This finding was not surprising as Edin and Abbs (1991) and Edin (1992) undoubtedly showed that "cutaneous input from the moving part of the body also plays a specific role in motor control".

If the MEP reflects somatosensory input from the periphery, one can predict that this component will not be present in deafferented patients. For this purpose, we recorded the movement-related cortical activity and the movement kinematics in the deafferented patient GL and in seven healthy control subjects while they performed self-paced flexion movements of the index finger every 12–24 s (typical Bereitschaftspotential paradigm, Kornhuber and Deecke, 1965). To have a reasonable spatial resolution, we recorded the movement-related activity from 58 scalp positions. In addition, we performed advanced source reconstruction analysis for the deafferented patient and four controls. This analysis which takes into consideration the individual anatomy from the MRI data of the patient and the control subjects successfully defines the spatiotemporal pattern of activation of the motor areas during planning and execution of simple and complex movements (Ball et al., 1999; Kristeva-Feige, 2003).

The second aim of the study was to investigate the contribution made by the visual feedback to cortical activity and motor performance of this simple finger movement. For this purpose, movement-related activity and kinematics were investigated under two experimental conditions: with and without visual feedback of the moving finger.

The study demonstrated that the patient GL did not show any MEP although she performed the simple pulse movements almost as well as the control subjects. The results are discussed within the framework for optimal motor control (Wolpert and Ghahramani, 2000) and awareness of action.

Methods

Subjects

The deafferented patient GL, a right-handed 55-year-old woman, participated in the study (for detailed clinical description, cf. Forget and Lamarre (1987) and http://www.deafferented.apinc.org//). After two episodes of polyneuropathy (at the age of 27 and 31), the patient has been suffering from a strong sensory impairment of the whole body up to the nose due to affected large diameter peripheral sensory myelinated fibers. The impairment was documented by sural biopsy. The patient had a total loss of touch, vibration, pressure and kinesthetic senses and no tendon reflexes in the four limbs. Pain and temperature sensations are still present. The motor fibers are not affected and the patient can perform complex motor behavior under visual guidance.

Seven healthy right-handed females (mean age 54.5 ± 3.2 years) acted as age-matched control subjects. None of them had any history of neurological disease.

All subjects participated according to the declaration of Helsinki, with informed consent and the approval of the local ethics committee. The handedness of the patient and the controls was tested according to a modified Oldfield questionnaire (Oldfield, 1971). The subjects had no previous experience with similar experiments.

Experimental paradigm

During the experimental session, the subject sat in an electrically shielded, dimly lit room. The right hand and arm were supported in a rigid cast (Fig. 1A). Two experimental conditions were investigated in a given recording session in the following order:

• Self-paced movement without visual feedback (minus visual control—VC): The subjects performed a flexion of the right index finger. Movements were self-paced at irregular intervals between 12 and 24 s, starting from complete relaxation (Bereitschaftspotential paradigm). The index finger flexion was performed at the level of the metacarpophalangeal joint, without any flexion of the interphalangeal joints. The subjects were instructed to avoid any other movements and to fix their gaze on a green light emitting diode placed in front of them at
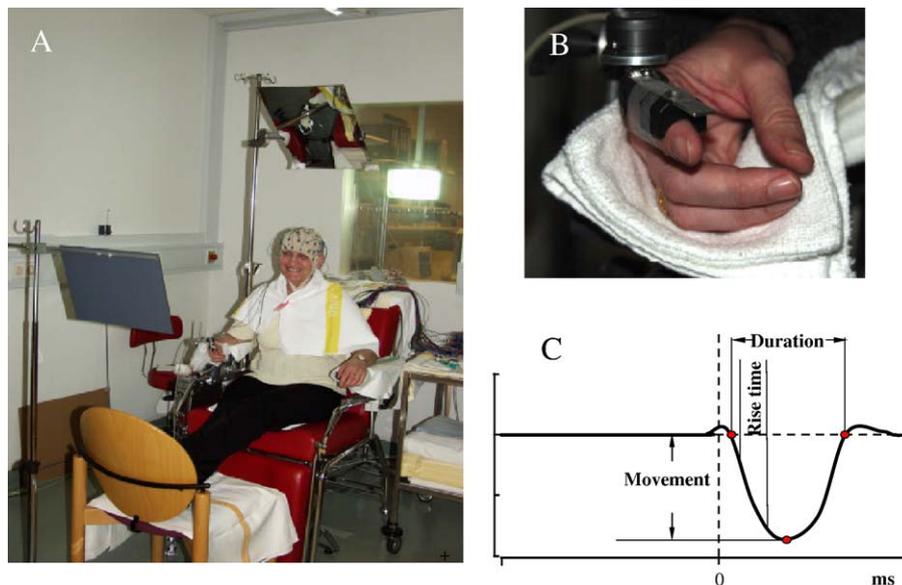

Fig. 1. Experimental set-up. (A) High-resolution EEG was recorded with an electrode cap from 58 scalp positions during movement execution. Two mirrors, one placed above and the other in front of the subject (here patient GL) provided a visual feedback of the movement. (B) The hand of one control subject with the goniometer recording the index finger kinematics. (C) Goniometric trajectory with the definition of the movement parameters (amplitude, duration and rise time).

the eye level. A wooden board was hiding the subject's finger. Thus, the subjects did not have any visual feedback of the moving finger.

- Self-paced movement under visual control (+VC): The motor task was the same as in the first condition but the subjects had a visual feedback of the movement provided by two mirrors (Fig. 1A), installed in such a way that the subjects saw their moving finger close to the fixation green diode.

Each subject was given several practice trials for both experimental conditions prior to the experiment. During the experimental session, the index finger flexion was repeated 90 times.

Recordings

EEG (bandpass 0–100 Hz; sampling rate 500 Hz) was recorded from 58 scalp positions, equally distributed over both hemispheres (NeuroScan, El Paso, TX, USA). This electrode layout was shown to be more appropriate for accurate source reconstruction. The electrooculogram (EOG) and the electromyogram (EMG) were recorded with surface electrodes (bandpass 0–200; sampling rate 1000 Hz). EMG was recorded using a tendon-belly montage from FDI muscle. Diagonal EOG was used to reject the trials contaminated with eye movements from further analysis. The EOG rejection was done semi-automatically. To specify the movement kinematic parameters, a goniometer was placed on the right index finger (Figs. 1A and B). EEG, EMG, EOG and goniometric signals (Fig. 1C) were stored and analyzed off-line.

After the EEG-recording, the electrode positions and the head contour of the subjects were digitized using a 3D ultra-sound localization device (ZEBRIS). The digitized head-contour was matched to the head-contour of the anatomical MRI using an automatic surface matching technique for registration of the co-ordinate systems of the two modalities (CURRY Software package, Neuroscan, El Paso, TX, USA).

Data analysis

Movement-related potential

Manual triggers were placed at the start of the movement, defined by the very beginning of the rectified EMG activity of the FDI, and the cortical DC-potentials were averaged time-locked to those triggers. Only trials where the movement started from a full relaxation were selected.

The analysis time was set from 3000 ms before to 2000 ms after the trigger. Artifact rejection was performed semi-automatically to exclude trials with eye movements and other muscle artifacts. Artifact-free trials were baseline-corrected using the time between 3000 ms and 2500 ms prior to the trigger. Fifty to seventy artifact-free trials were averaged per subject for each experimental condition. Averaging to the beginning of the goniometric signal did not change the result.

To describe the movement-evoked potential (MEP), we measured the peak latency after movement onset at the posterior parietal maximum over the contralateral sensorimotor area (CP3).

Motor performance

To define onset and end of the movements as well as kinematic parameters, the goniometric curves were analyzed for each subject trial-by-trial. Movement onset was defined as the point when the

goniometric curve departed from the baseline at the start of the trial. The end of the trial was set at the time point when the goniometric curve again reached the baseline.

To describe the motor performance, the following kinematic parameters were quantified: movement duration, movement amplitude in degrees and rise time (Fig. 1C). The duration of the movement was defined as the time between the start and the end points, and the rise time as the time between 10% and 90% of the maximal movement amplitude.

To investigate whether the patient's motor performance differed from that of healthy controls, we computed mean values and standard deviations of movement amplitude, rise time and duration for each subject and both conditions (+VC and −VC), as well as the variance of these parameters. A two-way ANOVA with a between factor group (patient, controls) and within factor condition (+VC and −VC) was applied.

Advanced source reconstruction

Data preprocessing

Electric source reconstruction for the patient and for four of the control subjects was performed on the basis of the individual brain anatomy obtained from MRI. This analysis used: (i) a realistic head model taking into account the shape and conductivity of the head compartments (skull, scalp and skin), (ii) distributed source models like cortical current density (CCD) not requiring a priori knowledge of the cortical sources and allowing for the reconstruction of simultaneously active multiple sources and (iii) source space model, in which the solutions are constrained to the segmented cortex.

For structural MRI, the 3D data set with full head coverage and 1 mm³ voxels was acquired using a volume-encoded magnetization prepared rapid acquisition gradient echo pulse sequence (MPRAGE) with TR 9.7 ms, TE 4 ms, TI 300 ms, flip angle 12°.

Image processing

Individual volumetric Magnetic Resonance Images (MRIs) (Fuchs et al., 2001; Wagner et al., 1995, 1997) were used for volume conductor modeling and visualization. The coregistration between digitized electrode coordinates and MRI was based on three landmarks (left preauricular point, right preauricular point, nasion) that were digitized together with the electrodes and also identified in the MRI volumes (Fuchs et al., 2002). Realistic Boundary Element Method (BEM) volume conductor models were set up and served as the forward model for source analysis. The triangle sizes used were 7, 9 and 10 mm for the inner skull, outer skull and the skin, respectively, yielding some 6000 to 7000 triangles per model.

EEG processing

The noise variances per channel were estimated based on the assumption that the 20% samples with the smallest mean global field power in the latency range from −3000 ms to +2000 ms can be regarded as noise and that the noise is of Gaussian type. Then, a Signal-to-Noise Ratio (SNR) transformation (Fuchs et al., 1998) was carried out in order to transform data units from μV to SNR. Thus, the contribution of the noisy channels is decreased. Next, a Principal Component Analysis (PCA) Golub and van Loan (1989) was carried out for the latency range of interest, from −200 ms to +400 ms. Due to the SNR transformation, the singular value of each individual component equals its SNR. Components with an SNR above 1.0 were selected and decomposed using an

Independent Component Analysis (ICA) (Makeig et al., 1996, 1999; Kastner et al., 2000) comprising the same time range.

Source analysis

Each individual independent component thus obtained was then subjected to source analysis, yielding a fixed source with an activation time course that equals the time course of the underlying ICA component. As the source model, a single equivalent current dipole (ECD) was used. In order to validate the appropriateness of the single ECD, two-dipole fits and sLORETA CDR analyses (Pascual-Marqui, 2002; Wagner et al., 2004) were carried out. As a result, a source model comprising several ECDs plus their respective activation time courses was obtained. These sources can typically be identified with components of brain activity or with artifacts (Makeig et al., 1999, 2002, 2004; Jung et al., 2001; Vigário et al., 2000).

Image segmentation, volume conductor modeling, PCA, ICA, source reconstruction and visualization were performed using the CURRY software (CURRY 5.0, Neuroscan, El Paso, TX, USA).

Results

Movement parameters with and without visual feedback

Representative examples of the goniometric trajectories with and without visual control are displayed in the lower part of Fig. 3, for the patient and four control subjects. The quantitative analysis of the trajectories is shown in Fig. 2 for the patient and all seven subjects.

The upper part of Fig. 2 shows mean amplitude duration and rise time values for each subject and the lower part shows their respective variance. It is obvious that the mean values obtained by the patient fit in the distribution of those of the control subjects, although they often are at the upper or lower border. The statistical analysis did not reveal any significant differences between patient GL and controls with respect to the mean values for movement duration, amplitude and rise time. No significant differences between the two conditions (−VC, +VC) were also found for the mean values. This was true for both patient and control subjects.

The biggest differences between patient and controls were in the variance values displayed in the lower part of Fig. 2, which shows that the patient values for both amplitude and duration are outstanding under −VC condition. For the amplitude variance, a two-way ANOVA could be performed with non-logarithmically transformed data, as the control values were normally distributed. This two-way ANOVA with a between factor group (patient, controls) and within factor condition (+VC and −VC) revealed a significant group ($F = 8.95$, $P = 0.024$) and condition effect ($F = 22.53$, $P = 0.01$), and a significant interaction between group and condition ($F = 12.37$, $P = 0.0126$). The amplitude variance was significantly higher in the patient GL in −VC condition (Fig. 2). The values of patient GL for the variance of the movement duration were at the upper border, i.e. most variable. As the control values were not normally distributed, no ANOVA could be performed for this parameter.

To quantify the change of performance over time (during the experimental session), the slope of the distribution of the individual values for each parameter was also computed. The two-way ANOVA revealed that in the −VC condition the patient GL became slower at the end of the experiment as compared to the healthy controls ($F = 6.99$, $P = 0.038$). The differences under the condition +VC were not significant.
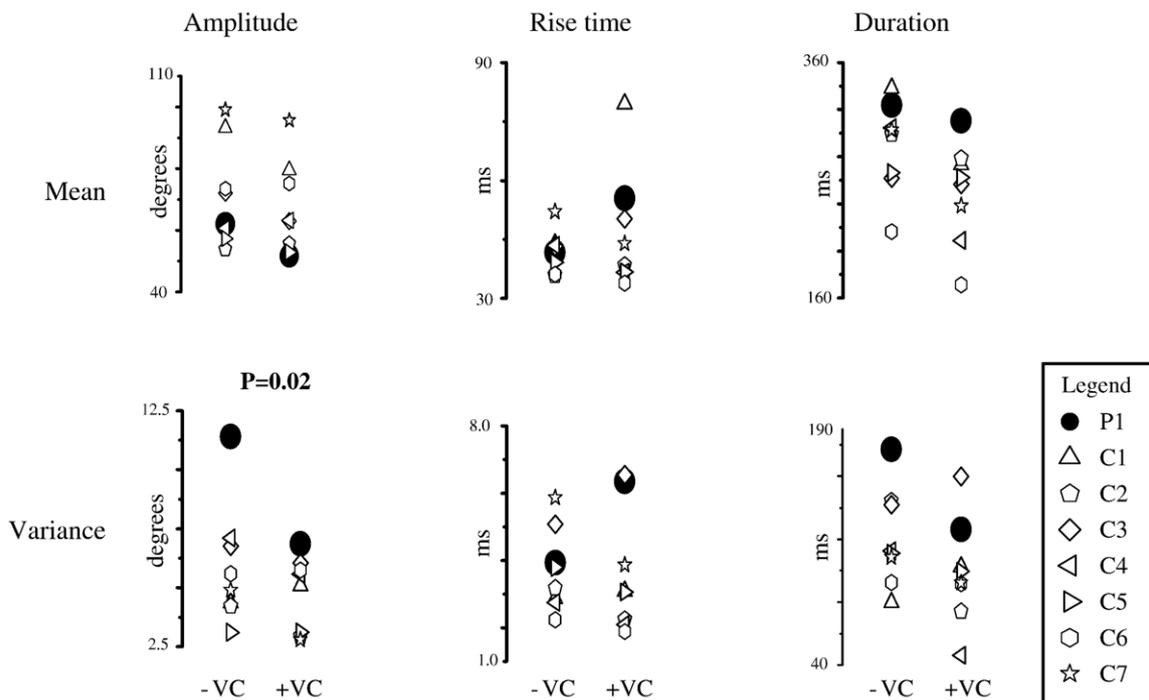

Fig. 2. Means (upper row) and variances (lower row) for the movement parameter values (amplitude, rise time and duration) under both experimental conditions (with and without visual control, +VC and −VC, respectively). Data for all controls (C1–C7) and for the patient GL (P1). Upper row: Note that the mean amplitude, rise time and duration of the patient GL are within the range of the controls' values. Lower row: Note the significantly higher variance of movement amplitude for the patient GL as compared to the controls in −VC condition ($P = 0.02$).

Movement-evoked potential (MEP)

Fig. 3 shows the parietal maximum of the MEP at CP3 electrode for the patient GL and for four of the controls underneath the respective goniometric trajectories for both +VC and −VC conditions.

Although all control subjects showed a large and well-defined MEP after movement onset (marked with a circle), no such MEP could be seen in the patient GL. The mean latency of the positive maximum at C3P in the controls was 93.7 ± 15.8 ms in the −VC and 92.0 ± 17.2 ms in the +VC condition. This difference was not significant (paired t test, $P = 0.5$). Although the Bereitschaftspotential in the patient GL had large amplitude, this amplitude was not outstanding as compared to those of the controls.

Source reconstruction

In the control subjects, independent component analysis (ICA) revealed two different components with characteristic activation time courses. The source analysis of the first component yielded a frontal medial source consistent with activation of the supplementary motor area (SMA). The component of interest for this study was the second ICA component displayed in Fig. 4A. In the controls, the source reconstruction revealed a lateral source consistent with an activation of the contralateral sensorimotor area. The orientation of this source at the movement onset was in the anterior direction and thus, consistent with prevailing motor cortex activation. The most striking feature of this source was the change of its orientation from anterior to posterior at about 30–40 ms after movement onset, indicating an activation of the primary somatosensory cortex. This reversal is consistent with our previous

results (Ball et al., 1999; Kristeva et al., 1991). The source reached its maximum strength at 100 ms after the movement onset. The cortical sources in +VC and −VC conditions did not show striking differences though a more complicated topographic pattern was observed under the +VC condition from the control subject (Fig. 4).

While all control subjects showed two strong ICA components (one medial corresponding to supplementary motor area activation, and one lateral corresponding to the contralateral sensorimotor area), the patient GL had only one significant ICA component with a source consistent with an activation in the depth of the precentral gyrus. This source was active before movement onset, had its maximum at movement onset and persisted during the execution of the movement (Fig. 4). No differences between conditions (+VC and −VC) were observed as the ICA time course, the topographic maps and the sources were nearly the same. This is true for both patient GL and the controls.

Discussion

Movement-evoked potential (MEP) reflects somatosensory input from the periphery

The basic hypothesis tested in this study was that the MEP, i.e. the cortical potential occurring at approximately 90 ms after a voluntary movement, would be absent from a deafferented patient suffering from a large-fiber sensory neuropathy. The present results confirmed this hypothesis as in contrast to the controls, the patient GL did not show any early postmovement MEP (Fig. 3). Thus, this finding provides clear evidence that MEP reflects sensory
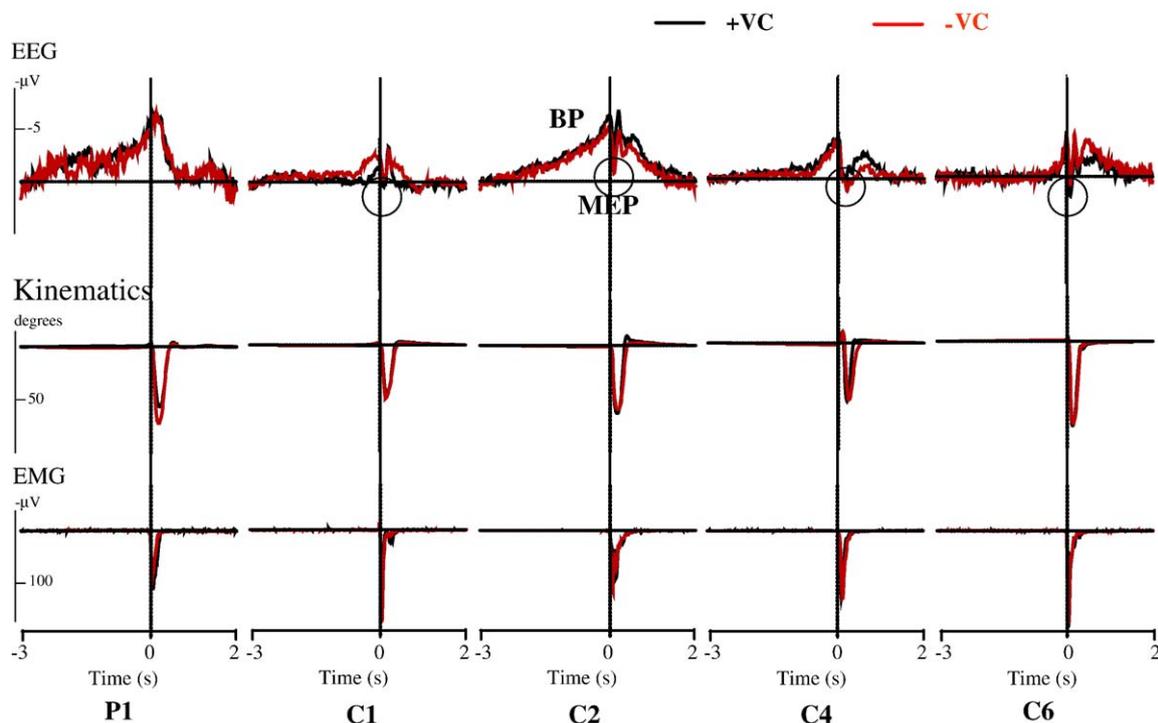

Fig. 3. Averaged movement-related cortical potentials at CP3 (i.e. at the parietal maximum of the MEP) preceding and during the self-paced index finger flexion with below the corresponding goniometric trajectory and EMG for the patient GL (P1) and for four controls (C1, C2, C4 and C6). Averages of 50 trials. Analysis time from 3 s before to 2 s after the movement onset. Black: +VC, red: −VC. BP (Bereitschaftspotential); MEP (movement-evoked potential). Note that the MEP (surrounded by a circle in the control subjects) is absent in the patient though she was performing the movement as well as the control subject.
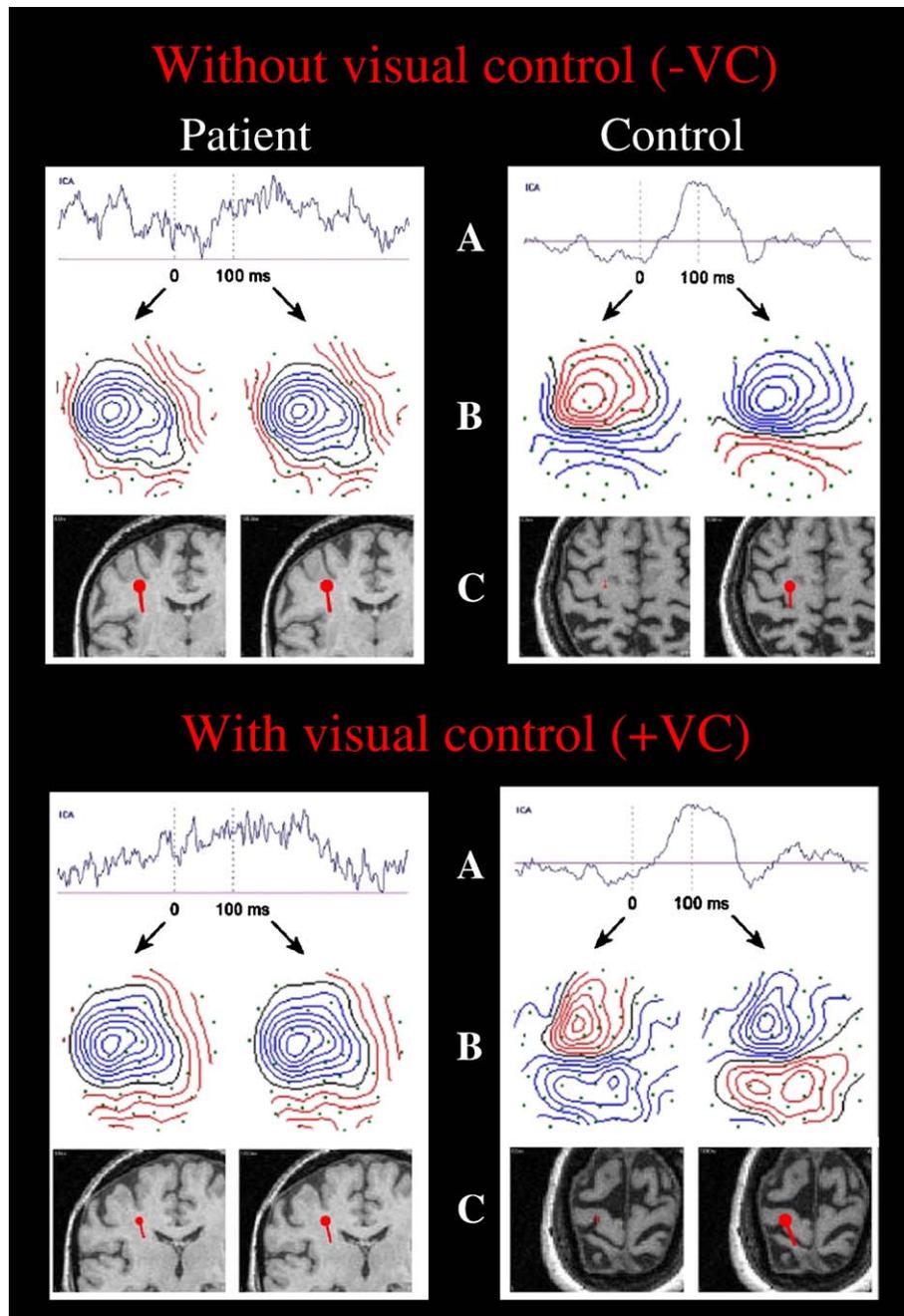
Fig. 4. Source reconstruction for the patient GL on the left and for one control subject on the right at movement onset and 100 ms after it. Upper part: without visual control (VC). Lower part: with visual control (+VC). (A) Time course of the ICA component, (B) topographic map of the ICA with negativity in blue and positivity in red. (C) MRI sections of the individual brains (coronal for the patient and horizontal for the control subject) with the superimposed source and its orientation. Note the reversal of the source direction from anterior to posterior in the control subject. Such reversal is absent in the patient, who shows a source in the depth of the central sulcus. No striking differences between +VC and −VC for both patient and control subject.

(cutaneous and proprioceptive) input from the moving part of the body.

This result is in contradiction with two of the earlier studies in the field of movement-related cortical potentials. Vaughan and Gross (1970) investigated the cortical motor potential for quick wrist extension movements in monkeys before and after upper limb deafferentation (i.e. after dorsal rhizotomy extending from C2 to T4). The motor potential according to these authors included the whole pre- and postmovement potential. The configuration of this potential was not altered after deafferentation though some changes

in timing. Therefore, the authors suggested that kinesthetic feedback is not registered in motor cortex during the performance of voluntary movements. However, their recordings were only from the hand region of the precentral cortex and, as shown in later studies by others (Toma and Hallett, 2003) and ourselves (Kristeva et al., 1979), the postmovement potential (MEP) has its source in the postcentral sulcus. This may partly account for their negative finding.

With respect to human subjects, Rothwell et al. (1982) reported the existence of a postmovement potential in the absence of

peripheral feedback in one deafferented patient. The fact that these authors only used an electrode array of 5 leads only which covered the motor but not the somatosensory cortex could explain their negative finding, as well as individual differences in the severity of the deafferentation. In another patient with a complete large-fiber peripheral neuropathy below the collar level, Cole et al. (1995) reported movement-related cortical potentials within normal limits. However, it is difficult to interpret the results of this report that contains neither figures of the recorded movement-related potentials nor quantification of the findings.

Source analysis

The results from the electric source reconstruction confirmed the absence of the MEP source in the patient GL as well (Fig. 4). All control subjects, in contrast, had a contralateral source. At movement onset, this source had an anterior orientation indicating prevailing activation of the contralateral motor cortex. At approximately 30–40 ms after movement onset, the orientation of this source changed towards posterior. The posterior orientation of this source and its maximum at approximately 90 ms after the movement onset is consistent with activation of the contralateral somatosensory areas as shown by us (Kristeva et al., 1979, 1991; Ball et al., 1999; Kristeva-Feige, 2003) and others (Toma et al., 2002). This MEP source with a posterior orientation was absent in the patient GL who instead had a deeper radial source that was active at the movement onset and did not change its orientation afterwards. The radial orientation of this deep source indicates activation in the depth of the central sulcus. This suggests that GL's whole organization of the motor command is different and possibly involves area 3a, i.e. this subdivision of the primary somatosensory cortex known to receive predominant input from low-threshold muscle afferents activated during the movement (Wiesendanger and Miles, 1982; Kristeva-Feige et al., 1995). An extension into the area 3b which normally receives cutaneous inputs from the periphery cannot be excluded either. Additional studies using methodologies with better spatial resolution (fMRI and MEG source reconstruction) and fMRI-constrained EEG source reconstruction in this exceptional patient are necessary to clarify this issue. This will shed more light as well on another puzzling finding of the present study that no medial SMA source was revealed in the patient GL by the source reconstruction.

Influence of visual feedback on cortical activity and performance

In the control group, the MEPs and their cortical sources were quite similar under both conditions (+VC and −VC). This was also the case for patient GL although visual guidance generally plays a major role in her motor performance. This suggests that the visual input from the moving finger does not exert a major influence on the generation of the simple movement required in the present investigation. This is not surprising in view of the lack of significant differences in the movement parameters under both conditions, except for the significantly higher variance in movement amplitude when the patient GL performed movements without visual control. This finding should be implemented by other non-invasive techniques covering the whole cortical and subcortical motor network. Weeks et al. (1999) have investigated with PET the influence of vision on a thumb-to-finger task in patients with severe but not complete pan-sensory neuropathies and compared the data with those of healthy controls. In the non-

visually guided movement task, the patient group activated primary motor, premotor and cerebellar regions. Without visual guidance, the contralateral and ipsilateral primary sensorimotor cortex was more strongly activated in the patients than in the controls. Similar observations have been reported in a pilot fMRI investigation with patient GL for simple hand and foot movements and for grip force control with and without visual feedback (Hepp-Reymond et al., 2001). Together, these findings strongly suggest that these areas are involved in motor processing independently of peripheral sensory input and thus are consistent with the present EEG findings.

With respect to the motor performance, the fact that the patient GL performs without visual guidance the finger movement almost as well as the controls, although she does not get any sensory input from the moving part of the body, indicates that her motor control relies on an "internal feedforward model", i.e. an internal motor representation predicting the future outcome of an action, mainly based on reactivation of motor memory traces (Wolpert et al., 1995; Wolpert, 1998; Wolpert and Ghahramani, 2000; Wolpert and Flanagan, 2001; Blakemore et al., 2002). It is quite likely that in GL the internal model is not only based on reactivation of motor memory traces but is also continuously trained and updated, as GL is constantly learning and developing new strategies, mostly with visual feedback.

But how to explain the higher variance of the movement amplitude when the patient GL performs the movements without vision? In deafferented patients, "the visual signals provide the only sensory information for making accurate movements" (Blakemore et al., 2002). These signals provide information about the position of the limb prior to movement and give a feedback about the accuracy of the movement. As a result, the motor system will learn to predict the movement outcome and update the current state without any somatosensory information. Newly activated regions, such as the parieto-occipital PO cortex and/or crossmodal plasticity in the parietal operculum SII may account for this performance, as suggested by the findings of Weeks et al. (1999). The brain learns to base such estimates solely on the stream of motor commands or upon visual information. In our study, when the patient GL performed index finger movements without visual feedback, her estimate for the movement amplitude was only based on the internal stream of the motor commands and therefore the amplitude variance increased. Lafargue et al. (2003) also reported a higher variance when patient GL had to produce a constant force output for a few seconds, although the mean motor output was not significantly different from that of the controls. The authors suggested a hierarchical model according to which the variance of motor output is processed at a higher level than the level of the motor output itself. Our present data suggest that the visual feedback from the moving finger most probably had access to this higher level since the amplitude variance only increased when this visual feedback was removed. However, it is still unclear why the patient GL did not show any other kinematic or behavioral impairment without visual guidance.

Functional significance of MEP

Our findings clearly showed that MEP reflects sensory input from the periphery. On the other hand, the patient GL performed the self-paced index finger flexion nearly as well as the controls but without any MEP, i.e. without any afferent sensory input to the cortex. These findings therefore raise the following question: what is the functional significance of the MEP for the execution of such

a simple movement that obviously does not require peripheral feedback to update the internal model?

Lafargue et al. (2003) investigated the ability of the same patient GL and of control subjects to assess different levels of isometric force. They found that the patient GL despite abnormal variability in motor output could assess and scale muscular force without visual feedback like the controls. This means that scaling muscular force is possible on the basis of internal signals only. However, the patient GL reported that she did not experience fatigue or the feeling of applying force throughout the experiment. Furthermore, she did not have any subjective conscious experience of the applied force. Hence, the authors drew the conclusion that "endogenous signals might need to interact with afferent input to gain access to consciousness".

In our study, the patient GL was aware of giving a command to execute the movements but she did not have the feeling of the movement itself. This means that she had a normal motor awareness but did not have a perceptual awareness of the movements. Therefore, we conclude that the internal model has to interact with the sensory input from the periphery, whose electrophysiological correlate is the MEP, to gain access to consciousness (Haggard, 2005). Sirigu et al. (2004) on the basis of the altered awareness of voluntary action in parietal and cerebellar patients proposed the existence of several internal models in predictive motor control, one for the consciousness of movement intention and planning and the other for processing of the performance itself. These various questions have to be clarified in patient GL by further studies where conscious intention and motor awareness have to be assessed in more detail as well as activity in other cortical and subcortical regions (Haggard, 2005).

In conclusion, the absence of MEP in the deafferented patient GL provides direct evidence that this potential reflects sensory information from the moving part of the body. Although the patient GL has no sensory feedback from the periphery, she was able to perform the simple pulse movements as well as the controls. She was aware of giving a command for movement but did not feel the movements, i.e. she had a normal motor awareness but did not have a perceptual awareness of the movements. This suggests that the sensory input from the periphery, the correlate of which is the MEP, may be mandatory in order to interact with some components of the internal model in order to get a perceptual awareness of action.

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