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Functional mapping of human sensorimotor cortex with electrocorticographic spectral analysis
I. Alpha and beta event-related desynchronization

Nathan E. Crone,[1] Diana L. Miglioretti,[3] Barry Gordon,[1,4,5] Jeffrey M. Sieracki,[1] Michael T. Wilson,[1] †Sumio Uematsu[2] and Ronald P. Lesser[1,2,5]

Departments of [1]Neurology and [2]Neurosurgery, The Johns Hopkins University School of Medicine, and Departments of [3]Biostatistics and [4]Cognitive Science, and the [5]Zanvyl Krieger Mind/Brain Institute, The Johns Hopkins University, Baltimore, Maryland, USA

Correspondence to: Dr Nathan Crone, Department of Neurology, The Johns Hopkins Hospital, Meyer 2-147, 600 N. Wolfe Street, Baltimore, MD 21287, USA. E-mail: ncrone@jhmi.edu

†*Deceased*

Summary
Human scalp EEG studies have shown that event-related desynchronization (ERD) in the alpha (8–13 Hz) and beta (15–25 Hz) bands may be used to detect functional activation of sensorimotor cortex. However, in most previous studies somatotopy has not been examined in detail and brief, self-paced movements, focusing on the planning of motor output, have been used. We recorded electrocorticographic (ECoG) signals in five clinical subjects during a visual–motor decision task that was designed to activate the representations of different body parts in sensorimotor cortex. To focus more on execution of motor output than on its planning, subjects were instructed to make sustained isometric muscle contractions in different body parts (tongue protrusion, fist-clenching or foot dorsiflexion) in response to randomized visual stimuli depicting each action. ECoG spectral analysis utilized a mixed-effects analysis of variance model in which within-trial temporal dependencies were taken into account, and the magnitude and statistical significance of alpha and beta ERDs were mapped onto a surface rendering of each subject's brain MRI. Cortical electrical stimulation was performed in all subjects for clinical purposes, and the resulting maps of sensorimotor function were compared with those generated by ECoG spectral analysis. During the early phases of the motor responses, alpha ERD commonly occurred in a diffuse spatial pattern that was not somatotopically specific. During the late phases, the spatial pattern of alpha ERD usually became more focused and somatotopically specific. Maps of alpha ERD were closer to cortical stimulation maps when alpha ERD was sustained throughout the late phases of the motor responses. Thus, the topography of alpha ERD more resembled traditional somatotopy when its temporal profile approximated that of the motor response. The topography of beta ERD was often more discrete and somatotopically specific than that of alpha ERD, but beta ERD was often transient and sometimes absent. Sometimes, unilateral limb movement produced sustained alpha and beta ERD over bilateral sensorimotor cortices, with overlapping patterns for different body parts. The topographical spread of alpha ERD beyond expected functional–anatomical boundaries during early (and sometimes late) phases of motor responses invites a re-examination of traditional assumptions about sensorimotor functional neuroanatomy, as well as the role of alpha ERD as an index of cortical activation. We agree with others that the somatotopic representations of different body parts overlap more than previously thought. Also, unilateral limb movements may be associated with both contralateral and ipsilateral activation of sensorimotor cortex. We conjecture that alpha ERD may reflect activity within a broad synaptic network with distributed cortical representations.

Keywords: event-related desynchronization; alpha band; electrocorticography; sensorimotor cortex; functional brain mapping

Abbreviations: BA = Brodmann area; ECoG = electrocorticography; ERD = event-related desynchronization; ERS = event-related synchronization; FFT = fast Fourier transform; MEG = magnetoencephalography

Introduction

The traditional view of a static and strictly somatotopic representation of different body parts in sensorimotor cortex, conceptually crystallized by Penfield and Boldrey (1937), has given way in recent years to a more complex functional–anatomical map in which the representations of different body parts are more diffuse and overlapping, capable of dynamically changing over both short and long time scales (Kaas *et al*., 1983; Asanuma 1991; Pons *et al*., 1991; Ramachandran *et al*., 1992; Recanzone *et al*., 1992*b*; Nudo *et al*., 1996). This paradigmatic shift in the functional anatomy of sensorimotor cortex has been based largely upon neurophysiological studies in animals but, more recently, it has received further impetus from studies using new techniques for mapping brain function in humans (Pascual-Leone *et al*., 1994; Schlaug *et al*., 1994). For example, in PET and, more recently, functional MRI studies, regional changes in cerebral metabolism and blood flow have been utilized to measure neuronal activity associated with movement indirectly (Grafton *et al*., 1991; Kim *et al*., 1993). In contrast, electromagnetic techniques such as EEG and magnetoencephalography (MEG) have been used to measure the summed activity of cortical neurons directly. Although the spatial resolution of electromagnetic measures has often been suboptimal due to the inverse problem of source localization at the scalp surface, they have always offered a potential means of studying neuronal population dynamics with a temporal resolution high enough to correlate neural and behavioural events.

In addition to signal averaging in the time domain (event-related potentials), analyses of electromagnetic signals have also focused on event-related changes in the frequency domain. Observations of reactivity in the occipital alpha (Berger, 1930; Adrian and Matthews, 1934) and central mu (Jasper and Andrews, 1938; Gastaut, 1952; Chatrian *et al*., 1959) rhythms have supported the hypothesis that rhythmic activity in the alpha band (~8–13 Hz, which includes the mu rhythm) is generated by cortex that is resting, and that when cortex is active, this activity is reduced or suppressed. Although the neurophysiological mechanisms underlying these EEG rhythms and their reactivity are only partially understood (Steriade *et al*., 1990; Lopes da Silva, 1991), scalp EEG studies have consistently demonstrated power suppression in the alpha band, dubbed event-related desynchronization (ERD), during tasks activating sensorimotor cortex (Pfurtscheller and Aranibar, 1979). Under these conditions, ERD has also been observed in the beta (15–25 Hz) band (Pfurtscheller, 1981), a phenomenon first described by Jasper and Andrews (1938).

Alpha ERD and beta ERD can also be observed in electrocorticographic (ECoG) recordings (Arroyo *et al*., 1993; Crone *et al*., 1993; Toro *et al*., 1994). Although ECoG requires the surgical implantation of subdural electrodes, the close proximity of these electrodes to the cortical surface overcomes some of the important technical limitations of scalp EEG. Because of the closer electrode spacing (usually 1 cm) and lack of spatial blurring from the scalp, skull and dura mater, ECoG recordings have a spatial resolution which is inherently superior to that of scalp EEGs (Cooper *et al*., 1965; Gevins *et al*., 1994). Because ECoG electrodes lie directly on the cortical surface, and because they are more distant than scalp electrodes from muscle, and therefore electromyographic activity, the signal-to-noise ratio of ECoG recordings is also better than scalp EEG. In addition, activity in the beta band and other high frequencies is better recorded with ECoG because there is less spatial summation and phase cancellation than in scalp recordings (Pfurtscheller and Cooper, 1975). For these reasons, we decided to re-examine alpha ERD and beta ERD in sensorimotor cortex using ECoG spectral analysis, in which enhancements of Pfurtscheller's (1989) basic strategy are applied to ECoG signals. In a companion paper (Crone *et al*., 1998) we present the results of using ECoG spectral analysis in higher frequency bands (30–100 Hz).

The primary goals of this study and its companion were interdependent: (i) to investigate the utility of different spectral indices of cortical activation and (ii) to use these indices to explore the functional anatomy of sensorimotor cortex in humans. Any new measure of functional brain activation should be validated by comparison with a fully established measure, but since no such measure has been agreed upon, new functional brain mapping techniques are usually evaluated in a region of the brain where several other, more established measures have already been used to construct a model, however preliminary, of its functional anatomy. Therefore, most new measures have been evaluated with tasks that activate sensorimotor cortex or visual cortex, because these brain regions have been so extensively studied, both in animals and in humans. However, this strategy may be complicated by the fact that functional–anatomical models continue to evolve, often in response to observations made with new methods. For example, the most conspicuous and universally recognized features of the functional anatomy of sensorimotor cortex include (i) its somatotopic organization, (ii) its crossed lateralization and (iii) its division of sensory and motor function by the central sulcus. Although new techniques of functional activation, i.e. PET, functional MRI and MEG, have used these features as benchmarks for comparison and self-validation, studies in animals and in humans have suggested that these benchmark features are oversimplified abstractions of a more complicated underlying structure. In spite of these difficulties, we adopted a similar strategy to evaluate the ability of alpha ERD and beta ERD to measure functional cortical activation. Because previous studies of these measures using scalp EEG have often produced results at variance with traditional benchmark features of sensorimotor organization, we chose to re-examine these features with ECoG spectral analysis.

To examine the somatotopic organization of sensorimotor

cortex using alpha ERD and beta ERD, we designed a motor task in which movements of different body parts (i.e. tongue, arm and leg) were expected to produce activation in somatotopically defined regions of the contralateral precentral gyrus. Few studies of alpha ERD and beta ERD have specifically examined somatotopy. In studies of alpha ERD using scalp EEGs (Pfurtscheller and Neuper, 1994; Pfurtscheller *et al*., 1994), a somatotopic pattern was not consistently shown during movements of different body parts. In a study using both scalp EEG and ECoG recordings, the brisk self-paced movements of a finger were compared with those of the foot, but not with those of the tongue (Toro *et al*., 1994). Both finger and foot movements produced alpha ERD over contralateral cortical hand regions, but the onset of alpha ERD prior to finger movement was much earlier in this region than during foot movement. The spatial pattern and temporal course of beta ERD, however, was similar for finger and foot movements. In another ECoG study (Arroyo *et al*., 1993), tongue movement was studied, as well as movements of hand and foot, but this study focused primarily on changes in the mu rhythm and the frequency spectrum was analysed with a coarse temporal resolution (2.6 s). However, these authors did demonstrate somatotopy, using a strict criterion for mu rhythm blocking (>90% reduction). In the present investigation, and in one previous study (Crone *et al*., 1993), we recorded ECoG signals and analysed their power spectrum with a higher temporal resolution (~200 ms) to confirm the somatotopic specificity of both alpha and beta ERD during tongue, arm and leg movements.

Scalp studies of alpha ERD have typically contradicted the widely accepted crossed lateralization of sensorimotor function by showing bilateral ERD in association with unilateral limb movements. This bilaterality has been observed with scalp EEG (Chatrian *et al*., 1959; Pfurtscheller and Aranibar, 1979; Pfurtscheller, 1989), MEG (Salmelin *et al*., 1995) and ECoG (Arroyo *et al*., 1993; Toro *et al*., 1994). For the most part, brief, self-paced movements have been used in these studies to elicit ERD, and distinctions between contralateral and ipsilateral limb movements have been made on the basis of asymmetries in the magnitude or time course of ERD, usually during the planning stages before movement onset. For example, alpha ERD in scalp EEG (Pfurtscheller, 1989) occurs only over contralateral sensorimotor cortex prior to movement onset, but at movement onset and thereafter, alpha ERD is bilateral and nearly symmetrical in magnitude. Similar results have been obtained with MEG (Salmelin *et al*., 1995) and with ECoG (Toro *et al*., 1994), also using self-paced finger movements. We thought a different motor task might allow us to examine this phenomenon from a different perspective. In particular, we chose to focus on execution of the motor output rather than its planning, and to accomplish this we devised a non-verbal, visually cued motor task in which the motor response consisted of a sustained muscle contraction.

The strategy outlined above for validating new functional activation measures could also be complicated by the variability of functional anatomy between individual human subjects (Ojemann, 1979). This could be particularly problematic for functional mapping techniques that rely upon averaging across subjects, e.g. PET and many scalp EEG studies. In addition, in most such studies, no other measures of the subjects' functional anatomy are used, so there can be no corroborative accounting for individual variability. Models of functional anatomy derived from the literature are based upon other individual subjects, often under vastly different experimental conditions. Because the clinical situations of our subjects required functional mapping in preparation for resective treatment, we were able to compare our results with ECoG spectral analysis with those of a more established functional mapping technique using cortical electrical stimulation. Cortical stimulation mapping is known to provide a reasonably accurate map of many cerebral functions (Ojemann, 1983). In many instances, it produces a hyperacute, reversible functional lesion of the brain (Li and Chou, 1962; Landau *et al*., 1965). This method thus provided a complementary verification of sensorimotor functional anatomy in each of our subjects. This paper describes, in detail, the results of using ECoG spectral analysis to map sensorimotor cortex in five individual clinical subjects, one of whom is our major focus because recording electrodes covered most of his sensorimotor strip and because clinical circumstances required two different electrode implantations (6 months apart), allowing us to study the reproducibility of our findings. By studying five different clinical subjects with ECoG spectral analysis and cortical stimulation, we were also able to examine the inter-individual variability of the functional organization of sensorimotor cortex from the complementary perspectives of an activation technique and a reversible lesioning method, respectively.

Methods

Subjects

In a subset of patients undergoing surgery for the treatment of intractable epilepsy, subdural electrodes are implanted on the surface of the brain and left in place for 7–14 days in order to localize a seizure focus and to map brain function (with cortical electrical stimulation) prior to focal resection (Lesser *et al*., 1987). This special clinical situation gives us the unique opportunity to record ECoG signals while patients are awake and able to cooperate with experimental tasks, without exposing them to any risks beyond those already assumed for strictly clinical purposes. Five patients agreed to participate in this research study and gave their informed consent according to a protocol approved by the Joint Committee on Clinical Investigation of The Johns Hopkins Medical Institutions, in compliance with standards of the Declaration of Helsinki. One of these patients (Subject 5) did not have a definite history of epilepsy but did have a left posterior mesial temporal arteriovenous malformation and required functional mapping to determine a safe margin of surgical resection. This patient

Table 1 *Clinical characteristics and experimental tasks for each subject*

Subject	Age (years)	Handedness	Language lateralization*	Seizure focus	Lesion location	Location of ECoG electrodes	Body parts tested
1A	30	Right	Left	Left frontal	Left inferior frontal encephalomalacia	Left frontoparietal	Tongue/hand
1B	31	Right	Left	Left frontal	Left inferior frontal encephalomalacia	Left frontoparietal	Tongue/hand/leg
2	23	Right	Left	Left anterior temporal	Left mesial temporal sclerosis	Left and right frontoparietal	Tongue/hand
3	22	Right	Bilateral	Left precuneus, right cingulate	Left precuneus venous angioma	Left and right frontoparietal	Tongue/hand/leg
4	23	Right	Left	Left anterior temporal	Left mesial temporal sclerosis	Left frontal–temporal–parietal	Tongue/face/hand/leg
5	38	Right	Not tested	None	Left posterior mesial temporal AVM†	Left frontal–temporal–parietal	Tongue/face/hand/leg

*Determined by intracarotid amobarbital procedure. †AVM = arteriovenous malformation.

was treated with phenytoin for a few episodes of uncertain diagnosis, 3–4 years before her surgery. After a few months the phenytoin was discontinued, and she had no further episodes. While subdural electrodes were implanted in this patient, she had no epileptic seizures and no spontaneous epileptiform discharges on ECoG.

Prior to subdural electrode implantation, all clinical subjects underwent a thorough presurgical work-up, including prolonged video/EEG monitoring, neuropsychological testing, three dimensional MRI and cerebral angiography with the intracarotid amobarbital procedure (not done in Subject 5). The clinical characteristics of the subjects are summarized in Table 1. Each subject's full scale IQ (Wechsler Adult Intelligence Scale) was in the normal range; in Subject 1 it was 103, and in Subjects 2–5, it was 112, 82, 112 and 87, respectively. In each subject, sensorimotor function was verified to be normal by the absence of focal motor or sensory deficits on neurological examination. Subject 1 had left inferior frontal encephalomalacia that did not involve the cortical regions under study (see Fig. 3 below).

In each subject, the surgical implantation of subdural electrode arrays was performed according to established procedures (Lesser *et al*., 1990). The location of implanted electrodes was based upon the clinical situation of each individual subject and therefore could not be uniform across subjects. Even if identical implantations were justified, they would be technically difficult to accomplish, and if this could be accomplished, there would still be individual differences in neuroanatomy and functional organization. These are limitations that we had to accept in order to receive the benefits of ECoG recordings. In this paper we present our spectral analyses of ECoG recorded over the left frontoparietal convexity in one subject (Subject 1; see Fig. 3 below), over both left and right frontoparietal convexities in two subjects (2 and 3) and over the left frontoparietal operculum in two subjects (4 and 5; see Figs 9 and 10 below, and also Table 1).

Subject 1 underwent subdural grid implantation twice (1A and B). Because video/EEG monitoring with scalp electrodes had failed to lateralize or localize his seizure focus clearly, this patient's first subdural implantation was planned as a screening procedure, and included multiple subdural electrode strips over bilateral frontal, parietal and temporal lobes, with the most extensive coverage over left frontoparietal regions (see Fig. 8 below). Seizure discharges were found anterior and superior to Broca's area, in the left inferior and middle frontal gyri. In order to define the seizure focus better and perform more complete functional mapping in these regions, a second implantation was carried out with detailed coverage of left anterior frontal regions, as well as left frontoparietal regions. Both ECoG and cortical electrical stimulation were performed after both implantations, enabling a direct comparison of the cortical maps generated by the two techniques in the same individual, 6 months apart.

All subjects with epilepsy were taking tapering doses of anti-epileptic medications, and all were seizure-free for at least 6 h prior to testing. In Subject 5 cortical electrical stimulation produced frequent after-discharges at low stimulus intensities, and treatment with phenytoin was required to complete her functional mapping. All subjects had fully recovered from the surgical implantation of subdural electrodes (time after surgery ranged from 4 to 13 days), and all were alert and attentive at the time of testing. On-line ECoG analysis during experimental recordings allowed immediate detection of artefacts and electrographic seizure activity. In all subjects, the ECoG was free of seizure patterns throughout testing.

Cortical electrical stimulation maps

All subjects underwent cortical stimulation mapping according to established clinical procedures (Lesser *et al*., 1987). Each subdural grid consisted of a 1.5-mm-thick silastic sheet embedded with platinum–iridium electrodes (4-mm diameter with a 2.3-mm diameter exposed surface) equally spaced with 1-cm centre-to-centre distances. Constant current electrical stimulation between pairs of adjacent electrodes was given using a Grass S-88 or S-12 cortical stimulator (Grass Instrument Co., Quincy, Mass., USA) with 1–5-s trains of 50 Hz, 0.3-ms, alternating polarity square-wave pulses, starting with a stimulus intensity of 1 mA and increasing in 0.5–1.0-mA increments up to a maximum of 15 mA, with adjustments to avoid prolonged after-discharges. While the stimulus intensity was increased, each subject reported any unusual or involuntary sensations or movements, and once a maximal stimulus was determined, disruption of motor function was detected by observing the subject during wiggling movements, in turn, of the tongue, the fingers of both outstretched arms, and the toes and feet of both legs. All testing sessions were recorded on videotape, incorporating views of the patient and of relevant EEG channels on the same screen.

Experimental procedures

A visual–motor decision task was designed to investigate the somatotopic distribution of cortical responses during sensorimotor performance. We asked each subject to make a sustained voluntary muscle contraction in one of several possible body parts (tongue, face, arm or leg) in response to a visual stimulus depicting the particular body part. The visual stimuli consisted of black-and-white line drawings depicting different motor actions: tongue protrusion, eye-winking, fist-clenching or dorsiflexion of one foot. These visual stimuli were designed to minimize overt and covert verbal mediation of the task. We expected the subject's motor responses to produce a somatotopically specific pattern of brain activation in sensorimotor cortex. Each of the pictured body parts was presented on a video monitor in a random sequence in order to minimize somatotopically specific pre-stimulus motor preparation. The subject was instructed to begin the action depicted as quickly as possible, and to sustain the muscle contraction until the visual stimulus disappeared—a fixed interval of 3 s after it appeared. We therefore expected the task to produce more sustained brain activation than would be seen with a briefer motor response. Furthermore, we expected that the simple temporal profile of the task would facilitate the temporal correlation of motor responses with changes in the ECoG frequency spectrum. When the visual stimulus disappeared, the subject relaxed and waited for the next stimulus while maintaining gaze fixation on a black dot in the centre of the video screen. The minimum time between the offset of any one stimulus and the onset of the next stimulus was 3.8 s.

Although tongue protrusion was always in the midline and involved bilateral tongue muscles, in all subjects the visual–motor decision task was performed separately for limb movements on the right (e.g. tongue, right face, right arm and right leg) or on the left (e.g. tongue, left face, left arm and left leg) sides. In Subject 4, all four body parts (25 trials for each body part and 100 trials in total) were tested on the right side only (contralateral to the grid). In two of the three subjects in whom ECoG electrodes were unilateral (1 and 5), both sides of the body were tested in order to study cortical activation associated with ipsilateral limb movements. In Subject 5, all four body parts were tested on both sides (25 trials for each body part on each side, 100 trials on each side and 200 trials in total). In order to maximize the number of trials recorded without exhausting the subject, three subjects (1A, 2 and 3) were presented with only the tongue and hand stimuli (50 trials for each body part on each side, 100 trials on each side and 200 trials in total), and one subject (1B, second implantation) was presented with only the tongue, arm and leg stimuli (50 trials for each body part on each side, 150 trials on each side and 300 trials in total).

A simple visual–motor task was also used in one of our subjects (1B) to investigate the effect of the decision component of our visual–motor decision task on somatotopic patterns of cortical activation. In this task our subject was tested with a series of 50 stimulus–response trials using only one body part. This task utilized the same visual stimuli used in the visual–motor decision task (the tongue, the right or left arm, or the right or left leg). However, instead of asking the subject to decide which body part to move in response to a randomized visual stimulus, the subject was asked to make the same movement for every trial, in response to a visual stimulus which was identical in every trial. This task condition, which will be referred to as the 'simple' condition, is contrasted in this paper with the 'decision' condition described above.

The testing apparatus consisted of a dual computer system. The first computer was responsible for experimental execution and control, including stimulus presentation, response verification and response scoring. The second computer was responsible for digital ECoG. Visual stimuli were presented by the first computer on a video monitor approximately 1 m in front of the subject in the centre of his or her visual field. A unique ECoG file was recorded for each experimental task. Breaks were taken during the testing session if the subject became sleepy, impatient, or if they lost concentration. Each task typically lasted ~10–15 min, and a testing session typically consisted of three or four experimental tasks.

Subdural ECoG

Up to 64 channels of ECoG signals were amplified (5×1000) and filtered (1–100 Hz, 6 dB per octave and a 60 Hz notch) using Grass amplifiers (Model 12A5). All ECoG signals were recorded with a referential montage using a single intracranial (subdural) reference electrode chosen for its relative inactivity

and greatest distance from the active recording electrodes. We did not use a scalp or non-cephalic (e.g. neck) reference electrode because a companion study (Crone *et al*., 1998) used the same ECoG signals to study higher frequencies (30–100 Hz) that overlapped the power spectrum of electromyographic activity. The amplified ECoG signal was multiplexed and digitized (1-kHz sampling rate) using a DAP 2400/6 converter (Microstar Laboratories, Bellevue, Wash., USA) in an IBM-compatible 486 33-MHz computer. Experimental markers such as trial number, stimulus onset and stimulus offset were transmitted from the experimental control computer to the ECoG recording computer and multiplexed in parallel with the ECoG signal. The digital ECoG file was stored on a 1-Gb hard disk and later archived on non-rewriteable optical media.

ECoG signal analysis

Rationale

There may be a significant amount of natural variability in the background EEG that is not necessarily associated with task-related cortical processing (Oken and Chiappa, 1988). In addition, changes in arousal, as well as in the focus of attention, may occur within fractions of a second and affect the EEG (and ECoG) power spectrum in unpredictable ways. Since these variables could not be fully controlled in our experiments, we recorded a large sample of ECoG during activation conditions and compared its power spectrum with that of another large sample of ECoG without activation, i.e. baseline. This was done by recording the ECoG continuously during many trials (25–50) of the same set of neural operations, i.e. task conditions. In order to take into account the short-term variability of the ECoG power spectrum, we compared short segments of task-activated ECoG following each stimulus presentation with short segments of baseline ECoG recorded immediately before each stimulus presentation. Since we did not know a priori the exact latency or duration from stimulus onset at which brain activation would occur, we took an exploratory approach and divided the segments of ECoG during activation (post-stimulus), and during baseline (pre-stimulus), into equally spaced 200-ms epochs. A measure of the difference, or change, between the power spectra in pre- and post-stimulus epochs was then calculated, and statistical analyses were performed to determine whether the magnitude of change was significantly different from zero.

Data reduction

For this study and a companion study (Crone *et al*., 1998) examining high frequency (30–100 Hz) ECoG activity, ECoG signals were first digitally filtered with a finite impulse response filter created in FDAS version 1.4 (Momentum Data Systems, Costa Mesa, Calif., USA) with a passband of 8–100 Hz and a 70 Hz notch (video refresh rate = 70 Hz).

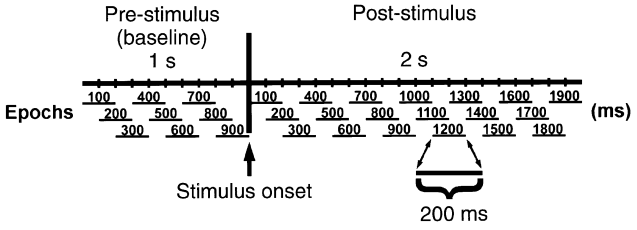

Fig. 1 Method of segmenting EEG into epochs prior to FFT. Epochs are 200 ms in length and are labelled according to their temporal midpoints. Adjacent epochs overlap each other by 50% (100 ms).

Because differences in the distance between active and referential intracranial electrodes were significant and could affect the spatial distribution of the power spectrum, all multi-channel ECoG signals were remontaged using an average reference. This procedure produces a spatial distribution of power spectra which is relatively independent of the location and activity of the reference electrode (Lehmann, 1987). Although it may also act as a spatial high-pass filter, we did not consider this effect to be undesirable since we were interested in local changes in the power spectrum reflecting regional cortical activation, rather than widespread power changes that might be more related to global state alterations (e.g. attention or arousal). Although the average reference could potentially allow widespread EEG activity to manifest itself in an 'island' of inactivity, widespread power changes were not observed in our data. In addition, any potential blurring this method might have produced in spite of the generous spatial sampling of the subdural electrode arrays was felt to be acceptable given the level of anatomical analysis used in this study.

Prior to spectral analysis ECoG signals were segmented into 200-ms epochs that overlapped in time by 50% (i.e. by 100 ms). This segmentation was chosen as a compromise between the temporal and frequency resolutions of the fast Fourier transform (FFT) used to estimate the power spectrum. The temporal centre of each epoch had a fixed temporal relationship ($n \times 100$ ms for the nth epoch) to the time of stimulus onset. These epochs covered the 1 s before (9 epochs) and 2 s after (19 epochs) stimulus onset (Fig. 1).

We used the FFT to estimate power in the alpha and beta bands. However, performing the FFT on 200-ms epochs yields power measurements at 5 Hz intervals with equivalent bandwidths of 5 Hz, and does not allow a specific measurement of alpha power. Therefore, to localize our power measurements better within the frequency domain, each 200-ms EEG segment was 'zero-padded' to 512 samples before applying the FFT. This procedure increases the number of power estimates (frequency points) by the number of zeroes appended to the original signal, resulting in an interpolation in the frequency domain (Marple, 1987; Porat, 1997). However, it does not change the equivalent bandwidths (centre lobe) of the power estimates. In our case zero padding yielded power estimates at 1.95 Hz intervals (e.g. 1.95, 3.9, 5.85, 7.8 and 9.75 Hz), with equivalent bandwidths of ~5 Hz

each (e.g. 9.75 ± 2.5 Hz). Power in the alpha band, commonly defined as 8–13 Hz (Niedermeyer and Lopes da Silva, 1993), was calculated by summing the power estimates at 9.75, 11.7 and 13.65 Hz. The estimate at 7.8 Hz was not used because it was centred at just <8 Hz and because the digital filter used was not capable of suppressing all power, which was considerable, <8 Hz. These choices may have excluded some power in the 8–10.3 Hz frequency range in our alpha power estimate (from the estimate centred at 7.8 Hz, range 5.3–10.3 Hz) and may have included some power in the 7.3–8 Hz range in our estimate (from the estimate centred at 9.75 Hz, range 7.25–12.25 Hz). Nevertheless, we thought that given the limitations of the FFT, these choices led to the best estimate of power in the lower alpha band. The power estimate at 13.65 Hz (range 11.15–16.15 Hz) was included because it contained power from the upper alpha band. Theoretically, it also contained power from the lower beta band, but we expected that the rapid decline in power from alpha to beta bands made this contribution insignificant. To confirm this, all subsequent statistical analyses were also performed with an alpha-plus band, in which the power estimate at 15.6 Hz was summed with the estimates at 9.8, 11.7 and 13.7 Hz. Comparing the results obtained for the alpha band and the alpha-plus band demonstrated that any contribution of activity in the beta band to power changes in the alpha band was inconsequential. Therefore, in the remainder of this paper we refer to the alpha band only. Similarly, we estimated power in the beta band, commonly defined as 15–25 Hz, by summing power estimates at 17.6, 19.5, 21.5, 23.4 and 25.4 Hz. We did not include the estimate at 15.6 Hz because much of its equivalent bandwidth was <15 Hz and because we did not want the upper alpha band to contaminate our measurement of beta power.

Although the spatial and temporal patterns of ERD have been shown to differ for different frequency bands within the alpha and beta bands (Pfurtscheller and Berghold, 1989), we chose to limit our present study to an analysis of the broad bands that have been most commonly defined as alpha and beta. There are many different ways in which the alpha and beta bands can be subdivided. Because we did not have unlimited resources to investigate all potential permutations, and because individual power estimates (e.g. 9.8 and 11.7 Hz) were likely to be much more variable (yielding less statistical power) than the sum of several estimates, we chose to study ERD in the alpha and beta bands defined above.

Artefact rejection

All research test sessions were recorded on videotape, and a record of response accuracy and trial validity was kept for each trial. Stimulus–response trials were excluded if the subject was obviously distracted or if the motor response was excessively delayed, indicating a lack of concentration or response certainty. Motor responses were recorded with surface EMG, and reaction times (times between stimulus and EMG-response onsets) were later estimated by visual analysis for each trial. Surface EMGs were recorded with bipolar electrodes situated posterior to the chin (mylohyoid and geniohyoid muscles) and in both arms over the ulnar aspect of the proximal third of the flexor surface (flexor carpi radialis and ulnaris, palmaris longus and flexor digitorum superficialis muscles). When foot dorsiflexion was included in a task, surface EMG was also recorded from electrodes over the tibialis anterior muscle in both legs. The reaction times for different body parts under different task conditions are detailed for Subject 1B in a companion paper (Crone *et al*., 1998), in which they were found to have a more obvious correlation with event-related synchronization (ERS) in the gamma band than we found with alpha or beta ERD.

Although all subjects learned the experimental tasks and performed them without difficulty, a few trials with ambiguous or incorrect motor responses were excluded from analysis in each subject. Examples of ambiguous motor responses included those in which: a response was not sustained; a response from a different or contralateral body part occurred simultaneously; and a response from the previous trial continued into the baseline period of the current trial.

Identification of ECoG artefacts was done by visual inspection of the raw ECoG files by a board certified electroencephalographer (first author) before digital filtering. Channels that were affected by electrode artefact were excluded from subsequent analyses, and individual stimulus–response trials were excluded if there was any motion artefact or epileptiform activity, i.e. even a single epileptic spike or sharp wave.

Statistical analyses

Prior to statistical analysis a natural logarithmic transformation was performed on the absolute power spectral density values from the FFT to approximate a Gaussian (normal) distribution. This transformation has been found by us and by others (Gasser *et al*., 1982; Oken and Chiappa, 1988) to be best suited for EEG power spectral analyses.

To determine whether the spectral density values in post-stimulus epochs were significantly different from those in pre-stimulus (baseline) epochs, we first calculated a measure of the change in log power (ERD) for each post-stimulus epoch within each trial. In order for this measure to account for task-independent temporal variability in the power spectrum, the log powers in all pre-stimulus baseline epochs were averaged for each trial and subtracted from the log power in each post-stimulus epoch of the same trial. A separate mixed-effects ANOVA (analysis of variance) model was fitted for each channel (as many as 64 channels) and each frequency (alpha and beta) using the change value as the dependent variable and the post-stimulus epoch as the independent variable. Since repeated measurements were taken over time (i.e. multiple trials and multiple epochs within a trial), it was important to account for temporal dependencies within the data. Because the correlation between

power measurements made within each trial (i.e. in different epochs) may depend upon the time separation between the measurements (serial correlation), we used a first-order autoregressive covariance structure for the residual term of the ANOVA. Because we expected the magnitude of ERD to be relatively homogeneous within trials and heterogeneous between trials, a random effect for the trial number was also included in each ANOVA model.

Formally, for each channel and frequency bin, and for a task with N trials and 19 post-stimulus epochs per trial, we fitted the following model for the jth epoch of the ith trial:

$$Y_{ij} = X_{ij}\beta_j + Z_{ij}\gamma_i + \varepsilon_{ij}, \text{ with } i = 1, \ldots, N \text{ and } j = 1, \ldots, 19$$

where Y_{ij} is the observed change in log power for the jth epoch of the ith trial from the average over the ith trial's baseline epochs; X_{ij} is a dummy variable indicating the jth post-stimulus epoch; β_j is the mean change in log power for the jth post-stimulus epoch; Z_{ij} is a dummy variable indicating the ith trial; γ_i is the random effect parameter for the ith trial, where $\gamma_1, \gamma, \ldots, \gamma_N$ are assumed to be N mutually independent Gaussian random variables with mean zero and common variance; and ε_{ij} is the residual or error term for the jth epoch of the ith trial, and is assumed to be normally distributed with mean zero, variance σ^2, and covariance between ε_{ij} and $\varepsilon_{ij'}$ of the form $\sigma^2 \times \rho^{|j-j'|}$ where $0 < \rho < 1$.

For more detailed information on the above model, see Diggle *et al*. (1994). We constructed t-type confidence intervals as the estimated mean $\pm\, t_{\nu, 0.975} \times$ SE where $t_{\nu, 0.975}$ is the 97.5th percentile of the t_ν-distribution (resulting in a 95% confidence level), (ν is the residual degrees of freedom, and SE (the estimated standard error) is adjusted for the random effect of trial and takes into account the serial correlation over time, within a trial. The mean values and confidence limits were then back-transformed to give the geometric mean percentage change from baseline (ERD) and 95% confidence limits. These values were then plotted against time to illustrate the magnitude and statistical significance of ERD over time. If the confidence intervals do not include zero, then ERD is significant at the 0.05 level. Confidence intervals were not adjusted for multiple comparisons. Thus, if there were no post-stimulus change in the power of a particular frequency band in a particular channel, we would expect the upper confidence limit to drop below zero by chance in one out of 40 single epochs. However, based upon previous accounts of ERD, we did not expect to observe it in single epochs. Rather, we sought to detect clusters or trends of ERD in adjacent epochs, which were much less likely to occur by chance. Since this was an essentially exploratory analysis of the topographical and temporal behaviour of ERD, without an a priori hypothesis about a single location or time for ERD to occur, it was more important to us to preserve the nominal behaviour of the individual confidence intervals than to control for the overall type I errors. For these reasons we present the entire time series of ERD in many of the figures in this paper. However, when it was necessary to summarize the spatial locations of significant ERD, we chose only those electrodes in which the confidence limits of ERD did not include zero during at least three consecutive epochs. This criterion represented a compromise between optimal temporal resolution and the need to define a cluster with at least two non-overlapping epochs. To evaluate this criterion we performed repeated (n = 2000) simulations of our analyses on detrended time-series (a subset of data from Subject 1B) that were randomly selected with respect to electrode and trial, albeit preserving the autocorrelation structure of the original data. Only 15 out of 2000 simulations (0.75%) resulted in three consecutive epochs in which the 95% confidence limits did not include zero. Although the three-consecutive-epoch criterion could have been met by chance in a few of the many electrode sites analysed, this criterion was far exceeded in nearly all selected electrode sites, which were themselves often clustered in a non-random distribution. Likewise, when we illustrated the magnitude of ERD with a colour scale, the magnitude was calculated as the average of three consecutive epochs, and if it was maximal (red), it was always significant in at least these three consecutive epochs.

Neuroanatomical correlations

The position of the recording electrodes with respect to underlying cortical gyral anatomy was in most cases verified by direct visualization by the neurosurgeon and surgical epileptologist, in the operating room, during both implantation and removal. At this time, photographs were taken and graphical notes were made depicting the relationship of each electrode position to identifiable cortical landmarks, i.e. the sylvian and other cortical veins and, when possible, the sylvian fissure and central sulcus. Skull X-rays were also taken, immediately post-operatively. We used post-implantation, pre-resection 3D CT in order to visualize the implanted subdural electrodes, and pre-implantation 3D MRI reconstructions to obtain surface renderings of the cortical gyri. These two images underwent 3D reconstruction and coregistration (Automated Image Registration, UCLA) to show the location of the grid electrodes with respect to gyral anatomy. In order to verify the accuracy of the coregistration, estimated by Woods *et al*. (1993) to be within 3 mm, we applied multi-modal fiducial markers (Neuromedical Supplies, Sterling, Va., USA) to a cadaver brain, and attached a subdural grid identical to that used in our clinical subjects. We then coregistered CT and MRI scans of the brain. This study indicated that our 3D CT/MRI coregistration methods localized subdural electrodes on the cortical surface anatomy with an approximate maximum error of $\pm$4 mm (Fig. 2). We considered this degree of error to be acceptable, given the relative coarseness of the anatomical correlations (e.g. gyri and sulci) made in this study.

Results

In order to investigate the utility of alpha and beta ERD as tools for functional brain mapping, we examined their

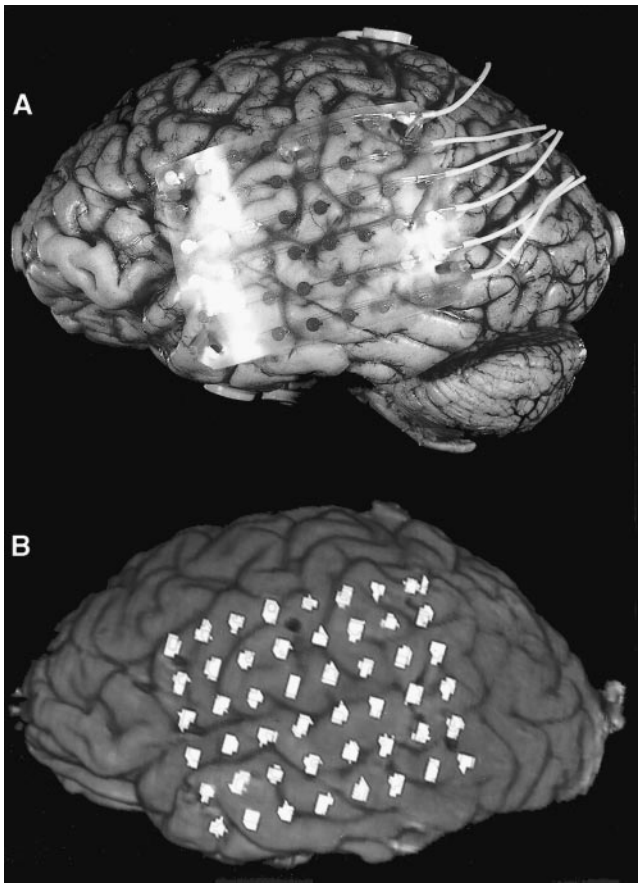

Fig. 2 (**A**) A photograph of a cadaveric brain with an electrode array attached to the surface with plastic pins. (**B**) Coregistration of the brain (from 3D MRI) and electrodes (from 3D CT). Note close correspondence with photograph.

topographical and temporal patterns during muscular contractions of different body parts in five different clinical subjects. Because coverage of sensorimotor cortex by subdural electrodes was most comprehensive in Subject 1B (second implantation of Subject 1), we report our results with this subject in the greatest detail.

Subject 1 (A and B)

Alpha ERD and somatotopy

For each body part tested in Subject 1B statistically significant ERD occurred in the majority of recording electrodes (Fig. 3); it involved a wide cortical region in and around sensorimotor cortices. Therefore, the presence of significant alpha ERD alone was not a sufficient criterion to distinguish a somatotopically distinct pattern for different body parts. However, examination of the time course of ERD revealed more specific spatiotemporal patterns for each body part. In particular, we observed two different temporal patterns of alpha ERD—sustained and transient, which appeared to correspond to relatively distinct spatial patterns.

When alpha ERD occurred continuously from its onset to the end of the post-stimulus time period (2 s) of spectral analysis, we called it sustained. The temporal pattern of sustained alpha ERD thus corresponded to the subject's motor response, which was also sustained throughout the post-stimulus period of analysis. The spatial pattern of sustained alpha ERD was typically concentrated over somatotopically specific regions of sensorimotor cortex, adjacent to the central sulcus. Alpha ERD also occurred in a transient temporal pattern, operationally defined herein as any statistically significant ERD (lasting for at least three consecutive epochs) ending before the end of the 2-s period of analysis (usually within 1600 ms after stimulus presentation in this subject). Transient alpha ERD occurred in spatial patterns that typically involved cortical regions immediately adjacent to, but outside the pre- and post-central gyri, or regions within them that corresponded to body parts not involved in the motor task.

For each body part, the topographical pattern of alpha ERD was nearly identical under the decision and the simple conditions, indicating a good degree of reproducibility (cf. Figs 3 and 4). Tongue protrusion produced the same basic patterns of ERD during two decision conditions (right and left) and one simple condition (not shown). The only notable exception to this reproducibility was that under the decision condition, tongue and arm movements (Fig. 4) produced sustained alpha ERD over parasagittal regions of perirolandic cortex (channels 33, 41, 42 and 43)—near putative leg representations—whereas contraction of these muscles during the simple task produced only transient alpha ERD at these electrodes (Fig. 3).

Other parameters of alpha ERD, such as the latency, slope and magnitude of power suppression, appeared to complement the distinction between sustained and transient ERD, but specific quantitative analyses of these parameters were deferred for future investigations. Nevertheless, the shortest latency, steepest slope and maximal magnitude of ERD often appeared to coincide with sustained alpha ERD (Figs 3 and 4). These different measures of the degree of ERD appeared to behave similarly, perhaps providing different perspectives of the same phenomenon, i.e. reactivity in the 8–13 Hz band in association with cortical activation. In Subject 1B, the magnitude of alpha ERD appeared to define a somatotopic pattern of putative cortical activation, during both the early and the late phases of task execution (Fig. 5). However, in other subjects (see Subjects 2–5 section below) the magnitude of alpha ERD was not specific for mapping different body parts until the late phase of task execution, when a distinction between sustained and transient ERD could be made. Likewise, although the onset latency of alpha ERD often appeared to be shortest in electrodes where alpha ERD was sustained, it was not by itself sufficient to define a somatotopic pattern for different body parts consistently in any of our subjects. For this reason, we simply summarized the spatial locations of sustained alpha ERD in many of the illustrations of our results. In addition, we did not observe a consistent relationship among subjects between the onset latencies of alpha (or beta) ERD and the EMG-recorded motor responses.

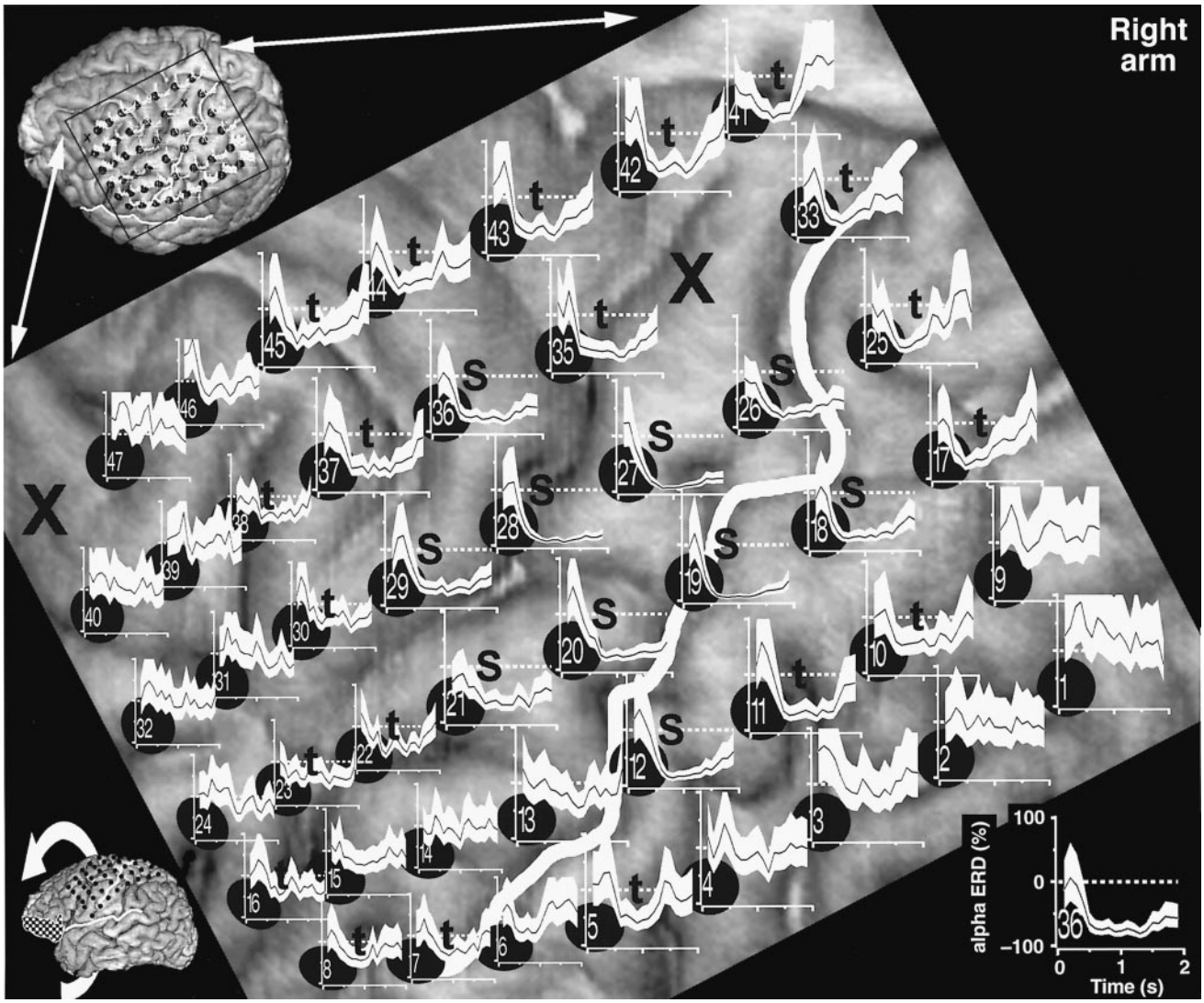

Fig. 3 Plots of alpha ERD, for each subdural electrode over the left frontoparietal cortical regions, in Subject 1B during fist-clenching with the right (contralateral) arm during the simple visual–motor task. The horizontal axis shows post-stimulus time in seconds, and data points occur every 100 ms. Alpha ERD (geometric mean of the percentage change in post-stimulus alpha power from baseline) is plotted as a black line, and white areas above and below it represent 95% confidence intervals. 'S' denotes sustained alpha ERD, and 't' denotes transient alpha ERD (in at least three consecutive epochs). Plots are superimposed on the cortical surface anatomy of this subject, rendered from a 3D MRI (see brain in lower left corner), which is rotated laterally (see brain in upper left corner) to expose the entire array of electrodes. Solid white lines in insets demarcate the sylvian fissure and central sulcus, and the chequered area in the lower left brain image demarcates the maximal surface projection of an underlying region of frontal encephalomalacia. 'X' denotes an electrode site in which the ECoG could not be recorded reliably.

Further analyses of ERD onset latency may require a temporal resolution finer than that used for this study.

Alpha ERD has been shown by others to occur during the preparation for self-paced movements (Pfurtscheller and Berghold, 1989). During our externally paced visual–motor task, a similar phenomenon could have occurred after onset of the visual stimulus, in preparation for the muscle contraction. This might have contributed to the rather diffuse and non-specific pattern of alpha ERD in the early stages of muscle contraction. In addition, it is conceivable that a similar preparatory ERD could have occurred in conjunction with the relaxation of muscles. To study this possibility we performed additional analyses of ERD on a subset of data (right hand fist-clenching under the simple condition), extending the period of analysis to 5.8 s after stimulus onset, just short of the baseline period for the next trial. Although we sometimes observed an additional ERD in association with muscle relaxation, it did not occur until after the offset of the visual stimulus, i.e. 3 s after onset of the visual stimulus, and it ended before the baseline period of the next trial.

In Subject 1B, we saw a brief (500–900 ms) augmentation of alpha power (alpha ERS) in the hand area (see electrodes 27 and 28 in Fig. 2) during contralateral leg movements (in the decision condition only). However, in our other subjects, we did not see early alpha ERS over arm areas during tongue

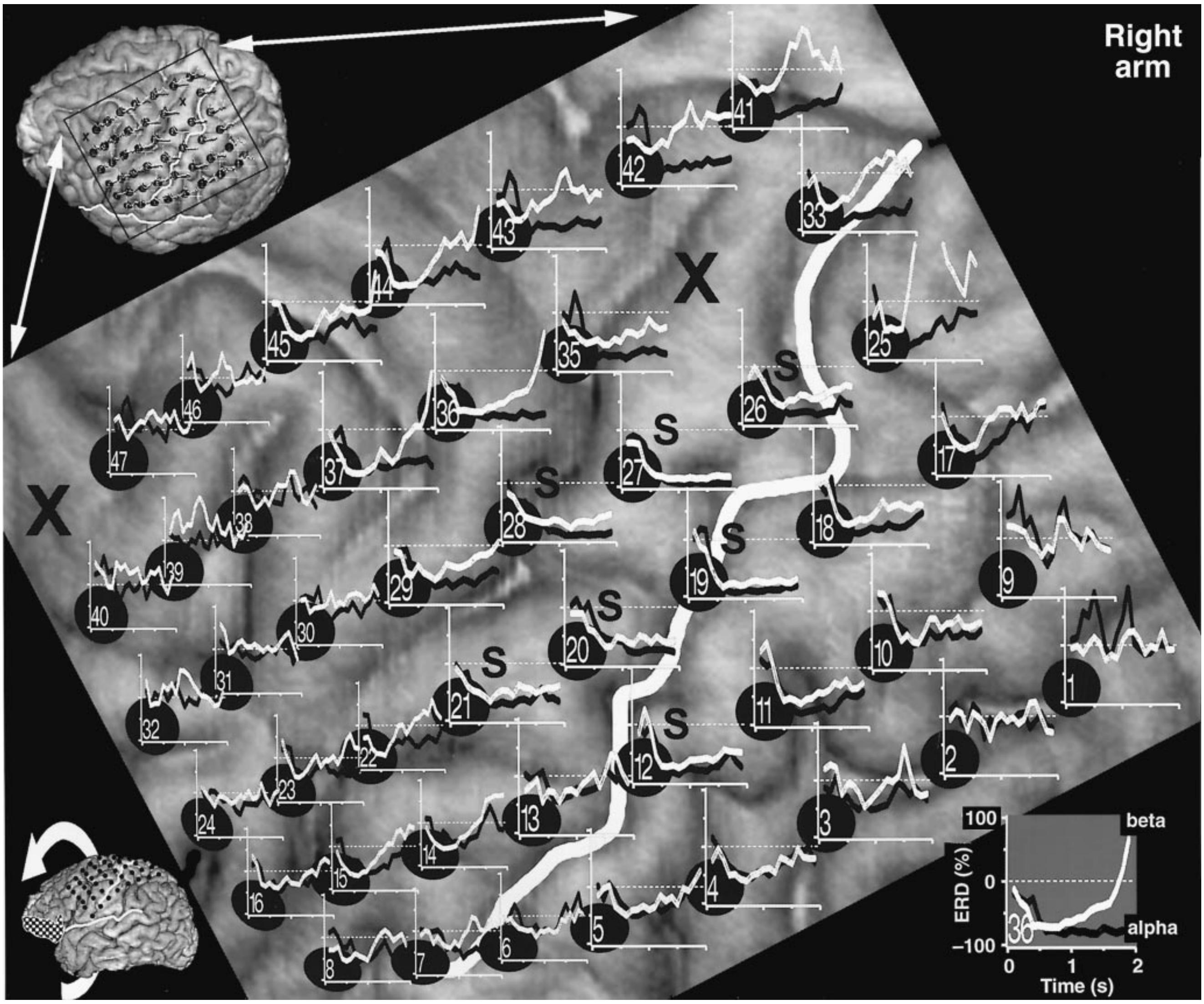

Fig. 4 Plots of alpha (thick black line) and beta (15–25 Hz, thick white line) ERD, in Subject 1B during right (contralateral) fist-clenching under the decision condition. 'S' indicates that both alpha and beta ERD are sustained. Plot axes and anatomical markers are the same as in Fig. 2; confidence intervals are not shown.

or leg movements, as predicted from previous studies of alpha ERS (Pfurtscheller, 1992; Pfurtscheller and Neuper, 1994). This could have resulted from differences in the designs of our respective motor tasks. Likewise, when we extended the time period of analysis, we only rarely observed alpha ERS in association with muscle relaxation, but when we did, it did not continue into the baseline period of the next trial.

Alpha ERD with ipsilateral limb movement

In Subject 1B, power suppression in the 8–13 Hz band also occurred during ipsilateral (left) limb movements. During ipsilateral fist-clenching under the decision condition, alpha ERD occurred over putative arm regions of sensorimotor cortex, but it was sustained to 2 s in only one electrode (see asterisk in Fig. 6). In the electrodes where contralateral fist-clenching produced the maximum sustained ERD under the decision condition, ipsilateral fist-clenching produced only transient ERD. Furthermore, in most electrodes the ERD associated with contralateral fist-clenching appeared to begin earlier or coincide with onset of the EMG-recorded motor response (geometric mean, 659 ms; see Crone *et al.*, 1998), but the ERD associated with ipsilateral fist-clenching appeared to begin later than the motor response onset (618 ms). This finding may parallel those obtained with scalp EEGs using self-paced movements, in which there is bilateral ERD at movement onset and thereafter, but only contralateral ERD during movement preparation (Pfurtscheller, 1989). During the simple condition, ipsilateral fist-clenching also produced a transient ERD. Although there was also a similar trend in the latency of ERD onset, i.e. shorter for contralateral than for ipsilateral fist-clenching, the latency differences were less impressive, perhaps related to the shorter motor response latency and the lack of a decision component in the task.

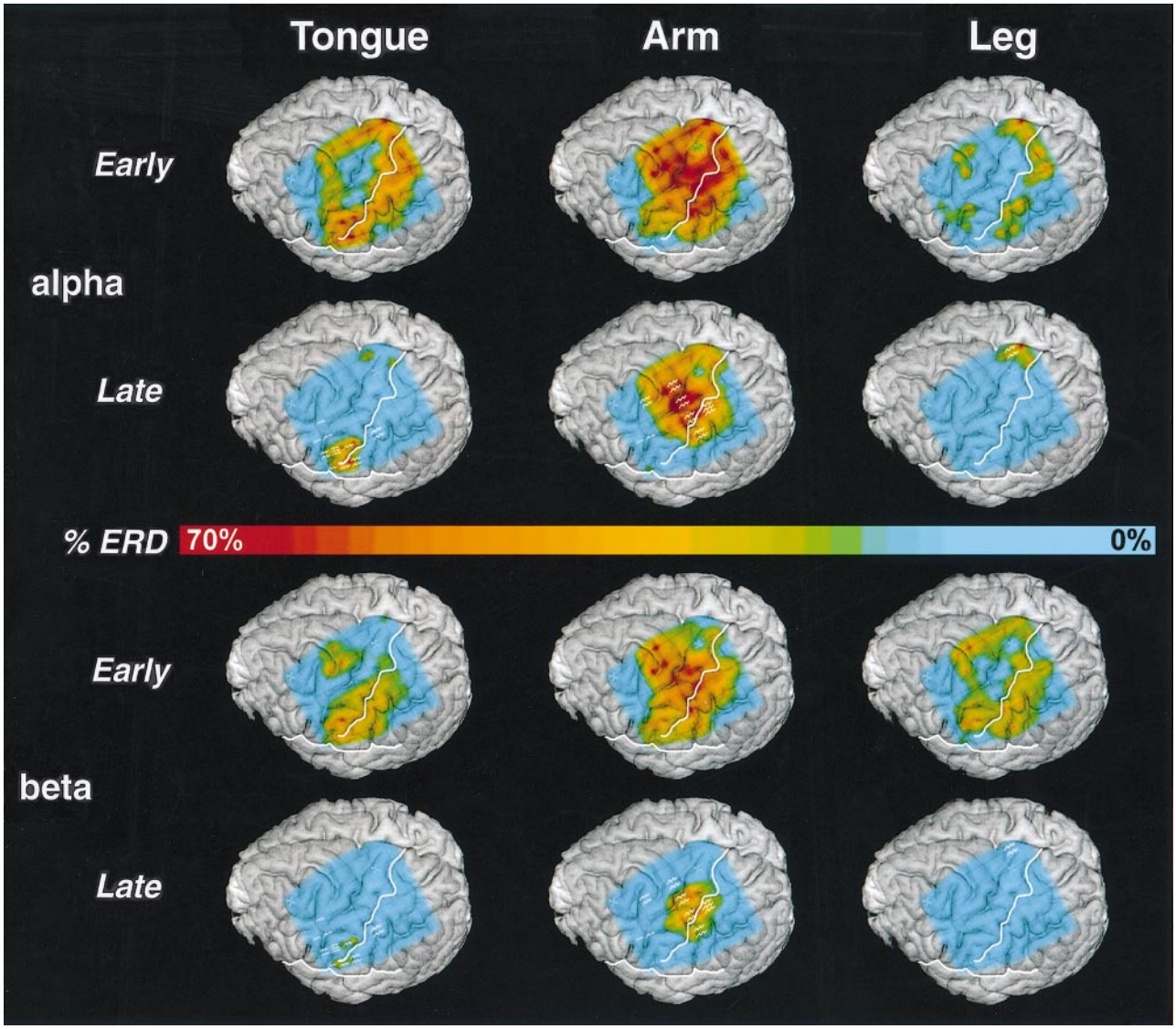

Fig. 5 Colour plots of alpha and beta (15–25 Hz) ERD over left frontoparietal cortex of Subject 1B during early and late post-stimulus time periods, illustrating the distinction between transient (early) and sustained (late) power changes. Early ERD was calculated by averaging the percentage (geometric mean) ERD for three 200-ms post-stimulus epochs centred at 600, 700 and 800 ms. Late ERD was calculated by averaging the percentage ERD in the 1700, 1800 and 1900-ms epochs. Separate plots are shown for each body part under the decision condition, i.e. during tongue protrusion, right (contralateral) fist-clenching and right foot dorsiflexion. ERD measures at each electrode (original values at thin line crossings) were used for interpolation with an inverse-distance weighting algorithm to produce a smooth colour map. Lightning bolt icons denote electrical stimulation-induced disruption of motor function in the body part (tongue, right arm or right leg) indicated at the top of the column: one bolt = slowing or cessation of ongoing motor activity (wiggling tongue, fingers or toes); two bolts = involuntary motor response (e.g. posturing); and three bolts = clonic muscular contractions. Brain orientation and anatomical markers are as in Fig. 3. Faded 'x' denotes electrode site in which ECoG could not be recorded reliably.

In contrast, ipsilateral foot dorsiflexion produced sustained alpha ERD over parasagittal perirolandic cortex, during both the decision and the simple conditions (Fig. 7). In this case, the latency and magnitude of ERD during ipsilateral and contralateral limb movement were not noticeably different. In the decision task, the onset of ERD associated with both contralateral and ipsilateral foot dorsiflexion appeared to occur before their motor response onsets (660 and 676 ms, respectively), but in the simple task alpha ERD began at roughly the same time as movement onset in either the contralateral or ipsilateral foot. Cortical stimulation in this region (see under Leg in Fig. 5), however, produced only a contralateral thigh movement.

Beta (15–30 Hz) ERD and ERS

In Subject 1B, the spatial distribution of power changes in the 15–25 Hz and 20–30 Hz bands, corresponding to the beta band, were similar to power changes in the 8–13 Hz band, but their time courses were noticeably different. In

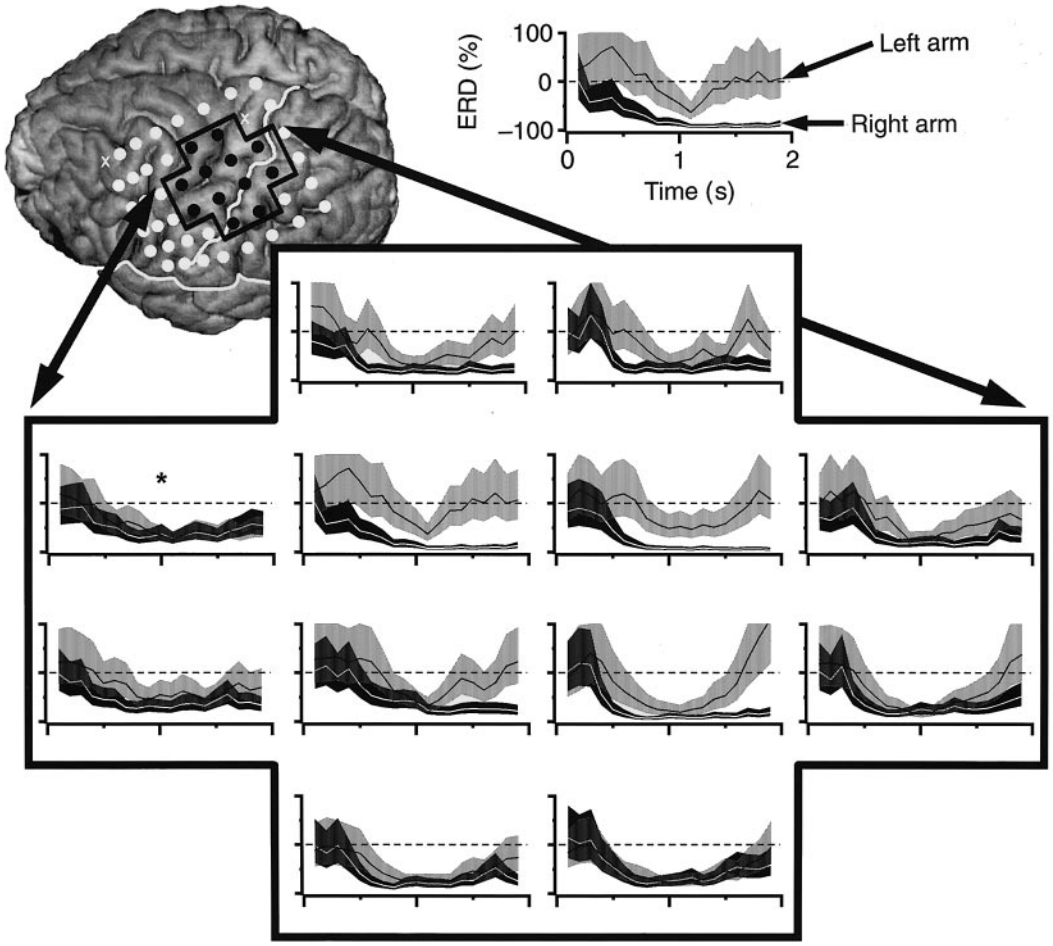

Fig. 6 Plots of alpha ERD over the left frontoparietal cortex of Subject 1B during fist-clenching with the right (contralateral) arm (white line = mean, black areas = 95% confidence intervals) and with the left (ipsilateral) arm (black line = mean, grey areas = 95% confidence intervals) under the decision condition. Note that alpha ERD is sustained in all electrodes during contralateral fist-clenching but transient in all but one (asterisk) during ipsilateral fist-clenching.

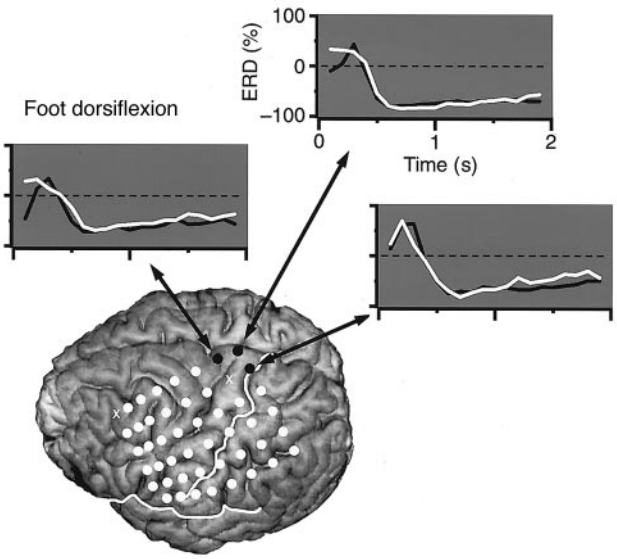

Fig. 7 Plots of alpha ERD over the left parasagittal cortex of Subject 1B during right/contralateral (black line) and left/ipsilateral foot dorsiflexion (white line) under the simple condition.

particular, beta ERD typically began somewhat earlier than alpha ERD, and for many cortical regions beta ERD had less magnitude (Fig. 4). The greatest consistency between alpha and beta ERD occurred in those electrodes nearest the central sulcus, where both were sustained and had their greatest magnitude (Fig. 5). Examination of transient beta ERD during muscle contractions in different body parts did not reveal any more of a somatotopic pattern than transient alpha ERD. In general, beta ERD was no more prevalent anterior to the central sulcus than it was posterior to the central sulcus, as suggested by other workers (Jasper and Penfield, 1949; Salmelin and Hari, 1994) using different motor tasks.

Several findings suggested that the topographical pattern of beta ERD might be more specific, somatotopically, for different body parts. Unlike alpha ERD, beta ERD was not sustained over parasagittal perirolandic cortex during arm (Fig. 4) or tongue (not shown) movements under the decision conditions. In addition, beta ERD was not sustained over parasagittal regions during ipsilateral leg movements, as was the case for alpha ERD (Fig. 7). Furthermore, during contralateral arm movements, sustained beta ERD occurred over a smaller region of sensorimotor cortex (electrodes 19 and 27) than sustained alpha ERD (Figs 4 and 5).

On the other hand, several findings suggested that, although beta ERD may be a more somatotopically specific measure of sensorimotor activation than alpha ERD, either our methods were less capable of detecting beta ERD, or beta ERD was less capable of detecting functional activation than alpha ERD. For example, beta ERD was not sustained during tongue or foot movements under either the simple or the decision condition (Fig. 5). Under these same task conditions, alpha ERD was sustained over the appropriate regions, albeit with less magnitude than during arm movements. The lack of sustained beta ERD might have occurred because the electrode array did not cover the most active regions of sensorimotor cortex representing tongue and foot movements, and/or because movement of these body parts did not require, or produce, as much cortical activation as movement of the arm. The former explanation seems less likely because cortical stimulation produced a considerable number of tongue motor responses in electrode sites where tongue protrusion produced only transient beta ERD (Fig. 5). An alternative explanation is that the neurophysiological mechanisms underlying beta ERD are capable of habituation in the setting of continued cortical activation.

We also observed a rebound of beta power, similar to the ERS described by Pfurtscheller *et al*. (1996), in which an overshoot of power above baseline occurs after recovery from beta ERD (Fig. 4, electrodes 14, 25, 33, 36, 37, 41, 44 and 45). In the experiments of Pfurtscheller *et al*. (1996), the motor output (button-pushing) was very brief, and beta ERS was seen only afterwards. However, we observed beta ERS during the sustained motor response. Similar to the findings by Pfurtscheller (1981), recovery and rebound of beta power typically occurred earlier than the recovery, or rare rebound, of alpha power. With further inspection of the temporal course of beta ERS (not shown), we found that for each body part, it occurred earlier for the simple condition than for the decision condition, particularly in those electrodes anterior to the central sulcus, with latency differences similar to those for the onset of EMG activity under these two task conditions.

In our experiments beta ERS often occurred outside, or on the outskirts of, the pre- and post-central gyri. In addition, beta ERS did not coincide with the maximum of alpha ERD, and it did not occur in a particularly somatotopic pattern. Based upon scalp EEG recordings, Pfurtscheller *et al*. (1996) concluded that beta ERS occurs anterior to the central sulcus. In Subject 1B, beta ERS occurred in more electrodes anterior to the central sulcus, but our ECoG recording did not cover as much territory posterior to the central sulcus. Interestingly, the earliest and highest rebound occurred in one electrode situated just posterior to the central sulcus, regardless of which body part was moved or which task condition (decision or simple) was used (electrode 25 in Fig. 4).

When we extended the post-stimulus period of ECoG spectral analysis in a subset of data from Subject 1B (see Alpha ERD and somatotopy section), we also observed a brief beta ERS in association with muscular relaxation in a few of the recording channels. However, this ERS did not continue into the baseline period of the next trial.

Reproducibility of ECoG spectral analysis at different times

Subject 1 underwent two different implantations of subdural electrodes (referred to as 1A and 1B), separated by 6 months, for invasive video/EEG monitoring and functional mapping with cortical electrical stimulation. This allowed us to examine the reproducibility of ECoG spectral analysis within the same individual subject over a significant span of time. Comparison of the maps generated by ECoG spectral analysis on two separate occasions, using the decision condition of the visual–motor task (the simple condition was not used with 1A), revealed a match which was not perfect but which, overall, was quite good considering the difference in electrode placements (Fig. 8).

Comparison of ECoG spectral analysis and cortical electrical stimulation maps

Cortical electrical stimulation was performed in this and our other subjects for the clinical purpose of pre-resection functional localization. Although the effect of this technique on cortical function is still incompletely understood (Li and Chou, 1962), at an operational level it appears capable of producing a hyperacute, reversible disruption of normal motor function (Landau *et al*., 1965) which is clinically useful (Uematsu *et al*., 1992*a*). Cortical electrical stimulation of sensorimotor cortex may result in either negative motor symptoms, presumably due to cortical deactivation, or

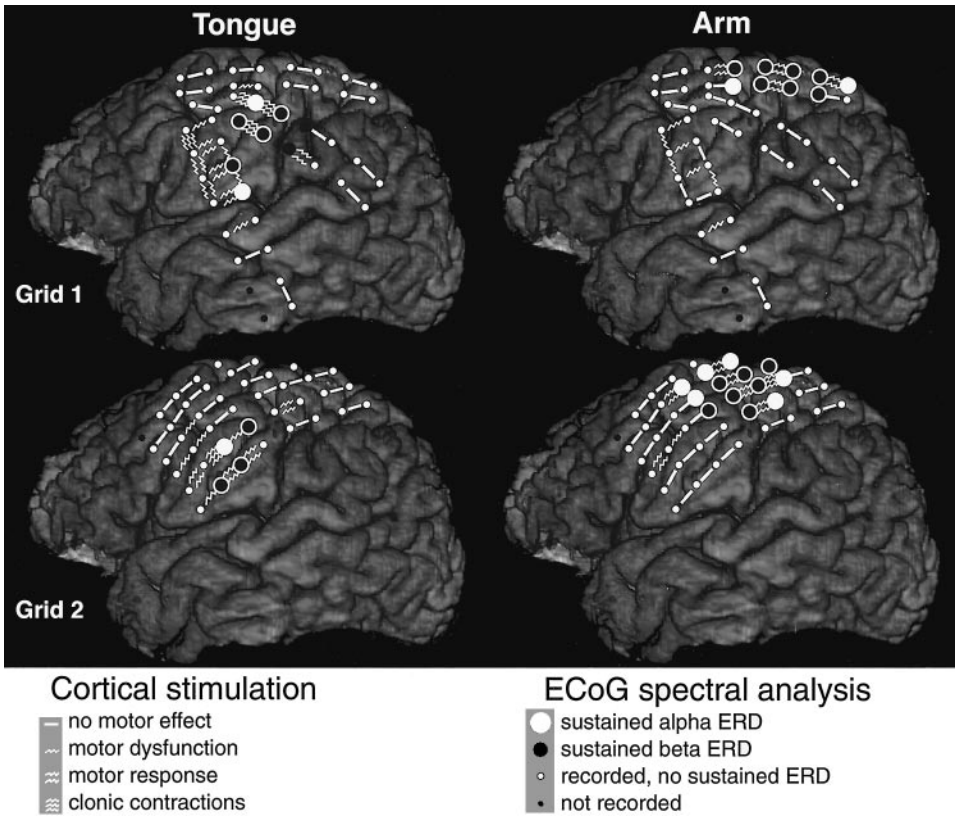

Fig. 8 Maps of ECoG spectral analysis and cortical stimulation results during two different implantations of grid electrodes in Subject 1B. Both ECoG spectral analysis and cortical stimulation maps of tongue (left column) and arm (right column) function are are shown for both the first (top row) and second (bottom row) grid placements. Symbols refer to sustained alpha and/or beta ERD. Symbols representing the results of cortical electrical stimulation are the same ones used in Fig. 5.

positive symptoms, perhaps due to abnormal cortical activation. In all our subjects cortical electrical stimulation in and adjacent to the paracentral cortex produced a variety of symptoms indicating disruption of motor function. Because cortical stimulation was not expected to (and did not) reproduce the movements used in the ECoG spectral analysis of visual–motor tasks, any stimulation-induced change in the movement of one or more limbs, or in the tongue, was operationally defined as a disruption of motor function in that body part. However, the production of involuntary movements (positive responses), particularly clonic movements (not associated with after-discharges), was considered more localizing than slowing or arrest of voluntary movements (negative responses), which have been found in previous studies to occur outside the primary sensorimotor cortices, with a relatively non-specific distribution along the central sulcus (Nii *et al*., 1996). We therefore used a hierarchical categorization of the effects of cortical stimulation on motor function (e.g. Figs 5 and 8): no motor effect; motor dysfunction only (negative responses); motor responses (positive responses) without clonic contractions; and motor responses with clonic contractions. Note that positive motor responses, with and without clonic contractions, were often accompanied by motor dysfunction (slowing or arrest of voluntary movements), but motor dysfunction alone (a negative response) was, by definition, not accompanied by any positive motor response.

As in previous studies with cortical stimulation mapping (Uematsu *et al*., 1992*a*; Urasaki *et al*., 1994; Nii *et al*., 1996), in Subject 1B there was some overlap in the effects of cortical stimulation on tongue and hand motor function (Fig. 8). With some electrode pairs, cortical stimulation produced a positive motor response in one body part and a negative response in the other body part, but with other electrode pairs cortical stimulation produced positive motor responses within both body parts. In addition, cortical stimulation produced positive motor responses in regions both anterior and posterior to the precentral gyrus, and in many electrode pairs cortical stimulation produced both sensory (not shown) and motor responses, as shown in previous studies (Uematsu *et al*., 1992*a*, *b*). Nevertheless, the cortical stimulation maps that were obtained from the two implantations of Subject 1 were very similar, although not identical, due in part to the different placement of electrodes (Fig. 8).

Comparison of the cortical stimulation maps with those of ECoG spectral analysis for different body parts revealed a moderate degree of congruence between these two methods of functional brain mapping (Figs 5 and 8). Sustained alpha and beta ERD were usually recorded from electrodes at

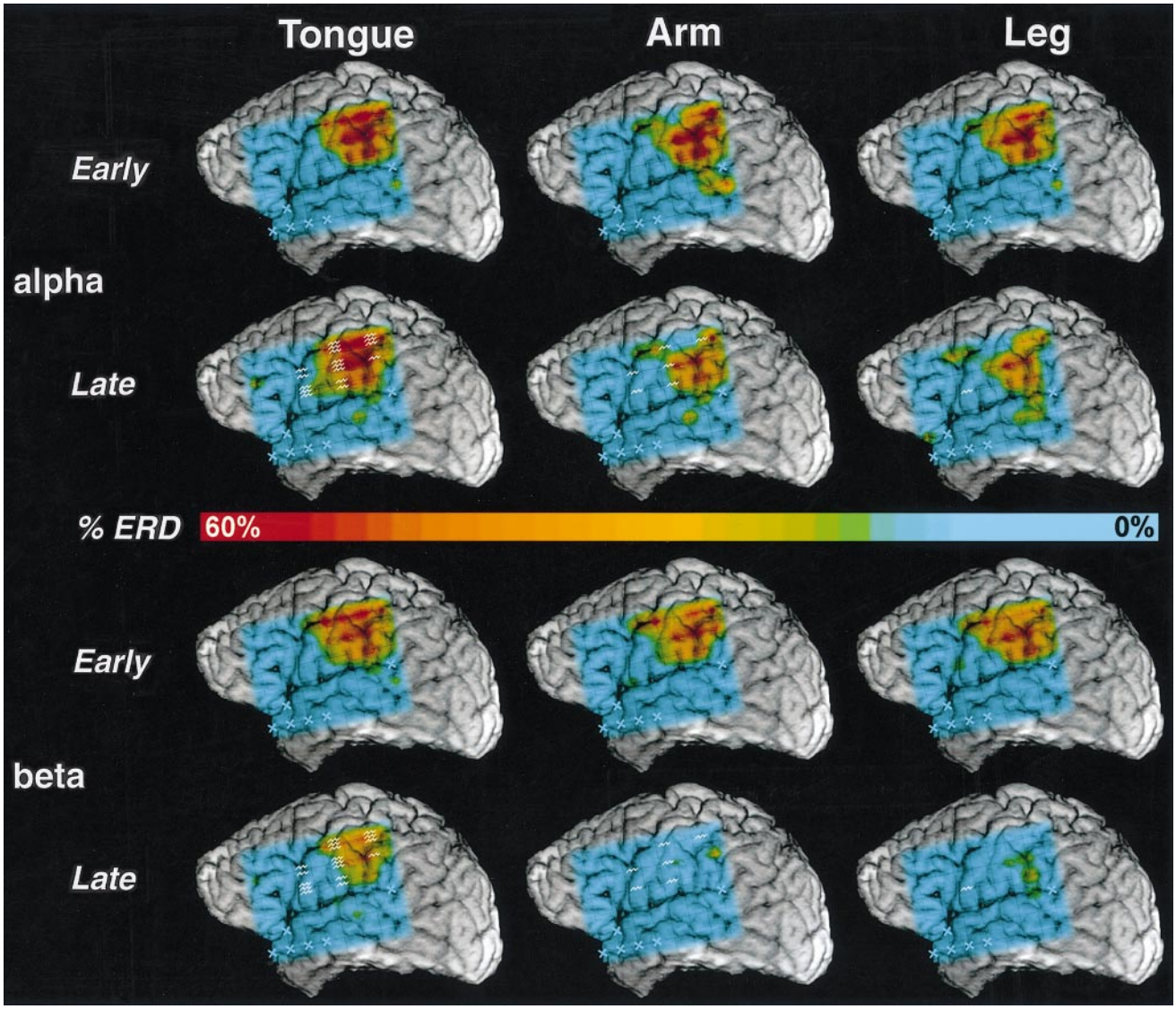

Fig. 9 Colour plots of alpha and beta ERD, as in Fig. 5, for Subject 4. Lightning bolts represent motor responses with cortical stimulation as in Figs 5 and 8.

which cortical stimulation produced some kind of disruption of motor function in the same body part. This disruption was more often evidenced by a positive response than by a negative response alone, but an exclusive relationship between ERD and positive or negative responses was not observed. Cortical stimulation produced negative, and sometimes positive, motor responses in regions outside the paracentral cortex, in which sustained ERD was not observed. Significant, but transient alpha ERD occurred in many, but not all, of these other cortical regions (see 'Early' spectral maps in Fig. 5).

Similar relationships between cortical stimulation, alpha and beta ERD and somatotopy were also observed in the other subjects we tested (see Figs 9, 10, 11 and 12).

Subjects 2–5

Subdural ECoG recordings were made in four other subjects using the same visual–motor decision task used for Subject 1 (Table 1). All these subjects were tested with the decision condition, but none were tested with the simple condition, which was used only with Subject 1B. In Subjects 4 and 5 the task involved moving one of four different body parts (25 trials each): tongue, face, arm or leg. However, because cortical stimulation was not used to study facial movements, the results of ECoG spectral analysis during eye-winking (similar to those for tongue protrusion) are not presented in this paper. For these and other reasons (see Experimental procedures in the Methods section above), only tongue and hand movements (50 trials each) were used with Subject 2. With Subject 3, only tongue, hand and leg movements were used as with Subject 1.

Somatotopy: transient versus sustained alpha ERD

With a few notable exceptions (see below), our results in Subjects 2–5 were consistent with those detailed for Subject 1.

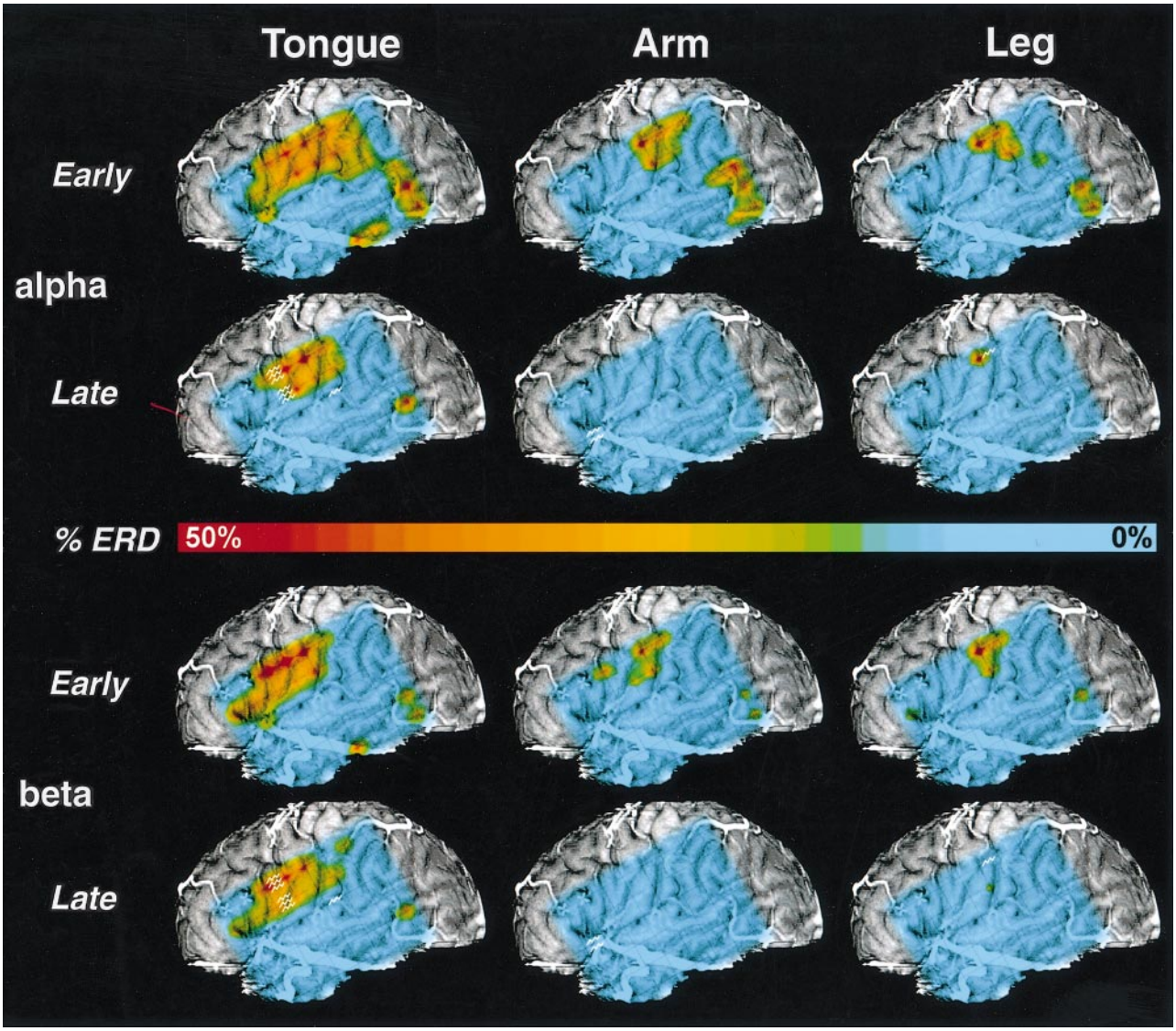

Fig. 10 Colour plots of alpha and beta ERD, as in Fig. 5, for Subject 5. Lightning bolts represent motor responses with cortical stimulation as in Figs 5 and 8.

As in Subject 1, statistically significant alpha ERD occurred over putative sensorimotor cortices in all our other subjects during the visual–motor task. Similarly, during the early phases of the subjects' motor responses, the movement of different body parts produced alpha ERD in similar, overlapping topographical patterns, without a clear somatotopic distinction between different body parts. For example, in Subjects 4 and 5 electrode coverage was limited to the most inferior/lateral aspects of sensorimotor cortex (Figs 9 and 10). In these regions the magnitude of early alpha ERD was greatest during tongue protrusion, but ERD of nearly equal magnitude was found in a similar distribution during the early phases of fist-clenching and foot dorsiflexion. However, as in Subject 1, closer inspection of the time course of alpha ERD in these subjects revealed that when it was sustained throughout the late phases of the motor response, its spatial pattern was much more discrete and somatotopically specific, in this case for the tongue.

In Subject 2, tongue protrusion and fist-clenching produced sustained alpha and beta ERD in non-overlapping patterns. However, tongue protrusion produced sustained alpha ERD in only one electrode (Fig. 11). This electrode may have been situated on the superior margin of the tongue area of the sensorimotor homunculus. However, we can only speculate about this subject's underlying functional anatomy, since the distribution of recording electrodes was sparse and did not include parasylvian cortex, as in the other subjects.

Sustained alpha ERD allowed the differentiation between movements of different body parts in all but one of our subjects (Figs 8, 9, 10 and 11). In Subject 3 (Fig. 12) sustained alpha suppression was observed over a wide area during both tongue and hand movements. Although better

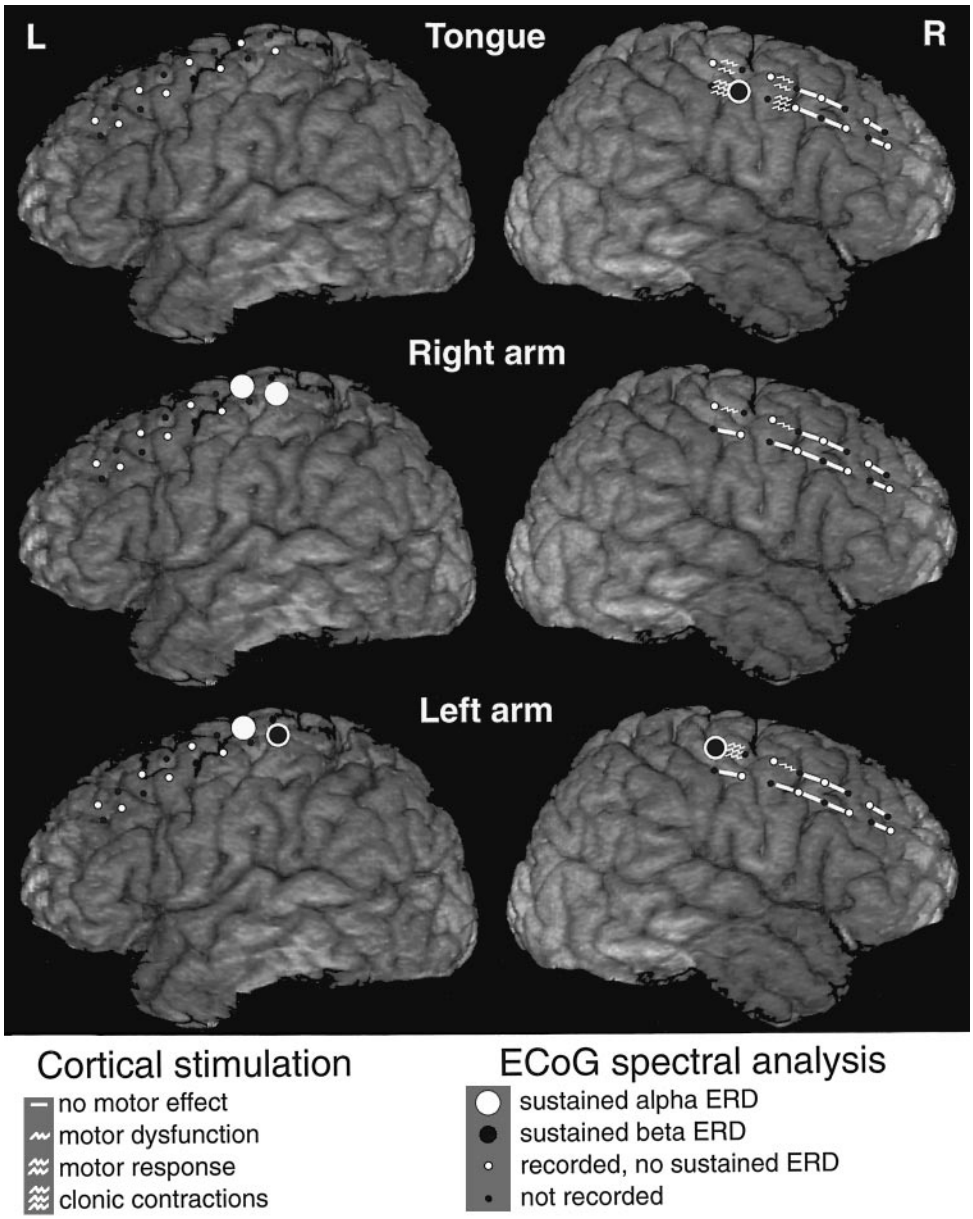

Fig. 11 Maps of ECoG spectral analysis and cortical stimulation results in Subject 2 during tongue protrusion, right fist-clenching and left fist-clenching under the decision condition. Foot dorsiflexion was not used with this subject. L = left hemisphere; R = right hemisphere.

coverage of the sensorimotor strip with a more evenly spaced array of recording electrodes might have allowed a clearer somatotopic distinction to be made, in this case even sustained alpha and beta ERD were not able to discriminate between tongue and hand movements.

Relationship of ERD to the central sulcus

The spatial distribution of alpha ERD with respect to the central sulcus appeared to vary depending upon the subject. In Subject 4 the spatial distribution of alpha ERD was mostly posterior to the central sulcus (Fig. 9). In two other subjects (1 and 5) it was most robust over precentral regions (Figs 5 and 10, respectively). In Subject 5 (Fig. 10) the maximum magnitude of beta ERD was ~1 cm anterior to that of alpha suppression during the tongue task. However, in our other subjects there was no compelling evidence for a pre- or post-central predilection for alpha ERD, nor was there a consistent distribution of beta ERD anterior to the central sulcus, as suggested by others (Salmelin and Hari, 1994).

Ipsilateral ERD

At least transient alpha ERD occurred with ipsilateral limb movements in all subjects in whom both contralateral and ipsilateral limb movements were tested (Subjects 1, 2, 3 and

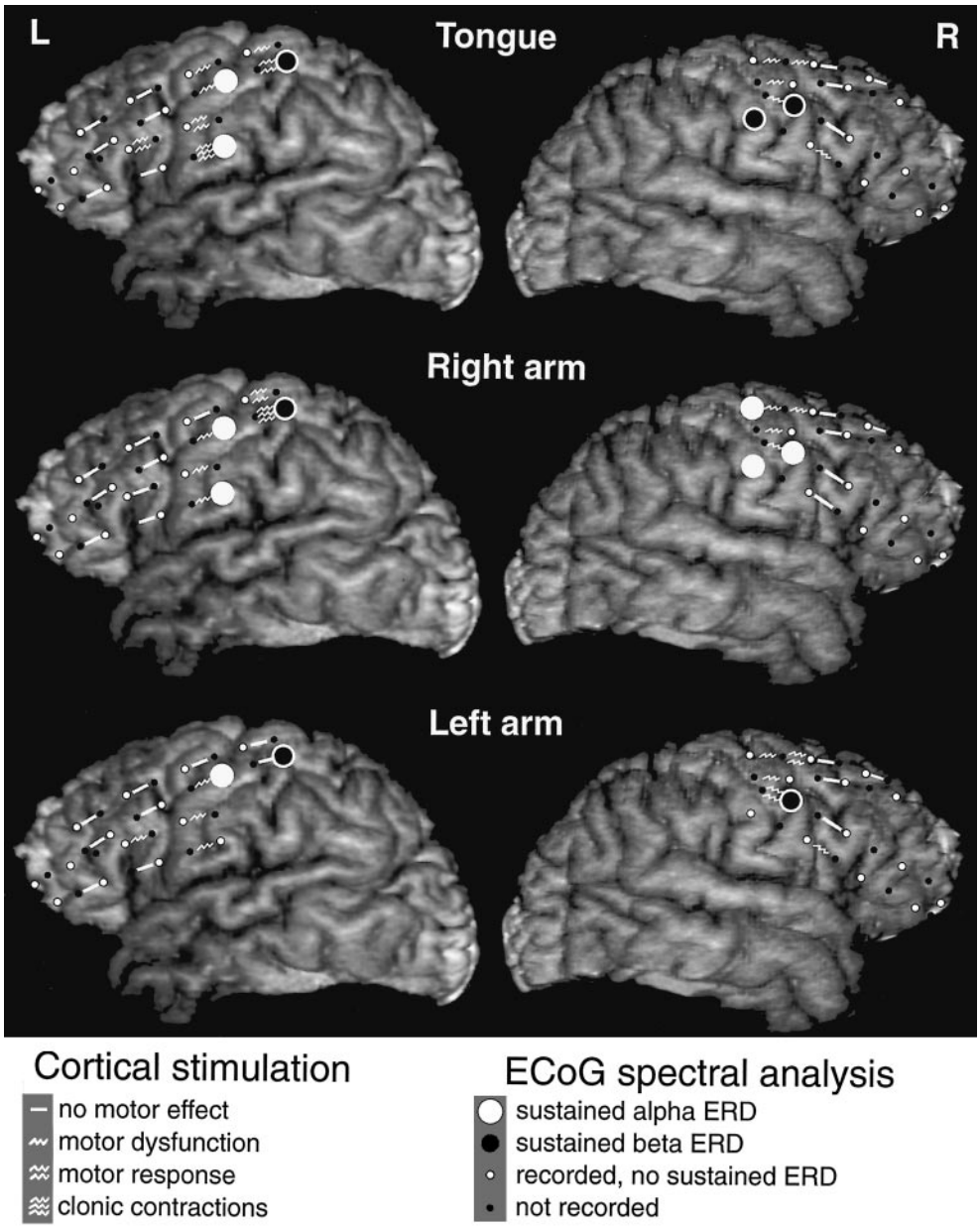

Fig. 12 Maps of ECoG spectral analysis and cortical stimulation results in Subject 3 during tongue protrusion, right fist-clenching and left fist-clenching under the decision condition. Foot dorsiflexion was also used in this subject, but it did not produce sustained alpha or beta suppression in the visible electrodes. L = left hemisphere; R = right hemisphere.

5). In Subject 5, alpha ERD was only transient (not sustained) during ipsilateral fist-clenching, similar to that in Subject 1. In Subjects 2 and 3 bilateral putative hand regions were covered by recording electrodes. In Subject 3 alpha ERD was sustained over putative hand regions in both hemispheres during right and left fist-clenching (Fig. 12). It is interesting to note that both left and right intracarotid amobarbital injection suppressed speech function in this subject, suggesting some degree of mixed dominance, even though he was right-handed. In Subject 2, left fist-clenching produced sustained alpha ERD and beta ERD over bilateral sensorimotor cortices, but right fist-clenching produced sustained alpha ERD only over contralateral sensorimotor cortex (Fig. 11). Although it has been shown that alpha and beta ERD in right-handed subjects is more lateralized prior to right finger movements than prior to left finger movements (Stancak and Pfurtscheller, 1996*a*, *b*), the sparse electrode coverage in our Subject 2 did not allow us to examine this particular issue.

Beta ERD

Similar to our findings in Subject 1, beta ERD in our other subjects was typically less sustained than alpha ERD, and

frequently rebounded above baseline. When these timing differences were taken into account, the spatial distribution of maximal beta ERD was found to coincide roughly with that of maximal alpha ERD. Early, transient suppression of both alpha and beta frequencies occurred over premotor cortical regions (anterior to the precentral sulcus) in Subjects 1 (Fig. 3) and 2, but this was not a consistent finding.

Additional findings

In two subjects (4 and 5) alpha suppression also occurred over posterior temporal and temporal-occipital regions, as well as over inferior parietal regions, early during the course of task execution, perhaps reflecting processing of the visual stimulus and selection of the appropriate body part for movement, respectively.

Discussion

Overall strategy

The goals of this study were to validate alpha and beta ERD as indices of cortical activation in ECoG recordings, and to investigate their utility for functional localization of sensorimotor cortex in a clinical setting. The most important obstacle to our first goal was the lack of any verifiable gold standard as to where and when task-related cortical activation should occur—a problem we suspect is present in any attempt to verify a correlative index of brain function. Therefore, we chose to study a region of the brain in which the functional anatomy is already relatively well known and in which functional activation can be verified via a direct behavioural response. In addition, we compared the results of ECoG spectral analysis with those of cortical stimulation in each patient in order to account for any individual differences in functional neuroanatomy.

The visual–motor task used in this study differed from the self-paced button-pushing task used by other investigators. Our task called for a sustained isometric muscle contraction in response to a visual stimulus. By requiring a sustained motor response, we hoped to focus our spectral analyses more on execution of the motor response. In contrast, analyses of ERD during self-paced button-pushing have focused on planning of the motor response. Because we used the visual cue rather than the motor response as our temporal reference point for averaging the EEG power spectrum, the correlation of power changes with motor responses was free to vary, and the sustained course of motor responses allowed us to examine this correlation over a more extended time scale. We therefore expected that a different perspective on the correspondence between motor performance and ECoG spectral changes would provide additional information about the response properties of alpha and beta ERD, as well as the utility of these measures as tools for clinical functional localization.

Our methods for data reduction and statistical analysis also differed from those of others. Although these differences might have accounted for some of the variance between our results and theirs, the general agreement of our findings with those of others suggests that both alpha ERD and beta ERD are robust phenomena that are easily detectable, regardless of the exact methods used.

Alpha ERD

Consistent with previous findings by other investigators (Pfurtscheller, 1989; Toro *et al*., 1994) and ourselves (Crone *et al*., 1993), activation of sensorimotor cortex always produced statistically significant power suppression in the alpha (~8–13 Hz) band. Alpha ERD was present in all five of our subjects, suggesting that it is a relatively sensitive index of task-related cortical activity, particularly when recorded from the cortical surface. Although the neurophysiological mechanism of this phenomenon is not yet known, it has come to be called 'event-related desynchronization' (ERD), and here we have adopted Pfurtscheller's term.

Somatotopic specificity

In the present study statistically significant alpha ERD often occurred outside the paracentral cortex, in a pattern with considerable overlap between the putative somatotopic representations of different body parts. Nevertheless, by requiring a greater consistency between the temporal profile of alpha ERD and the temporal structure of our subjects' motor responses, a more somatotopic pattern of putative activation could often be delineated. In particular, alpha ERD appeared to be more somatotopically specific when it was sustained throughout the motor response. If a briefer motor response had been used, like that of many previous studies, it might have been more difficult to make these somatotopic distinctions using alpha ERD.

In addition to the temporal concordance between alpha ERD and motor performance, the magnitude of alpha ERD provided another means of examining its functional specificity. When its magnitude could be mapped with a 2D array, alpha ERD was typically maximal in the same regions where it was sustained, i.e. in somatotopically defined regions near the central sulcus. In Subject 1B the magnitude of alpha ERD in putative tongue and leg areas was significantly less than it was in arm areas. However, this could have been caused by either incomplete or off-centre coverage by the electrode array (especially over the putative leg area), or by less activation of these regions by our task. Also, we cannot rule out the possibility that less effort was expended during these particular movements.

The variability of the mapping of different body parts to the pre- and post-central gyri is well known (Penfield and Jasper, 1954; Nii *et al*., 1996). In our study, the distribution of alpha ERD along the sensorimotor strip also appeared to vary from subject to subject. For example, in the three

subjects in whom there were sufficient electrodes for such a determination, the distribution of maximal sustained alpha ERD during tongue protrusion was somewhat more ventral in Subject 5 than in Subject 4, and with Subject 1 it overlapped both. However, within this range of variability the cortical topography of sustained alpha ERD appeared to meet our expectations of somatotopy, although there were some important exceptions.

Exceptions to somatotopy

In Subject 1B, sustained alpha ERD occurred, albeit with lower magnitude, over parasagittal precentral cortex, near putative leg representations, during arm and tongue movements. Although studies with scalp EEGs (Pfurtscheller, 1989) have shown a broad distribution of alpha suppression over central and paracentral head regions during upper limb movements, we expected that ECoG recordings would allow sufficient spatial resolution to discriminate a limb-specific somatotopic pattern of alpha suppression. This apparent exception might have occurred because the parasagittal electrodes were close to the supplementary motor area, where somatotopic organization is probably more compressed (Orgogozo and Larsen, 1979; Kurata, 1992). Along these lines, it is interesting to note that this parasagittal activation was observed under the decision condition but not under the simple condition, suggesting selective activation, perhaps of supplementary motor area, by the decision component of the task.

In Subject 3 (Fig. 12), tongue protrusion and fist-clenching produced sustained alpha ERD in almost all the same electrodes. Even sustained beta ERD was not somatotopically specific in this subject. The limited coverage of recording electrodes and the use of an average reference might have caused inaccuracies in the spatial pattern of spectral changes in this subject. Nevertheless, the spatial pattern of sustained alpha and beta ERD was relatively consistent with cortical stimulation, which also suggested some degree of representational overlap for different body parts. Additional electrodes would probably have clarified this, but the choice, number and positioning of electrodes was perforce limited by the specific clinical situation of this subject.

In Subject 5, sustained alpha ERD occurred during foot dorsiflexion in one electrode that was more ventral than expected (Fig. 10). Sustained beta ERD did not occur here, which is perhaps consistent with the greater somatotopic specificity of this index (see below). However, cortical stimulation at this site caused slowing of toe movements, suggesting that this area might somehow participate in lower extremity motor function. Lower extremity responses to cortical stimulation have been found in this region in a few previously reported cases (Penfield and Boldrey, 1937; Lesser *et al*., 1984). The electrode in question appeared to be located on the anterior edge of the precentral sulcus, and might have corresponded to Brodmann area (BA) 6 or to a superiorly displaced secondary sensory area in this subject. This electrode was also on the edge of the subdural grid, raising the possibility that more anterior and superior cortical regions, perhaps including the inferior supplementary motor area, were really responsible for its recording and stimulation characteristics.

Along with the apparent exceptions to somatotopy noted above, we consistently observed alpha ERD in a non-somatotopic pattern during the early phases of our subjects' motor responses. This transient, diffuse alpha ERD could have resulted from non-specific activation of all somatotopic representations prior to settling on the particular body part called for by the visual stimulus. However, this phenomenon often continued beyond the onset of the motor response, and a similar transient response was still seen in Subject 1B when only one body part was used in the simple condition (Fig. 3).

Studies of neural plasticity have suggested a significant degree of somatotopic overlap in the sensorimotor cortex. The representation of body parts is capable of reorganization in response to peripheral nerve section (Kaas *et al*., 1983; Wall *et al*., 1986), sensory stimulation (Wang *et al*., 1995), intracortical microstimulation (Recanzone *et al*., 1992*a*), cortical bicuculline iontophoresis (Jacobs and Donoghue, 1991), and motor (Nudo *et al*., 1996) and sensory (Recanzone *et al*., 1992*b*) learning. The rapid time scale of this reorganization has suggested that it may be due to changes in the effectiveness of previously existing synapses. Until recently this plasticity was for the most part demonstrated only within single limbs and was thought, possibly, to be limited to this spatial scale (Jain *et al*., 1995). However, more large-scale reorganizations have been demonstrated in monkeys after forelimb deafferentation (Pons *et al*., 1991; Florence and Kaas, 1995) and in human amputees (Ramachandran *et al*., 1992), in whom stimulation of the face can produce activity in the forelimb area of sensorimotor cortex or sensations in a phantom limb, respectively. On a shorter time scale, studies of motor and sensory learning in monkeys (Nudo *et al*., 1996) and humans (Pascual-Leone *et al*., 1994; Schlaug *et al*., 1994) have shown substantial enlargement of motor representations during skill acquisition. Although the debate continues as to what degree these reorganizations are due to cortical versus subcortical plasticity (Carlen *et al*., 1978; Asanuma 1991; Garraghty and Kaas, 1991; Rasmusson *et al*., 1992), the experimental evidence suggests that the cortical representation of body parts may be, to some extent, dynamically allocated from a pool of cortical resources in which the neuronal circuitry for different body parts is massively overlapping. However, the mechanisms for the stability and reorganization of such a circuitry are still being explored (Asanuma, 1991; Garraghty and Kaas, 1992; Donoghue, 1995; Kaas and Florence, 1997).

The somatotopically overlapping pattern of alpha suppression in some of our subjects presumably did not occur in response to the manipulations used in the aforementioned studies. However, we cannot exclude the possibility that the forced decision of our visual–motor task caused an expansion of motor representations. We also cannot exclude the possibil-

ity that the neurophysiological abnormalities associated with chronic epilepsy caused an aberration of functional organization in some of our subjects. However, we think this is unlikely to explain all our findings (Krauss *et al*., 1990).

If our observations of cortical-somatic overlap in the pattern of alpha ERD are accurate, and not due to abnormal neural organization, then the studies of neural plasticity noted above might offer some help in understanding the response properties of alpha ERD. In particular, alpha ERD might arise from activation of not only the real-time neural substrate of motor output, but also cortical regions that are wholly or partially capable of supporting the same motor function through short- or long-term mechanisms of synaptic plasticity.

ERD over post-central cortex

In some of our subjects, sustained alpha ERD occurred not only over precentral cortex, but also over post-central cortex. Neuroanatomical and neurophysiological studies in both animals and humans have suggested that some degree of motor processing takes place in post-rolandic cortex. Approximately one third of corticospinal fibres originate in the post-central gyrus (Coulter *et al*., 1976). In addition there are reciprocal connections between pre- and post-central cortices, particularly between BA 4 and BA 6, and between BA 2 and BA 5, respectively (Jones *et al*., 1978). On a functional level, investigators have theorized that sensory and motor processing are intimately linked by the functional necessity of guiding motor actions with sensory information (Asanuma and Mackel, 1989). Electrical stimulation of the post-central gyrus in humans can often elicit motor responses (Foerster, 1936; Penfield and Boldrey, 1937; Uematsu *et al*., 1992*a*, *b*; Nii *et al*., 1996). Penfield estimated that tongue movement occurred almost equally often with stimulation of either the post-central or the precentral gyrus, and overall ~20% of all motor responses originated from stimulation of the post-central cortex. Our visual–motor task required our subjects to maintain a strong isometric muscle contraction during presentation of a visual stimulus. Although there was no external feedback to let our subjects know whether their muscle contractions were adequate, they had to use sensory feedback cues to ensure that the contraction was sustained. This could have engaged the post-central gyrus functionally.

In Subject 4, sustained alpha ERD was observed over the inferior parietal lobe during movements of all body parts (Fig. 9). This area of the inferior parietal lobe could have been engaged by our task because of the need for continued attention to sustained muscle tension. Although our subjects were asked to focus their eyes on the monitor at all times, this subject may have covertly visualized or focused his attention on the body part being moved. Activation of the inferior parietal lobule has been documented during rhythmic fist-clenching (Ingvar and Philipson, 1977), self-paced sequential finger opposition (Wessel *et al*., 1995) and mental rotation of the hand (Bonda *et al*., 1995). Activation of this area has also been seen in stroke patients after recovery from hemiplegia (Chollet *et al*., 1991; Weiller *et al*., 1993), suggesting that this area might serve a nascent auxiliary motor function. This cortical region projects to inferior premotor cortex (BA 6) (Cavada and Goldman-Rakic, 1989), which in turn projects to the primary motor cortex (BA 4). This area has also been proposed as a centre for integration of visual inputs with motor outputs, also through connections with premotor cortex (BA 6) (Godschalk and Lemon, 1989). Alternatively, the inferior parietal lobe might have been activated in this subject by covert verbal mediation of our visual–motor task. This area probably includes the supramarginal gyrus, in which stimulation may produce dysphasia (Van Buren *et al*., 1978). Cortical stimulation did not produce a motor or sensory response here. Nevertheless, in spite of these unexpected findings in the inferior parietal lobule, sustained alpha ERD occurred in this subject over ventral pre- and post-central gyri only during tongue protrusion, not during fist-clenching or foot dorsiflexion.

Ipsilateral ERD

We expected to see bilateral activation of the sensorimotor strip with tongue protrusion, but based upon classical functional neuroanatomy, one might have expected to see activation of only the contralateral sensorimotor cortex during unilateral limb movements. Instead, at least transient alpha ERD was found with ipsilateral limb movements in all subjects in whom this was tested (i.e. all except 4). In Subject 1B, alpha and beta ERD occurred only transiently over putative arm regions during ipsilateral arm movements, but sustained alpha ERD occurred over putative leg regions during both contralateral and ipsilateral foot dorsiflexion. In Subjects 2 and 3, in which electrodes were implanted bilaterally, unilateral fist-clenching produced sustained alpha ERD over both left and right sensorimotor cortices (only with the left fist in Subject 2).

In their initial description of the mu rhythm, Chatrian *et al*. (1959) noted that it was blocked over both left and right central regions in response to unilateral limb movements and tactile stimulation. He also noted that blocking over the contralateral hemisphere occurred slightly earlier, and was more marked, than over the ipsilateral hemisphere. Using self-paced thumb movements, Pfurtscheller showed that alpha ERD occurs over contralateral central regions (sensorimotor strip) ~1.75 s before movement onset (Pfurtscheller and Berghold, 1989). However, he also observed alpha suppression over ipsilateral central regions ~700 ms before movement onset, which became bilaterally symmetrical after movement onset (Pfurtscheller and Klimesch, 1991). Because our task was externally paced, we could not examine the pre-movement time course of alpha ERD specifically, but we did find that in Subject 1B alpha ERD associated with contralateral fist-clenching appeared to begin earlier than the ERD associated with ipsilateral fist-clenching. In addition, because our task called for a sustained motor

response, we found that in many (but not all) cases alpha ERD was more lateralized to contralateral sensorimotor cortex during the prolonged execution of a motor task. Nevertheless, we still observed a significant amount of ipsilateral alpha ERD, both transient and sustained, in association with unilateral limb movements. Our findings therefore appear to complement those of others using different methods and, in turn, they provide converging evidence for some degree of ipsilateral sensorimotor activation during unilateral limb movements.

Although most studies using measures of cerebral metabolic activity or blood flow have shown only contralateral sensorimotor activation during unilateral limb movements, a few studies have shown some degree of ipsilateral activation (Yoshii *et al*., 1989; Chollet *et al*., 1991; Colebatch *et al*., 1991; Grafton *et al*., 1992; Kawashima *et al*., 1993, 1994; Kim *et al*., 1993), particularly with non-dominant hand movements. In these studies the magnitude and spatial extent of activation were always greater in the hemisphere contralateral to arm movement. In contrast to metabolic measures of activation, electromagnetic measures have often detected signal changes over the sensorimotor cortices bilaterally in association with unilateral limb movement. The absence of ipsilateral activation in most studies using metabolic measures could be due to a lower sensitivity for detecting brain activation with these techniques. The typical change of signal from baseline reported in most studies is $<50\%$ (Grafton *et al*., 1991; Kim *et al*., 1993), whereas the magnitude of alpha ERD in scalp EEG is frequently $>50\%$ (Pfurtscheller and Aranibar, 1979). This apparent difference in sensitivity may also be reflected by the difference in the typical area of activation revealed by these techniques. Whereas the area of sensorimotor cortex activated in most metabolic studies is <1.5 cm^2 (Kim *et al*., 1993), the area of alpha ERD in Subjects 1, 4 and 5, in whom this could be judged, was typically greater than this.

Electrical stimulation of sensorimotor cortex in monkeys and humans has not typically produced motor responses in ipsilateral limbs (Foerster, 1936; Penfield and Boldrey, 1937; Uematsu *et al*., 1992*a*). Nevertheless, a study of graded finger-key pressing using regional cerebral blood flow and transcranial magnetic stimulation (Dettmers *et al*., 1996) showed that as the force of key-pressing increased, there was a logarithmic increase in blood flow and a decrease in stimulation thresholds in both contralateral and ipsilateral primary motor cortices, with greater magnitude contralaterally.

One potential explanation for the bilaterality of alpha suppression with unilateral limb movements is that transcallosal axonal connections are transmitting desynchronizing signals between the two hemispheres. It is possible that through these pathways the neurophysiological mechanisms which underlie alpha reactivity, about which much is still unknown, may operate with some degree of synchrony or synergy between the two hemispheres. The association of agenesis of the corpus callosum with mirror movements has suggested to some (Freiman *et al*., 1949) that transcallosal inhibitory pathways prevent ipsilateral movements in normal adults. In such a scenario, alpha desynchronization might occur contralateral to a limb movement because of excitatory synaptic activity, and ipsilateral to the limb movement because of inhibitory synaptic activity. In this case, alpha power suppression would not discriminate between excitatory and inhibitory cortical activity.

An alternative explanation for our findings is that not only contralateral but also ipsilateral sensorimotor cortices participate in the production of unilateral limb movements. Approximately 10–15% of pyramidal tract axons originate in the ipsilateral sensorimotor cortex of humans (Glees and Cole, 1952; Nyberg-Hansen and Rinvik, 1963) and monkeys (Glees and Cole, 1952; Yakovlev and Rakic, 1966). The functional significance of ipsilateral corticospinal pathways has been studied in monkeys (Evarts, 1966; Tanji *et al*., 1988; Aizawa *et al*., 1990). The majority of these projections appear to be anatomically and functionally associated with proximal limb musculature (Kuypers, 1964; Evarts, 1966). We cannot exclude the possibility that our subjects used proximal muscles to fix their arms and legs during fist-clenching and foot dorsiflexion, respectively. However, involvement of the ipsilateral sensorimotor cortex in more distal motor functions has also been implied by the effects of lesions in monkeys (Glees and Cole, 1952) and in humans (Colebatch and Gandavia, 1989; Jones *et al*., 1989).

Ipsilateral cortical representations appear to serve a role in the recovery of motor function after lesions of contralateral sensorimotor cortex. Studies of regional CBF in stroke patients have shown activation of sensorimotor cortex ipsilateral to movements of the paretic hand (Brion *et al*., 1989), with greater activation in those with worse residual function (Weder *et al*., 1994). However, analysis of data from individual patients revealed that ipsilateral sensorimotor activation was also associated with mirror movements in the unaffected limb (Chollet *et al*., 1991; Weiller *et al*., 1993). Recovery of motor function in contralateral limbs after hemispherectomy may also depend on ipsilateral representation, at both cortical and subcortical levels (Hazemann *et al*., 1970; Benecke *et al*., 1991). In 1932, Fulton and Keller reported that, in monkeys, the paralysis produced by cortical removal of the foot area was increased by removal of the foot area on the opposite side.

Based upon our findings and those of others, outlined above, there is ample evidence that at least some degree of ipsilateral sensorimotor activation is associated with limb movement. In addition, studies of planned and accidental lesions of sensorimotor cortex suggest that ipsilateral sensorimotor representations are not critical to function but may play an auxiliary or reserve role that becomes more important if contralateral sensorimotor representations are damaged. Following these lines of evidence, we conjecture that the bilaterality of alpha suppression may reflect not only the nascent connectivity of sensorimotor cortex but also its potential functional capacity (see Conclusions).

Beta ERD

We often observed beta ERD (15–25 Hz and, to a lesser degree, 20–30 Hz) in spatial and temporal patterns paralleling alpha power suppression, in association with putative cortical activation. Our findings were consistent with those of Pfurtscheller (1981) who showed that scalp-recorded alpha (mu) and beta were suppressed with equal frequency amongst different subjects while they repetitively squeezed a rubber ball. The beta frequency bands that were most reactive in our study (15–25 Hz) were similar to those reported by others in humans (Jasper and Andrews, 1938; Jasper and Penfield, 1949; Pfurtscheller, 1981; Pfurtscheller *et al*., 1996) and in animals (Rougeul *et al*., 1979). Pfurtscheller *et al*. (1996) studied 10 subjects with scalp EEG and found that the mean frequency band of beta reactivity was 17–23 Hz. Pfurtscheller (1981) also showed that beta reactivity occurred over a broad band, did not depend on the presence of a beta peak in the frequency spectrum and could not be explained by a harmonic response from an arch-shaped mu rhythm.

Consistent with previous scalp EEG studies (Pfurtscheller, 1981; Pfurtscheller *et al*., 1996) we noted that compared with alpha ERD, beta ERD had a somewhat earlier latency but was more transient, ending earlier and sometimes resulting in a subsequent rebound above baseline power levels. The same behaviour was observed for the 20 Hz oscillations recorded by Salmelin and Hari (1994) with MEG during thumb movements.

In this study the cortical topography of beta power suppression was more focused than that of the alpha band. However, we did not see a consistent difference in the topographical distribution of alpha and beta suppression with respect to the central sulcus, as others have reported. Jasper and Penfield (1949) reported that the central fissure formed a boundary separating beta and alpha rhythms and they described precentral beta rhythm blocking with fist-clenching. On the other hand, Papakostopoulos *et al*. (1980) reported beta activity in pre- and post-central areas in man. Salmelin and Hari (1994) found that, with externally and internally paced thumb movements, the neuromagnetic sources for 10 Hz activity were posterior to those for 20 Hz activity, and they hypothesized that 20 Hz activity is a somatomotor rhythm. Using scalp EEGs, Pfurtscheller *et al*. (1996) noted a similar distinction in one of their subjects during self-paced index finger movements, but they noted no such distinction in another subject using the same task. It is possible that differences between our methodology and those of others were responsible for the lack of such a distinction, in our data, between the spatial distributions of alpha and beta ERD with respect to the central sulcus.

Because its spatial pattern was more focused, and at times more somatotopically restricted, beta ERD appeared to be a more specific index of cortical activation than alpha ERD. However, the absence of beta ERD under some conditions of cortical activation and the brevity of its temporal course suggest several possible explanations that are not mutually exclusive. Among these, it is possible that our methods did not detect beta ERD as well as alpha ERD. Alternatively, it is possible that beta ERD is a less sensitive index of cortical activation and that more cortical activation is needed to produce it and to keep it sustained. Finally, the neurophysiological mechanisms of beta ERD could be different from those of alpha ERD, perhaps including a more rapid response to cortical activation and a greater capacity for habituation than alpha ERD. The future resolution of these issues could have important implications for the use of these measures in the functional mapping of human cortex. In particular, it may be more useful to examine the spatial and temporal patterns of both alpha and beta ERD than to examine either one alone.

Conclusions

In concordance with the findings of other investigators, our data suggest that alpha ERD is a sensitive index of task-related cortical activity, particularly when recorded from the cortical surface. For example, alpha ERD was present in all five of our subjects. However, given a traditional set of assumptions about the putative neural substrates of the task that we used, we made several unexpected findings that called into question the specificity of this index. In particular, the spatiotemporal pattern of alpha suppression did not consistently satisfy our expectations that the task would produce unilateral cortical activation in a circumscribed region of contralateral precentral cortex, in a somatotopically specific pattern. Instead we found a good deal of variability in the pattern of alpha suppression, and within individual subjects we found alpha suppression outside the expected somatotopic boundaries, posterior to the central sulcus, and in both contralateral and ipsilateral sensorimotor cortices. Although temporal correlations with motor performance (transient versus sustained) appeared to enhance the specificity of alpha ERD, they could not erase all inconsistencies between our data and our basic set of assumptions. We favour two potential explanations for these apparent inconsistencies: (i) our set of assumptions about functional neuroanatomy are too narrow and are in need of revision; and (ii) alpha suppression is an index of cortical resources which may be capable of supporting a function, and which may be participating, to some degree, in the execution of a function, but which may not necessarily be critical to its current on-line execution. We think that these explanations are not mutually exclusive, and that they may both be correct to some degree.

In the discussion of each of our unexpected findings we have cited evidence for why these findings might not have been unexpected given a different set of assumptions about the functional–anatomical substrates of our task. For example, there are ample reasons to suspect that our visual–motor task engaged both pre- and post-central cortices. Evidence from other investigators has also suggested that under certain

circumstances—both normal (e.g. motor learning) and pathological (e.g. stroke or amputation), ipsilateral premotor and sensorimotor cortex can be engaged during unilateral limb movements, and the somatotopic representation of a body part can expand and even overrun the representations of neighbouring body parts. These apparent exceptions to traditional functional neuroanatomy have been well documented, but their neurophysiological mechanisms remain a subject of debate. Taken as a whole, they suggest that motor performance is made possible by a broad network of sensorimotor cortical neurons in which neuronal resources may be dynamically reallocated, presumably via different levels of synaptic plasticity. Although the mechanisms for neuronal plasticity are still being investigated, the rapidity with which somatic representations can be modified has suggested that the basic neuronal wiring is already present and that adjustments in the balance of inhibitory and excitatory synaptic activity may be all that is needed to uncover the overlapping structure of cortical representation. Such a redundant neuroanatomical and neurophysiological structure is not at all inconsistent with coarse coding and vector transformations in the service of motor function (Georgopoulos *et al*., 1993; Churchland and Sejnowski, 1992). Such a scheme could also have had the selective advantage, during evolution, of preserving critical motor skills in spite of minor brain injuries.

The neurophysiological mechanisms underlying the alpha rhythm have been the subject of much debate. The mechanisms by which it is suppressed or blocked by cortical activation are also unknown, though the term 'alpha desynchronization' illustrates one common belief. Given the broader range of assumptions considered above, the suppression of alpha power over bilateral sensorimotor regions during unilateral limb movements, and its overlapping distribution during movements of different body parts, suggest that alpha suppression could to some extent reflect the synaptic network of a distributed cortical system which is not necessarily critical to the function being performed, but which is capable of supporting that function under different circumstances. Because the neurons participating in a functional system of this sort are interconnected, activation of a subset of the network may result in widespread alpha suppression. If this is the case and if such an index can be quantitatively characterized, it might provide valuable information about the potential for recovery from planned lesions, such as tumour resection or epilepsy surgery.

Although other investigators have found a topographical segregation of alpha and beta ERD across the central sulcus, we found no such distinction and no compelling evidence that the two phenomena have neuroanatomically distinct generators. Rather, our data suggest that the two phenomena are associated with the same cortical activation, but that the neurophysiological mechanisms of beta ERD may have a higher specificity and lower sensitivity for cortical activation, a more rapid response and a greater tendency to rebound, relative to alpha ERD. Nevertheless, it is possible that differences in our tasks and analytical techniques led to these different conclusions, and questions regarding the relationship between alpha and beta certainly warrant further quantitative examination. It may be that a combination of ERD parameters derived from both the alpha and beta bands will ultimately provide a better index of currently and potentially active functional representations in sensorimotor cortex.

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