BRAIN
RESEARCH

Research Report

Event-related power modulations of brain activity preceding visually guided saccades

Debora Brignani[a], Claudio Maioli[b], Paolo Maria Rossini[a,c], Carlo Miniussi[a,b,]*

[a]*Cognitive Neuroscience Unit, IRCCS San Giovanni di Dio Fatebenefratelli, Brescia, Italy*
[b]*Department of Biomedical Sciences and Biotechnologies, Physiology Section, University of Brescia, Italy*
[c]*AFaR.–Department of Neuroscience, San Giovanni Calabita Fatebenefratelli, and Neurology, University Campus Bio-medico, Roma, Italy*

A R T I C L E   I N F O

A B S T R A C T

To analyze the characteristics of the event-related desynchronization (ERD) and synchronization (ERS) of cortical rhythms during the preparation and execution of a lateralized eye movement, EEG was recorded in normal subjects during a visually guided task. Alpha and beta bands were investigated in three temporal intervals: a sensory period, a delay period and a saccade preparation period time locked with saccade onset. Modulations of ERD/ERS power, coupled with the task, reached the largest amplitudes over the frontal and parieto-occipital regions. Differences of oscillatory activity in the alpha bands revealed an intriguing pattern of asymmetry in parieto-occipital areas. Rightward saccades induced a larger desynchronization with respect to the leftward saccades in the left hemisphere, but not in the right. If representative, these findings are congruent to the established right-hemisphere dominance of the brain areas that direct attention. Moreover differences between the two alpha types emerged in the frontal areas before and during the saccade preparation periods, indicative of differential engagement of these areas depending on the task demands. In conclusion, the present approach shows that planning eye movements is linked with covert orienting of spatial attention and may supply a useful method for studying eye movements and selective attention-related processes.

1. Introduction

Saccades are short duration ballistic eye movements performed in order to foveate an image on the retina. They are generated in the brain stem, but there is extensive evidence from electrophysiological studies in monkeys (Bruce and Goldberg, 1985; Funahashi et al., 1989; Funahashi et al., 1990; Lynch et al., 1977; Schlag and Schlag-Rey, 1987) as well as from neuroimaging (Anderson et al., 1994; Fox et al., 1985; Law et al., 1997; Luna et al., 1998; Sweeney et al., 1996) and lesion studies in humans (Pierrot-Deseilligny et al., 2003a; Pierrot-Deseilligny et al., 1991b) that the cerebral cortex is involved in their control and preparation. Three main cortical areas are involved in the generation of saccades: the *frontal eye field* (FEF), located mostly at the intersection between the precentral sulcus and the superior frontal sulcus (Paus, 1996), the *supplementary eye field* (SEF), located on the medial surface of the superior frontal gyrus (Grosbras et al., 1999) and the *parietal eye field* (PEF), located in the intraparietal sulcus (Medendorp et al., 2003). These areas contribute to the triggering of saccades with, apparently, different roles depending on the type of saccade to be performed. Actually, saccades may be *reflexive*, externally

* *Corresponding author.* Department of Biomedical Sciences and Biotechnologies, Physiology Section, Faculty of Medicine, University of Brescia, Viale Europa 11, 25123 Brescia, Italy. Fax: +39 0303717443.
    E-mail address: miniussi@med.unibs.it (C. Miniussi).

triggered by the sudden appearance of a visual target; or *voluntary*, requiring a cognitive judgment in order to determine when and where to move the eyes. Reflexive saccades are triggered by the PEF (Braun et al., 1992; Pierrot-Deseilligny et al., 1991a), while the FEF are involved both in the active fixation and in the execution of voluntary saccades (Pierrot-Deseilligny et al., 2003b; Rivaud et al., 1994). By contrast, SEF has been shown to be involved during the repetition of pre-learned saccade sequences (Gaymard et al., 1990).

Saccadic eye movements, like all human motor activity, induce specific changes that can be documented in the ongoing EEG activity. These changes may consist both of power decrement/increment in given frequency bands. The former is called event-related desynchronization (ERD) (Pfurtscheller, 1977; Pfurtscheller and Aranibar, 1977) and the latter event-related synchronization (ERS) (Pfurtscheller, 1992). These EEG modifications are time-locked to the event but not phase-locked, and thus cannot be extracted by a simple linear method, such as averaging, but have to be detected by frequency analysis. One of the basic features of ERD/ERS measurements is that the EEG power within identified frequency bands is displayed relative (as percentage) to the power of the same EEG derivations recorded during a reference period. It is assumed that, with an increasing number of interconnecting neurons, and therewith an increasing number of coherently activated neurons, the frequency decreases while the amplitude increases (Singer, 1993). This reasoning sets a general agreement – however, with several exceptions – that a neural desynchronization, recorded by EEG, can be interpreted as an electrophysiological correlate of activated cortical areas involved in the processing of sensory, cognitive information or motor behavior (Pfurtscheller and Aranibar, 1977, but see also Klimesch et al., 2007).

In the last years, different types of movement (e.g. finger, hand, foot or limb movements, proximal or distal, externally triggered or self-paced movements, motor imagery) have been studied and specific ERD/ERS pattern has been demonstrated. In general, voluntary movements are accompanied by an ERD in the alpha and beta bands, which begins at about 1.5–2 s before the movement onset over the contralateral sensorimotor areas and becomes bilaterally symmetrical about the time of EMG onset. After movement execution, ERD is replaced by an ERS, which occurs earlier for the beta than for the alpha band (Derambure et al., 1993; Leocani et al., 1997; Pfurtscheller, 1981; Pfurtscheller and Aranibar, 1979; Pfurtscheller and Berghold, 1989; Pfurtscheller et al., 1999; Stancak and Pfurtscheller, 1996; Toro et al., 1994).

To the best of our knowledge, there are no studies that investigated the pattern of ERD/ERS during the preparation and execution of lateralized eye movements to a given location. Only few studies analyzed cortical oscillations associated with oculomotor processes (Kamoda et al., 2001; Kamoda et al., 2000; Skrandies and Anagnostou, 1999) but none of them performed any analyses on decreasing or increasing of power in given frequency bands. Moreover, this approach may shed light on a still controversial question that regards the tight relationship between spatial attention and eye movements systems. Whereas the two systems can work

independently, as when attention moves towards a peripheral location while eyes are holding fixation, the preparation of an eye movement appears always to induce a concurrent attentional shift towards the same location (Chelazzi et al., 1995; Kowler et al., 1995; Shepherd et al., 1986). The premotor theory of attention (Rizzolatti et al., 1987) asserts that the attentional orienting is equivalent to an eye movement planning; it just corresponds to a below-threshold activation of saccade motor programs. Actually, brain imaging (Corbetta et al., 1998; Nobre et al., 2000; Nobre et al., 1997), behavioral (Sheliga et al., 1995a,b; Sheliga et al., 1994), neurophysiological (Moore and Fallah, 2001) and neurological (Heide and Kompf, 1998; Heilman et al., 1993) studies provided evidence that covert orienting of spatial attention and planning eye movements are strictly linked both at functional and anatomical levels. In order to investigate this topic, we studied event-related power modulations before visually guided saccades, namely voluntary saccades internally triggered towards a target presented laterally in the visual field.

2. Results

2.1. Behavioral data

The behavioral data from 12 subjects were analyzed. No significant difference in saccade latency or error between the two response directions emerged from the ANOVA, showing that subjects executed rightward and leftward saccades with the same latency and accuracy. The mean latency across subjects of leftwards saccades ($N=719$) was 199 ± 43 ms, with a mean error of $-0.36\pm1.48°$. By contrast, rightwards saccades ($N=792$) had a mean latency of 204 ± 41 ms and yielded a mean error of $-0.42\pm1.54°$.

2.2. ERD/ERS data

2.2.1. Alpha band

2.2.1.1. Sensory period. As can be seen in the first box of Fig. 1 (sensory period), at the time of the visual cue presentation, we documented significant blocking of alpha rhythm (ERD) which was significantly higher over the parietal areas (mean=-49.5%) compared to the other cortical areas (mean=-24.2%), irrespectively of the alpha band type [*region*: $F(2,22)=28.80$, $p<0.001$]. Moreover, the statistical analysis revealed an interaction between *alpha type × region* factors [$F(2,22)=7.53$, $p=0.015$] indicating a difference over the central area during the sensory period, namely the lower band (-30.4%) showed a larger mean desynchronization than the upper band (-15.3%). Finally, as can be seen in Fig. 2, an effect related to the saccade direction emerged in the upper alpha band [*alpha type × saccade direction × hemisphere*: $F(1,11)=9.68$, $p=0.009$] showing that saccade direction modulated power in the left (leftward=-22.3%; rightward=-33.1%) but not in the right hemisphere. The left hemisphere was less desynchronized when the saccade was supposed to be towards the left hemispace while the right hemisphere was equally desynchronized for saccades aiming in both directions.
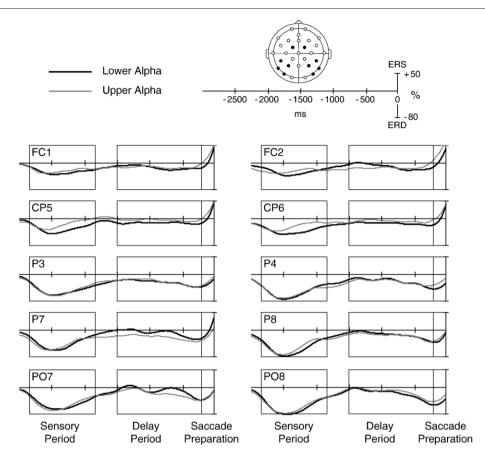
Fig. 1 – Grand average waveforms (*N*=12) of event-related power modulations of the alpha lower (black lines) and upper (grey lines) bands. In this and all subsequent figures, the electrode montage used for the experiment is shown at the top; the locations of the electrode sites for the sample waveforms are shaded black; the sketched boxes indicate the three temporal intervals considered for the statistical analysis (sensory period, delay period and saccade preparation).

2.2.1.2. Delay period. During the delay period, ERD of the alpha rhythm decreased towards baseline levels, with no statistically significant difference between the two alpha bands. Nevertheless, the parietal region still maintained a significantly larger desynchronization [$F(2,22)=5.29$, $p=0.018$] compared to central areas. Moreover, the statistically significant interaction between *alpha type* and *region* factors [$F(2,22)=5.33$, $p=0.016$] proved that this difference was due to a larger desynchronization of the upper alpha band over parietal regions.

2.2.1.3. Saccade preparation. During the saccade preparation, another desynchronization was observed over parietal and occipital areas. Inspection of the traces in Fig. 1 reveals that this blocking of alpha rhythm actually started at least 500 ms before the saccade onset and reached its greatest amplitude at about the beginning of the saccade preparation phase. Therefore, by taking into account the average saccadic latencies in our experimental sample (∼200 ms), we must conclude that a desynchronization in alpha rhythm developed over the parietal–occipital cortical areas during the 300 ms gap interval that followed the central fixation offset, and reached its maximum amplitude about the time of the appearance of the peripheral target.

On the contrary, a fast build-up of synchronization in the alpha rhythm was observed over the frontal and central areas within the 200 ms time interval preceding saccade onset. Statistically, the difference in alpha band power between frontal and central regions (mean=21.7%) and parietal region (mean=−25.7%) was significant [$F(2,22)=30.99$, $p<0.000$].

In addition, statistically significant differences between the two alpha bands emerged in the central areas, where the upper alpha rhythm showed a higher mean synchronization (25.6%) with respect to the lower alpha band (2.9%) [*alpha type×region*: $F(2,22)=6.95$, $p=0.013$]. Finally, as described in the sensory period, the significant interaction *saccade direction× hemisphere* [$F(1,11)=5.99$, $p=0.032$] showed that leftward saccades induced a larger synchronization in both the alpha bands compared to rightward saccades (16% vs. −2%) only in the left hemisphere.

2.2.2. Beta band
As can be seen in Fig. 3, a reduction of beta power was observed mainly over parieto-occipital regions during the sensory and the saccade preparation periods. However, modulations of this band were statistically less conspicuous than for the alpha rhythm. Nevertheless, the power desynchronization recorded over the parietal region (mean=−20.1%)
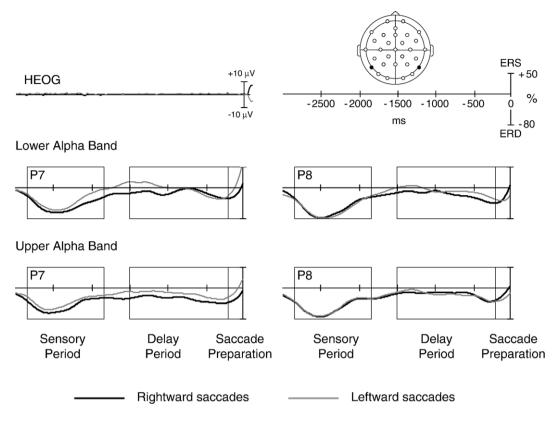
Fig. 2 – Grand average waveforms (N=12) of event-related power modulations of the alpha band (lower alpha band upward; upper alpha band downward) for rightward (black lines) and leftward (grey lines) saccades. In this figure, grand average of Horizontal EOG is included only for display purpose. In the analyses, the EOG was examined on a trial-by-trial basis and used to discard trials with anticipatory saccades or blinks.

showed a greater amplitude compared to that observed over frontal and central areas (mean = −1.5%) irrespective of the period considered in the analysis [region: $F(2,22) > 8.09$, $p < 0.005$].

3.   Discussion

This study is the first to provide an account of the event-related power modulations related to the programming of visually guided saccades. Alpha and beta bands were investigated in three temporal intervals: a sensory period, during which a visual cue informed subjects of the direction of the subsequent eye movement; a delay period, that required them to maintain the central fixation; a saccade preparation period, in which fixation was released and a saccade had to be executed towards a newly appearing peripheral visual target.

Our results show that brain activity resulted in a desynchronization of both alpha and beta rhythms during the sensory and saccade preparation periods, which reached the largest amplitudes over the parietal and occipital regions. The alpha blocking phenomenon has been regarded as reflecting a state of cortical activation which not only can be induced by the stimulation of visual or other sensory processing, but is also characteristic for planning, preparation of movements (Chatrian et al., 1959; Pfurtscheller and Berghold, 1989; Pfurtscheller et al., 1994) and cognitive tasks (Doppelmayr et

al., 2005; Klimesch, 1999; Klimesch et al., 1997). Furthermore, it is possible to divide the alpha rhythm into two frequency bands: lower 8–10 Hz, upper 10–12 Hz, which previous research suggests to have different functional meaning (Klimesch et al., 1992; Pfurtscheller et al., 2000). In general, the lower alpha desynchronization is obtained in response to almost any type of task. It is topographically widespread without any clear localization and probably reflects general cognitive and attentional demands. By contrast, the upper alpha desynchronization is more tightly coupled to specific tasks and, therefore, tends to be more localized at those areas which play an important role in specific cognitive processes. It is generally believed that upper alpha ERD most likely reflects computational processes such stimulus identification and response preparation in a movement task but in several studies, it has been associated to cognitive performance, in particular to the processing of memory and semantic tasks (Doppelmayr et al., 2005; Klimesch, 1999; Klimesch et al., 1997). Differences between the two alpha rhythm types emerged in the central areas during the sensory and the saccades preparation periods. In both intervals, the upper alpha band showed a significantly smaller desynchronization with respect to the lower alpha band, suggesting a less specific engagement of central cortical areas throughout the task.

Also a beta band desynchronization was observed during the sensory and the saccade preparation periods, even if of lower amplitude than the alpha blocking. Beta activity is
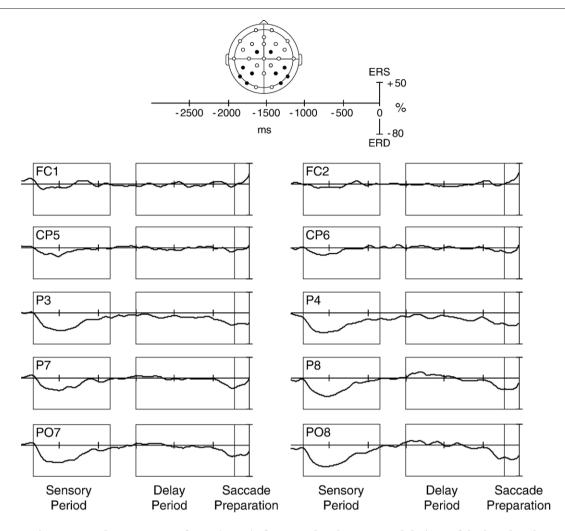
Fig. 3 – Grand average waveforms (N=12) of event-related power modulations of the beta band.

considered to be highly movement-reactive and its power does not necessarily decrease in association with the alpha blocking (Pfurtscheller and Aranibar, 1980). In the present experiment, similarly to the upper alpha band, the largest beta desynchronization was observed over the posterior-parietal cortical areas, reflecting the relevant role of these areas in attentional deployment and in the visuomotor integration processes underlying the oculomotor task.

The results reveal a neural process supporting the role of parietal areas in eye movement and attention tasks that can be derived from an intriguing pattern of asymmetry in hemispheric activation, observed during the sensory as well as the saccade preparation periods. Rightward saccades induced a larger desynchronization in the alpha band with respect to leftward saccades in the left hemisphere only. By contrast, the right hemisphere showed an overall larger desynchronization with respect to the left hemisphere, which was symmetrical for saccades to both directions (see Fig. 2). In other words, eye movement control is not distributed equally to the left and the right hemifields and the two hemispheres cover asymmetrically the visual field. The right hemisphere controls eye movements to either the left or right visual field, but the left hemisphere preferentially controls eye movements to the contralateral right visual field. This result

resembles attention-like phenomena, which show a lateralization to the right hemisphere.

It has been suggested (Corbetta and Shulman, 2002) that voluntary attention is mediated by parietal areas in conjunction with the FEFs; such a fronto-parietal network acts to maintain control on a given location. In our task, a shift of spatial attention during the sensory period is certainly induced by the cue stimuli and the expectation of a subsequent eye movement towards one side of the visual field. Furthermore, attentive resources need also to be allocated during the saccade preparation. In fact, the premotor theory of attention (Rizzolatti et al., 1987), as well as data from functional neuroimaging (Corbetta et al., 1998; Nobre et al., 2000), indicates a strict functional and anatomical relationship between attention and saccade programming. In this context, our results are consistent with the well established notion of right-hemisphere dominance for directing visuo-spatial attention (Mesulam, 1981). Accordingly, the observed directional asymmetry in the left hemisphere activation may reflect its engagement, mainly when attention is directed towards the contralateral side. By contrast, the larger and bidirectional response of the right hemisphere is consistent with its predominant role in shifting spatial attention to both visual hemifields (Miniussi et al., 2002). The present results suggest a

right-hemisphere overlap to the critical neural system that directs attention as well as eye movements to spatial locations.

In this study, a temporal gap was present between the offset of the central fixation point and the onset of the peripheral target towards which subjects had to execute the saccade. By taking into account the average saccadic latencies in our experimental sample (∼200 ms), we must conclude that the described desynchronization in the alpha and beta rhythms over the parieto-occipital cortical areas developed during the period just before and after the central fixation offset, and reached its maximum amplitude about the time of the appearance of the saccadic target. The timing of this response is reminiscent of the evoked potential called 'gap negativity' (Csibra et al., 1997; Everling et al., 1996; Gomez et al., 1996; Spantekow et al., 1999), with which it may, at least partially, share underlying neural mechanisms. Event-related potential studies in humans have shown that the disappearance of the fixation point, at a fixed delay before target presentation, elicits a distinct negative potential with a maximum at frontal and central electrodes, that peaks around the time of target appearance (Csibra et al., 1997; Everling et al., 1996; Gomez et al., 1996; Spantekow et al., 1999). This 'gap negativity' was interpreted as a readiness potential and reflects preactivation of the frontal circuits implicated in the generation of saccadic eye movements (Everling et al., 1996; Gomez et al., 1996).

Interestingly, power synchronization in the alpha rhythm started to develop over the frontal and central regions after the onset of the peripheral stimulus. In this respect, it should be noted that the appearance of the saccadic target also leads to the additional process of releasing the active fixation of the central point. Thus, a possible interpretation of this ERS in the alpha band is that it may be related to the disengagement of the frontal eye fields, which are involved both in the active fixation and in the suppression of voluntary saccades during the delay period (Pierrot-Deseilligny et al., 2003b; Rivaud et al., 1994). However, a note of caution should be applied to the interpretation of this last synchronization. Visual inspection of the EEG signal indicates that the more lateralized electrodes (i.e. F7/8; FC5/6) recorded a signal related to the horizontal eye movement response. The application of a highly selective band-pass filter to the EEG data during the ERD/ERS measurement procedure, may have induced ripples in the alpha band range in the time period immediately preceding the saccade onset. Therefore, part of this synchronization may be due to an artifact induced by the applied power analysis. It should be noted, however, that alpha synchronization was also clearly present in the central electrodes, where no signs of eye movement-related signals could be detected. This suggests that the observed ERS could at least partially originate from a neuronal source.

In conclusion, we have demonstrated that the present approach may supply a useful method for studying eye movements as well as selective attention-related processes and their mutual linkage, providing new evidence of cortical activation in a visually guided saccade task. Power modulation of brain rhythmic oscillations linked to the sensory and delay periods, including shifts of attention and maintenance of the fixation, revealed an asymmetrical parietal activation consis-

tent with the established notion of right-hemisphere dominance for directing visual spatial attention. These findings show that planning eye movements is linked at functional and neural levels with covert orienting of spatial attention.

4. Experimental procedures

4.1. Participants

Twenty-four healthy volunteers participated in the study and were enrolled after giving written informed consent. Four were discarded from the analysis for excessive noise in the recording and anticipatory eye movements. Other eight subjects were disqualified because they did not perform a sufficient number of regular saccades (see below). The remaining 12 participants (3 females) had a mean age of 23.7 years (range of 21 to 30). All were right handed according to the Edinburgh handedness inventory test (Oldfield, 1971) and had normal or corrected to normal visual acuity. The experimental methods were non-invasive and had ethical approval from the local committee.

4.2. Behavioral task and procedure

Participants sat in a dimly illuminated room and their head was stabilized with a combination of a chin rest and a head support bar. A large screen was placed in front of them, at a distance of 1.5 m. Each trial started with the appearance of a fixation target in the middle of the screen. Subjects had to perform a saccade to a peripheral visual target presented after a temporal delay during which they were requested to maintain central fixation. Before the delay period, a visual cue informed subjects about the direction of the subsequent saccadic response. The behavioral task is schematically displayed in Fig. 4.
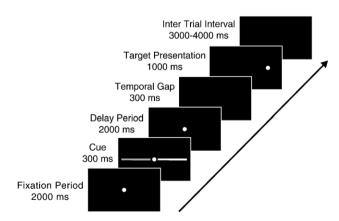

Fig. 4 – Schematic illustration of the behavioral task. Central fixation point and visual cue are in white and grey here, but were presented in red and green, respectively, during the experiment. Subjects fixated on a central point (0.2°). A brief (300 ms) lateralized line indicated the direction of the target to follow (left or right). After a fixed interval of 2000 ms, the central fixation disappeared. Subjects were instructed to make a saccade to the target that appeared 300 ms after the central fixation offset.

After subjects maintained fixation on a central target for a period of 2 s, a horizontal line (±20° of visual angle) of the same color as the central fixation point was flashed for 300 ms, informing subjects on the direction of the subsequent eye movement. In order to minimize interhemispheric differences related to the visual stimulation, an identical second line, but of a different color (distractor), also appeared together with the informative cue in the contralateral visual field. The central fixation target was maintained alone for a constant delay period of 2 s, during which subjects had to avoid making eye movements. At the end of this delay period, the central fixation point disappeared and, after a temporal delay of 300 ms, subjects had to move their gaze to a peripheral target (1000 ms) that appeared randomly in one of 8 possible positions, equally spaced along the horizontal meridian, within a range of ±20° of visual field with respect to the primary position. The intertrial interval was between 3000 and 4000 ms and the reappearance of the fixation point indicated the start of the next trial. All visual stimuli were presented on a black background. The central fixation point and the peripheral target were dots with a diameter of 0.43°. For half of the subjects, the color of the central fixation point, and therefore of the valid cue line, was red, while the color of the distractor line was green. For the other half of the subjects, the colors of the visual stimuli were reversed. At the beginning of the experiment, subjects were informed about the relationship between the colors of informative and distractor cues. Each experimental session was made up of 4 blocks, each one comprised of 64 trials (total number of trials: 256). Before the experiment, a training block familiarized subjects with the task condition. Subjects were instructed to maintain muscle relaxation during the experiment and to avoid blinking during the task.

4.3. EEG and eye movement recordings

The electroencephalographic (EEG) activity was recorded continuously from 29 sites using electrodes set in an elastic cap (Electro-Cap International, Inc.) and positioned according to the 10–20 International system (AEEGS, 1991). The montage included 3 midline sites (FZ, CZ and PZ), 13 sites over each hemisphere (FP1/FP2, F3/F4, F7/F8, FC1/FC2, FC5/FC6, C3/C4, T7/T8, CP1/CP2, CP5/CP6, P3/P4, P7/P8, PO7/PO8, O1/O2) and the right mastoid. Additional electrodes were used as ground and reference sites. The ground electrode was placed in front of FZ. The left mastoid served as reference for all electrodes. The recordings obtained from the right mastoid electrode were used off-line to re-reference the scalp recordings to the average of the left and right mastoids. Data were recorded with a band-pass filter of 0.01–100 Hz and digitized at a sampling rate of 500 Hz (SynAmps, NeuroScan). Electrodes-skin impedance was set below 5 kΩ.

The horizontal and vertical electrooculogram (EOG) was also recorded (DC-200 Hz low pass-filtered, 500 samples/s) through four additional electrodes, placed at the external canthi and above and below the right eye. A dedicated program (LabView 6, National Instruments) was set to calibrate EOG and to monitor eye movements during the experimental trials. Fixation was checked on line, on a dedicated screen, and verbal feedback was given to the subjects during the short breaks between experimental blocks.

4.4. Behavioral analysis

To verify accuracy and correct task performance, subjects' eye movements were analyzed off-line. Each trial of the EOG signal was visually inspected in order to check for artifacts and to evaluate the direction and the latency of the saccades (the latter being determined as the time interval between the appearance of the peripheral target and the saccade onset). Trials with blinks and anticipatory eye movements, as well as trials with saccades in the wrong direction, were excluded from the analysis. Moreover, we decided to include in the analysis only regular saccades namely saccades with latency between 140 and 350 ms; the other set of saccades were anticipatory or express saccades showing a random distributed amplitude (i.e. not related to the visual stimuli).

As accuracy measurement of eye movement responses, we computed the saccadic error, defined as the difference between the absolute values of the measured saccade amplitude and the actual target position. According to this definition, negative and positive error values indicate hypometric and hypermetric responses, respectively, independently of saccade direction.

4.5. ERD/ERS analysis

ERD/ERS activity after the saccade onset was not considered since previous studies suggested that electrocortical activity recorded at the onset and throughout saccades is spoilt by the activation of extra-ocular muscles (Nativ et al., 1990; Riemslag et al., 1988; Thickbroom and Mastaglia, 1985; Thickbroom and Mastaglia, 1986; Weinstein et al., 1991). EEG recordings were re-filtered digitally with a band-pass of 0.2–50 Hz. Then they were divided into epochs from −4.1 to 0 s with respect to the saccade onset. Only epochs corresponding to correct regular saccades were included in the EEG analysis. Moreover, epochs with artifacts (voltage exceeding ±100 μV) were automatically rejected. Spatial resolution of artifact-free EEG data was enhanced by surface Laplacian estimation (Babiloni et al., 1996; Perrin et al., 1989). This transformation acts as a spatial filter that reduces head volume conductor effects and annuls electrode reference influence. Therefore, a single trial analysis was carefully repeated on the Laplacian-transformed epochs, to discard trials contaminated by residual computational artifacts. Separate ERD/ERS in the alpha (lower 8–10 Hz; upper 10–12 Hz) and beta (13–20 Hz) bands were obtained for each subject and for each saccade direction (left, right). To quantify the event-related changes of the EEG power, we used 1 s during the fixation period as a reference and the time interval of 2.8 s before the saccadic onset as the activity period. ERD/ERS were computed according to the accepted approach (Pfurtscheller and Aranibar, 1977; Pfurtscheller and Aranibar, 1979; Pfurtscheller and Neuper, 1994), following the next steps: bandpass filtering of all Laplacian EEG epochs, squaring of the amplitude samples, averaging of power samples across all trials. For each data point, ERD/ERS were calculated in accordance with the standard formula: [(band-power-active-interval−band-power-reference-interval)/band-power-

reference-interval]*100. Smoothing of ERD/ERS traces was performed by using a moving averaging window of 100 ms.

Because there were no *a priori* hypotheses for how the task would affect ERD/ERS, we employed an analysis strategy that tested for effects of task over successive time bins. A preliminary data analysis was performed using mean power values every 100 ms, between 2900 and 0 ms before the saccade onset. In this way, three different temporal intervals were identified: a *sensory period* (2800–1800 ms before the saccade onset); a *delay period* (1500–200 ms before the saccade onset) and a *saccade preparation period* (200–0 ms before the saccade onset). Mean power values over these periods were employed as dependent variable in the ANOVA. In order to sample localized power modulation as evenly as possible, we adopted an analysis strategy using three separate symmetrical regions over the scalp: frontal (F3/4, FC1/2, FC5/6), central (C3/4, CP1/2, CP5/6) and parietal (P3/4, P7/8, PO7/8).

For each of the two frequency ranges (alpha and beta), an ANOVA with repeated measures was performed, with four within-subjects factors: *saccade direction* (left vs. right), *region* (frontal vs. central vs. parietal), *hemisphere* (left vs. right) and *electrode location*. For the alpha band analysis, an additional factor was tested: *alpha type* (lower vs. upper). These analyses were repeated for each temporal interval. The Greenhouse–Geisser epsilon correction factor was applied where appropriate to compensate for possible effects of non-sphericity in the compared measurements.

R E F E R E N C E S

AEEGS, 1991. American Electroencephalographic Society guidelines for standard electrode position nomenclature. J. Clin. Neurophysiol. 8, 200–202.

Anderson, T.J., Jenkins, I.H., Brooks, D.J., Hawken, M.B., Frackowiak, R.S., Kennard, C., 1994. Cortical control of saccades and fixation in man. A PET study. Brain 117 (Pt 5), 1073–1084.

Babiloni, F., Babiloni, C., Carducci, F., Fattorini, L., Onorati, P., Urbano, A., 1996. Spline Laplacian estimate of EEG potentials over a realistic magnetic resonance-constructed scalp surface model. Electroencephalogr. Clin. Neurophysiol. 98, 363–373.

Braun, D., Weber, H., Mergner, T., Schulte-Monting, J., 1992. Saccadic reaction times in patients with frontal and parietal lesions. Brain 115, 1359–1386.

Bruce, C.J., Goldberg, M.E., 1985. Primate frontal eye fields. I. Single neurons discharging before saccades. J. Neurophysiol. 53, 603–635.

Chatrian, G.E., Petersen, M.C., Lazarte, J.A., 1959. The blocking of the rolandic wicket rhythm and some central changes related to movement. Electroencephalogr. Clin. Neurophysiol., Suppl. 11, 497–510.

Chelazzi, L., Biscaldi, M., Corbetta, M., Peru, A., Tassinari, G., Berlucchi, G., 1995. Oculomotor activity and visual spatial attention. Behav. Brain Res. 71, 81–88.

Corbetta, M., Shulman, G.L., 2002. Control of goal-directed and stimulus-driven attention in the brain. Nat. Rev., Neurosci. 3, 201–215.

Corbetta, M., Akbudak, E., Conturo, T.E., Snyder, A.Z., Ollinger, J.M., Drury, H.A., Linenweber, M.R., Petersen, S.E., Raichle, M.E., Van, D., Essen, C., Shulman, G.L., 1998. A common network of functional areas for attention and eye movements. Neuron 21, 761–773.

Csibra, G., Johnson, M.H., Tucker, L.A., 1997. Attention and oculomotor control: a high-density ERP study of the gap effect. Neuropsychologia 35, 855–865.

Derambure, P., Dujardin, K., Defebvre, L., Bourriez, J.L., Jacquesson, J.M., Guieu, J.D., 1993. Spatiotemporal study of event-related desynchronization during self-paced movement. Neurophysiol. Clin. 23, 337–351.

Doppelmayr, M., Klimesch, W., Hodlmoser, K., Sauseng, P., Gruber, W., 2005. Intelligence related upper alpha desynchronization in a semantic memory task. Brain Res. Bull. 66, 171–177.

Everling, S., Krappmann, P., Spantekow, A., Flohr, H., 1996. Cortical potentials during the gap prior to express saccades and fast regular saccades. Exp. Brain Res. 111, 139–143.

Fox, P.T., Fox, J.M., Raichle, M.E., Burde, R.M., 1985. The role of cerebral cortex in the generation of voluntary saccades: a positron emission tomographic study. J. Neurophysiol. 54, 348–369.

Funahashi, S., Bruce, C.J., Goldman-Rakic, P.S., 1989. Mnemonic coding of visual space in the monkey's dorsolateral prefrontal cortex. J. Neurophysiol. 61, 331–349.

Funahashi, S., Bruce, C.J., Goldman-Rakic, P.S., 1990. Visuospatial coding in primate prefrontal neurons revealed by oculomotor paradigms. J. Neurophysiol. 63, 814–831.

Gaymard, B., Pierrot-Deseilligny, C., Rivaud, S., 1990. Impairment of sequences of memory-guided saccades after supplementary motor area lesions. Ann. Neurol. 28, 622–626.

Gomez, C., Atienza, M., Gomez, G.J., Vazquez, M., 1996. Response latencies and event-related potentials during the gap paradigm using saccadic responses in human subjects. Int. J. Psychophysiol. 23, 91–99.

Grosbras, M.H., Lobel, E., Van de Moortele, P.F., LeBihan, D., Berthoz, A., 1999. An anatomical landmark for the supplementary eye fields in human revealed with functional magnetic resonance imaging. Cereb. Cortex 9, 705–711.

Heide, W., Kompf, D., 1998. Combined deficits of saccades and visuo-spatial orientation after cortical lesions. Exp. Brain Res. 123, 164–171.

Heilman, K.M., Bowers, D., Valenstein, E., Watson, R.T., 1993. Disorders of visual attention. Bailliere's Clin. Neurol. 2, 389–413.

Kamoda, M., Touge, T., Ikeguchia, M., Nishioka, M., Takeuchi, H., 2000. A study of middle latency somatosensory evoked potentials during eye opening-closing and voluntary eye movement. Jpn. J. Clin. Neurophysiol. 28, 24–30.

Kamoda, M., Touge, T., Ikeguchia, M., Deguchi, K., Watanabe, S., Takeuchi, H., 2001. Changes of middle latency somatosensory evoked potentials (SEPs) and alpha rythmicity associated with oculomotor processes. Clin. Neurophysiol. 112, 2250–2254.

Klimesch, W., 1999. EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis. Brain Res. Brain Res. Rev. 29, 169–195.

Klimesch, W., Pfurtscheller, G., Schimke, H., 1992. Pre- and post-stimulus processes in category judgement tasks as measures by event-related desynchronization (ERD). J. Psychophysiol. 6, 185–203.

Klimesch, W., Doppelmayr, M., Pachinger, T., Russegger, H., 1997. Event-related desynchronization in the alpha band and the processing of semantic information. Brain Res. Cogn. Brain Res. 6, 83–94.

Klimesch, W., Sauseng, P., Hanslmayr, S., 2007. EEG alpha oscillations: the inhibition-timing hypothesis. Brain Res. Brain Res. Rev. 53, 63–88.

Kowler, E., Anderson, E., Dosher, B., Blaser, E., 1995. The role of attention in the programming of saccades. Vision Res. 35, 1897–1916.

Law, I., Svarer, C., Holm, S., Paulson, O.B., 1997. The activation pattern in normal humans during suppression, imagination and performance of saccadic eye movements. Acta Physiol. Scand. 161, 419–434.

Leocani, L., Toro, C., Manganotti, P., Zhuang, P., Hallett, M., 1997. Event-related coherence and event-related desynchronization/synchronization in the 10 Hz and 20 Hz EEG during self-paced movements. Electroencephalogr. Clin. Neurophysiol. 104, 199–206.

Luna, B., Thulborn, K.R., Strojwas, M.H., McCurtain, B.J., Berman, R.A., Genovese, C.R., Sweeney, J.A., 1998. Dorsal cortical regions subserving visually guided saccades in humans: an fMRI study. Cereb. Cortex 8, 40–47.

Lynch, J.C., Mountcastle, V.B., Talbot, W.H., Yin, T.C., 1977. Parietal lobe mechanisms for directed visual attention. J. Neurophysiol. 40, 362–389.

Medendorp, W.P., Goltz, H.C., Vilis, T., Crawford, J.D., 2003. Gaze-centered updating of visual space in human parietal cortex. J. Neurosci. 23, 6209–6214.

Mesulam, M.M., 1981. A cortical network for directed attention and unilateral neglect. Ann. Neurol. 10, 309–325.

Miniussi, C., Rao, A., Nobre, A.C., 2002. Watching where you look: modulation of visual processing of foveal stimuli by spatial attention. Neuropsychologia 40, 2448–2460.

Moore, T., Fallah, M., 2001. Control of eye movements and spatial attention. Proc. Natl. Acad. Sci. U. S. A. 98, 1273–1276.

Nativ, A., Weinstein, J.M., Rosas-Ramos, R., 1990. Human presaccadic spike potentials. Of central or peripheral origin? Invest. Ophthalmol. Visual Sci. 31, 1923–1928.

Nobre, A.C., Sebestyen, G.N., Gitelman, D.R., Mesulam, M.M., Frackowiak, R.S., Frith, C.D., 1997. Functional localization of the system for visuospatial attention using positron emission tomography. Brain 120, 515–533.

Nobre, A.C., Gitelman, D.R., Dias, E.C., Mesulam, M.M., 2000. Covert visual spatial orienting and saccades: overlapping neural systems. NeuroImage 11, 210–216.

Oldfield, R.C., 1971. The assessment and analysis of handedness: the Edinburgh inventory. Neuropsychologia 9, 97–113.

Paus, T., 1996. Location and function of the human frontal eye-field: a selective review. Neuropsychologia 34, 475–483.

Perrin, F., Pernier, J., Bertrand, O., Echallier, J.F., 1989. Spherical splines for scalp potential and current density mapping. Electroencephalogr. Clin. Neurophysiol. 72, 184–187.

Pfurtscheller, G., 1977. Graphical display and statistical evaluation of event-related desynchronization (ERD). Electroencephalogr. Clin. Neurophysiol. 43, 757–760.

Pfurtscheller, G., 1981. Central beta rhythm during sensorimotor activities in man. Electroencephalogr. Clin. Neurophysiol. 51, 253–264.

Pfurtscheller, G., 1992. Event-related synchronization (ERS): an electrophysiological correlate of cortical areas at rest. Electroencephalogr. Clin. Neurophysiol. 83, 62–69.

Pfurtscheller, G., Aranibar, A., 1977. Event-related cortical desynchronization detected by power measurements of scalp EEG. Electroencephalogr. Clin. Neurophysiol. 42, 817–826.

Pfurtscheller, G., Aranibar, A., 1979. Evaluation of event-related desynchronization (ERD) preceding and following voluntary self-paced movement. Electroencephalogr. Clin. Neurophysiol. 46, 138–146.

Pfurtscheller, G., Aranibar, A., 1980. Voluntary movement ERD: normative studies. In: Pfurtscheller, G., Buser, F.H., Lopes da Silva, F.H., Petsche, H. (Eds.), Rhythmic EEG Activities and Cortical Functioning. Elsevier/North-Holland Biomed Press, Amsterdam, pp. 151–177.

Pfurtscheller, G., Berghold, A., 1989. Patterns of cortical activation during planning of voluntary movement. Electroencephalogr. Clin. Neurophysiol. 72, 250–258.

Pfurtscheller, G., Neuper, C., 1994. Event-related synchronization of mu rhythm in the EEG over the cortical hand area in man. Neurosci. Lett. 174, 93–96.

Pfurtscheller, G., Neuper, C., Mohl, W., 1994. Event-related

desynchronization (ERD) during visual processing. Int. J. Psychophysiol. 16, 147–153.

Pfurtscheller, G., Pichler-Zalandek, K., Neuper, C., 1999. ERD and ERS in voluntary movement of different limbs. In: Pfurtscheller, G., Lopes da Silva, F.H. (Eds.), Handbook of Electroencephalography and Clinical Neurophysiology, vol. 6. Elsevier, Amsterdam, pp. 245–268.

Pfurtscheller, G., Neuper, C., Krausz, G., 2000. Functional dissociation of lower and upper frequency mu rhythms in relation to voluntary limb movement. Clin. Neurophysiol. 111, 1873–1879.

Pierrot-Deseilligny, C., Rivaud, S., Gaymard, B., Agid, Y., 1991a. Cortical control of reflexive visually-guided saccades. Brain 114, 1473–1485.

Pierrot-Deseilligny, C., Rosa, A., Masmoudi, K., Rivaud, S., Gaymard, B., 1991b. Saccade deficits after a unilateral lesion affecting the superior colliculus. J. Neurol., Neurosurg. Psychiatry 54, 1106–1109.

Pierrot-Deseilligny, C., Muri, R.M., Ploner, C.J., Gaymard, B., Demeret, S., Rivaud-Pechoux, S., 2003a. Decisional role of the dorsolateral prefrontal cortex in ocular motor behaviour. Brain 126, 1460–1473.

Pierrot-Deseilligny, C., Muri, R.M., Ploner, C.J., Gaymard, B., Rivaud-Pechoux, S., 2003b. Cortical control of ocular saccades in humans: a model for motricity. Prog. Brain Res. 142, 3–17.

Riemslag, F.C., Van der Heijde, G.L., Van Dongen, M.M., Ottenhoff, F., 1988. On the origin of the presaccadic spike potential. Electroencephalogr. Clin. Neurophysiol. 70, 281–287.

Rivaud, S., Muri, R.M., Gaymard, B., Vermersch, A.I., Pierrot-Deseilligny, C., 1994. Eye movement disorders after frontal eye field lesions in humans. Exp. Brain Res. 102, 110–120.

Rizzolatti, G., Riggio, L., Dascola, I., Umilta, C., 1987. Reorienting attention across the horizontal and vertical meridians: evidence in favor of a premotor theory of attention. Neuropsychologia 25, 31–40.

Schlag, J., Schlag-Rey, M., 1987. Evidence for a supplementary eye field. J. Neurophysiol. 57, 179–200.

Sheliga, B.M., Riggio, L., Rizzolatti, G., 1994. Orienting of attention and eye movements. Exp. Brain Res. 98, 507–522.

Sheliga, B.M., Riggio, L., Craighero, L., Rizzolatti, G., 1995a. Spatial attention-determined modifications in saccade trajectories. NeuroReport 6, 585–588.

Sheliga, B.M., Riggio, L., Rizzolatti, G., 1995b. Spatial attention and eye movements. Exp. Brain Res. 105, 261–275.

Shepherd, M., Findlay, J.M., Hockey, R.J., 1986. The relationship between eye movements and spatial attention. Q. J. Exp., A Psychol. 38, 475–491.

Singer, W., 1993. Neuronal representations, assemblies and temporal coherence. Prog. Brain Res. 95, 461–474.

Skrandies, W., Anagnostou, E., 1999. Electroencephalographic cortical oscillations and saccadic eye movements in humans. Neurosci. Lett. 261, 57–60.

Spantekow, A., Krappmann, P., Everling, S., Flohr, H., 1999. Event-related potentials and saccadic reaction times: effects of fixation point offset or change. Exp. Brain Res. 127, 291–297.

Stancak Jr., A., Pfurtscheller, G., 1996. Event-related desynchronisation of central beta-rhythms during brisk and slow self-paced finger movements of dominant and nondominant hand. Brain Res. Cogn. Brain Res. 4, 171–183.

Sweeney, J.A., Mintun, M.A., Kwee, S., Wiseman, M.B., Brown, D.L., Rosenberg, D.R., Carl, J.R., 1996. Positron emission tomography study of voluntary saccadic eye movements and spatial working memory. J. Neurophysiol. 75, 454–468.

Thickbroom, G.W., Mastaglia, F.L., 1985. Presaccadic 'spike'

potential: investigation of topography and source. Brain Res. 339, 271–280.

Thickbroom, G.W., Mastaglia, F.L., 1986. Presaccadic spike potential. Relation to eye movement direction. Electroencephalogr. Clin. Neurophysiol. 64, 211–214.

Toro, C., Cox, C., Friehs, G., Ojakangas, C., Maxwell, R., Gates, J.R., Gumnit, R.J., Ebner, T.J., 1994. 8–12 Hz rhythmic oscillations in human motor cortex during two-dimensional arm movements: evidence for representation of kinematic parameters. Electroencephalogr. Clin. Neurophysiol. 93, 390–403.

Weinstein, J.M., Balaban, C.D., VerHoeve, J.N., 1991. Directional tuning of the human presaccadic spike potential. Brain Res. 543, 243–250.