Reduction of EEG power during expectancy periods in humans

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Abstract. The contingent negative variation (CNV), and the associated event-related desynchronization (ERD) on motor areas and sensory areas, and increase of alpha in the ipsilateral to the cued stimulus side, are different brain signals that reflect motor, sensory, and cognitive activations related to the expectancy of the next stimulus. However, the possibility of an overall change in EEG oscillatory activity during expectancy periods has not been directly addressed. The present report tests whether the background oscillatory activity is modulated by a warning signal. During the expectancy period, the power spectral density (PSD) between 0 and 42.9 Hz – including delta, theta, alpha, beta, and gamma – decreased with respect to the baseline. These results suggest that during expectancy periods there is a generalised decrease in the oscillatory activity, and that reduction of the EEG power would facilitate the phasic and oscillatory neural activities triggered by the next target stimulus.

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INTRODUCTION

Most of the studies aimed at understanding how the human brain prepares for the next stimulus, and its possible responses, are based on analysis of the so-called contingent negative variation (CNV), which appears when a warning stimulus (S1) provides pertinent information regarding the arrival of a second stimulus (S2, the imperative stimulus) (Rockstroh et al. 1982, Walter et al. 1964). It is generally assumed that the CNV is a reflection of the subthreshold activation of the cortex during the S1-S2 period that would facilitate the processing of the next stimulus. The CNV is due to the tonic activation of frontal association cortex, motor cortex, and sensory cortex (Brunia 1999, Gómez et al. 2003). The extent to which the cortical activation associated to the CNV is task-related is under current investigation: for instance, the possibility that during preparatory periods the brain actively excites the areas assumed necessary for the processing of the next stimulus (Brunia 1999, Gómez et al. 2001, 2003).

CNV tonic activity is not the only neural signal that can be detected in the preparatory periods prior to the appearance of target stimulus. In fact, analysis using ERPs disregards possible modulations of the different spectral components present in the EEG (alpha, beta, gamma, etc.). Various approaches, such as Fourier-fast transform (Gómez et al. 1998), event-related desynchronization (ERD) (Pfurtscheller and Lopes da Silva 1999), or temporal spectral evolution (TSE) (Salmelin and Hari 1994, Vázquez et al. 2001) have been used to evaluate the modulations of the spectral bands related with attention, working memory, and other processes. Use of ERD has shown a modulation in alpha and beta bands in a variety of tasks related to the attentional and intentional preparation for the stimulus (Fu et al. 2001, Gómez et al. 1998, Pfurtscheller and Lopes da Silva 1999, Worden et al. 2000). For instance, ERD in the alpha range has been found in the motor cortex during preparation for movement (Pfurtscheller and Lopes da Silva 1999) and during expectancy of a visual feedback stimulus (Bastiaansen and Brunia 2001, Bastiaansen et al. 1999). Alpha increase has also been obtained during preparatory periods in areas that should be deactivated for a correct performance of the task (Fu et al. 2001, Worden et al. 2000), and prior to imperative stimulus in a category judgement task (Klimesch et al. 1992). The latter has been explained as the selective inactivation of some brain areas (Pfurtscheller and Lopes da Silva 1999).

From a neurobiological perspective, the CNV can be considered a consequence of the tonic depolarisation of apical dendrites in the cortex, which would allow a state of elevated excitability for the firing threshold to be reached (Rockstroh et al. 1982). On the other hand, brain oscillatory activity will result from synchronicity of the activity in more-or-less distributed networks that would include at least cortico-cortical and thalamic-cortical networks. The independence of alpha ERD and CNV has already been described (Filipovic et al. 2001). Underlying these oscillations would be not only the dynamic interaction between distributed groups of neurons, but also intrinsic membrane properties and the influence of neurotransmitter systems (Lopes da Silva 1991, Singer and Gray 1995).

The present report tests the possible reduction of EEG rhythmical activities due to warning in a broad spectral band (0-42.9 Hz). This proposal would allow testing whether – in addition to the modulatory effects of the preparatory periods on the EEG signal as indexed by CNV and ERD in both the alpha and beta bands (Pfurtscheller and Lopes da Silva 1999) – there could co-exist some oscillatory changes related to preparation in a broad spectral band over specific areas in the scalp. The hypothesis that the reduction operates on the same brain areas that are spontaneously oscillating during baseline periods will also be tested. The EEG spectral power of the baseline prior to the S1 stimulus (the baseline period) will be correlated with the EEG power modulation occurring in the expectancy period (pre-target) prior to S2. The similarity between topographies for different frequency bands analysed by correlations will be interpreted in terms of modulatory effects of brain areas already oscillating during the baseline period.

METHODS

Thirteen subjects (7 female, 5 male; 11 right-handed, 2 left-handed) between 18 and 28 years old took part in the experiment. The experiments were conducted with the informed consent of each subject. Participants were seated 50 cm in front of a computer screen. They were instructed to fix their eyes on a green square (0.91° of visual angle) which would appear in the center of the screen, and to press one button of the mouse as soon as a target (also a green square) appeared peripherally to the first square. The central stimulus was the warning stimulus, and the peripheral stimulus the imperative one. The eccentricity of the target was 11.4° from the fixa-

tion point in the left or right visual field of the horizontal meridian. The left button of the mouse had to be pressed when the target appeared in the left visual field, and the right one when the target appeared in the right visual field. Both responses were made with the right hand, using the index finger for left responses and the middle one for right responses. Each subject was given 500 trials.

Stimulus presentation was computer-controlled (Stim system, Neuroscan®). The event sequence within a trial was as follows: the central fixation point was on for 1 second, a temporal gap (200 ms) followed the offset of the fixation stimulus, and then the peripheral target was on for 1 second. The inter-trial interval (time between presentation of target stimulus and warning) was 2 seconds. The so-called gap paradigm was used because it has been proved particularly useful in inducing sensory and motor attention in subjects (Gómez et al. 2001, 2003).

Vertical and horizontal eye movements were recorded by electro-oculography (EOG). The electrodes were placed on the external canthi of both eyes for recording horizontal movements, and on the inferior and superior areas of the ocular orbit for recording vertical eye movements. EEG data were collected from 58 scalp sites of an extended version of the International 10-20 system, using tin electrodes mounted in an electrode cap. All the electrodes were referred to the left mastoid. Impedance was maintained below 5,000 ohms. Data were filtered using a band-pass of 0.01-100 Hz (1/2 amplitude low- and high-frequency cut-offs); the amplification gain was 30,000 (Synamps, Neuroscan®). Recordings were notch-filtered at 50 Hz. They were acquired at a sampling rate of 500 Hz, using a commercial AD acquisition and analysis board (Scan). Recordings were averaged off-line using an artifact-rejection protocol based on voltage amplitude. All the epochs for which the EEG or EOG exceeded 50 microvolts (10.8% of the sweeps) were automatically rejected and excluded from subsequent analysis. The algebraically linked mastoids were computed off-line and used as reference for purposes of analysis.

The power spectral density (PSD) for the period of 512 ms before the warning (S1) stimulus (baseline) and for the period of 512 ms before the target stimulus (expectancy period) was analysed by means of the Fast Fourier Transform. No windows were applied to the data. The obtained spectra were averaged separately for each subject and time window considered. Differences

in frequency components between the pre-S1 and the pre-S2 period were analysed, calculating the mean values of spectral power in selected frequency windows (0-3.9, 3.9-7.8, 7.8-11.7, 11.7-19.5, 19.5-31.2, 31.2-42.9 Hz). These frequency ranges will be considered as delta, theta, theta and low-alpha, high-alpha and low-beta, high beta, and finally gamma, respectively. They were selected to account for the whole frequency range between 0 and 42.9 Hz. The numbers are non-integers because the analysed epochs were of 512 ms. This epoch duration produces frequency bins of 1.95 Hz. Consequently all the frequency ranges are multiples of 1.95 Hz. The statistical analysis of the spectral power consisted of a two-factor analysis of variance (ANOVA) for each frequency band. The ANOVA factors were: (i) condition (EEG activity during baseline vs. EEG activity during expectancy period); and (ii) electrodes (58 electrodes). The ANOVA results were Geisser-Greenhouse corrected using the epsilon coefficient when necessary.

In order to assess whether the modulation of the different frequency bands was due to a reduction in the oscillatory activity of the generators active during the baseline period, the Pearson correlation coefficient was
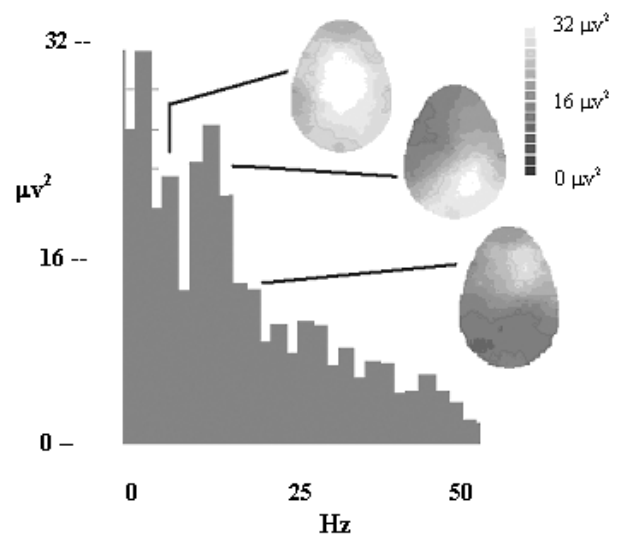

Fig. 1. Power spectral density (PSD) and topographical maps of the PSD values of the Oz electrode during the baseline condition. Note the decay of the spectrum as 1/f with a peak in the alpha range. The maps are for theta (7 Hz), upper alpha (12 Hz), and beta (19 Hz) bands. Note the central midline distribution of theta, the posterior topography of alpha, and the anterior distribution of beta. Delta presented a posterior distribution and gamma a frontal distribution (maps not shown).

computed between the PSD values in each frequency band and electrode during baseline vs. the PSD during the expectancy period minus PSD values during baseline. Basically, this procedure correlates the spectral power in the baseline condition (Fig. 3A) with the corresponding maps of the modulatory effect of expectancy (Fig. 3B). The underlying rationale of performing this correlation is that if the expectancy condition produces a reduction in the oscillatory brain generators that are active during the baseline period, the reduction in each electrode and frequency band should be proportional to

baseline values. The mathematical justification of the correlational method used in the present report appears in the appendix.

RESULTS

Figure 1 shows the frequency spectrum in electrode Oz, and the topographies for theta, alpha, and beta bands during the baseline period. The spectrum shows the typical 1/f decline with a peak in the alpha range. Note the frontal-central distribution of theta, the posterior distri-
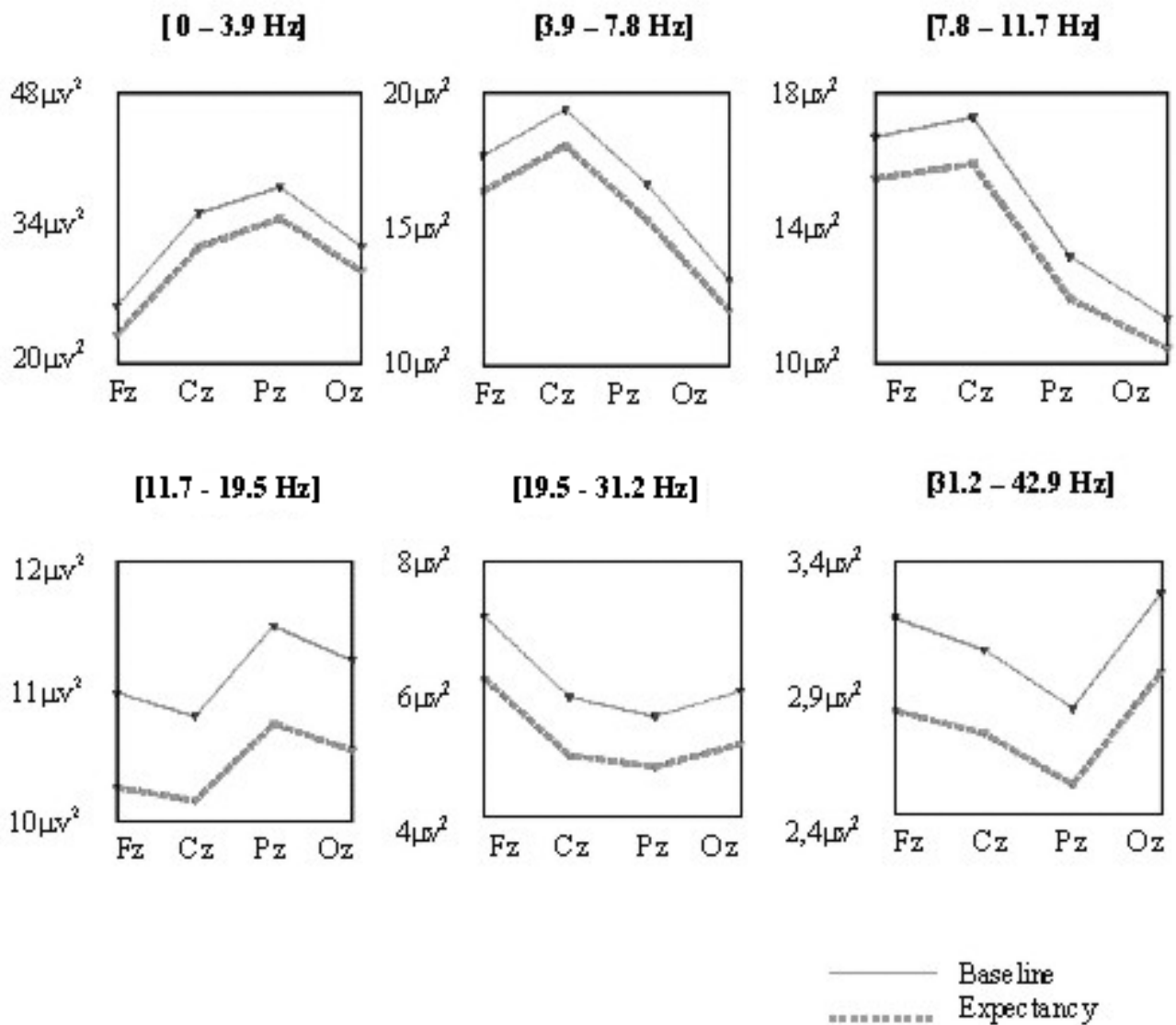

Fig. 2. Power spectral density (PSD) values for the whole frequency range between 0 and 42.9 Hz in four midline electrodes (Fz, Cz, Pz and Oz) for the expectancy (pre-target) and baseline (pre-warning) periods. The time window for the analysis was 512 ms in both cases. Note the reduction of PSD in midline electrodes during the expectancy period with respect to baseline for all the considered frequency ranges.

bution of alpha, and the frontal distribution of beta. Delta showed a higher amplitude in posterior sites, and gamma had a frontal distribution (maps not shown). The maps for the pre-target period showed a distribution similar to that of the baseline (maps not shown).

Figure 2 shows the mean values of the spectral power in the different frequency ranges analysed. Note the reduction of the spectral power in the pre-target condition with respect to the baseline condition for the whole frequency range and for all the displayed midline electrodes. The statistical analysis showed a statistically significant difference between the baseline and pre-target condition values (Table I) for all the frequency bands considered. The table also includes the percentage of reduction for each EEG band, collapsed for the 58 electrodes analysed. The reduction occurred for the whole frequency range. There was no interaction between the effects of the factors condition and electrode

except in the frequency range of low beta (11.7-19.5 Hz, $F_{1,3.753}=4.213$, $P=0.006$).

Figure 3A shows the topographies for the analysed frequency bands during the baseline period. Note the voltage distributions previously described. The maps for the subtraction of the spectral power in the baseline period from that in the expectancy period presented negative values (Fig. 2B), due to the higher spectral power values in the baseline condition with respect to the expectancy period. The maps of spectral differences showed a distribution quite similar to that for the baseline. The correlation coefficients of corresponding maps shown in Figs. 3A and 3B were highly statistically significant for all the frequency ranges considered (Table II), indicating a proportional (to baseline values) modulatory effect of the expectancy situations on the brain oscillations already present during the baseline period.
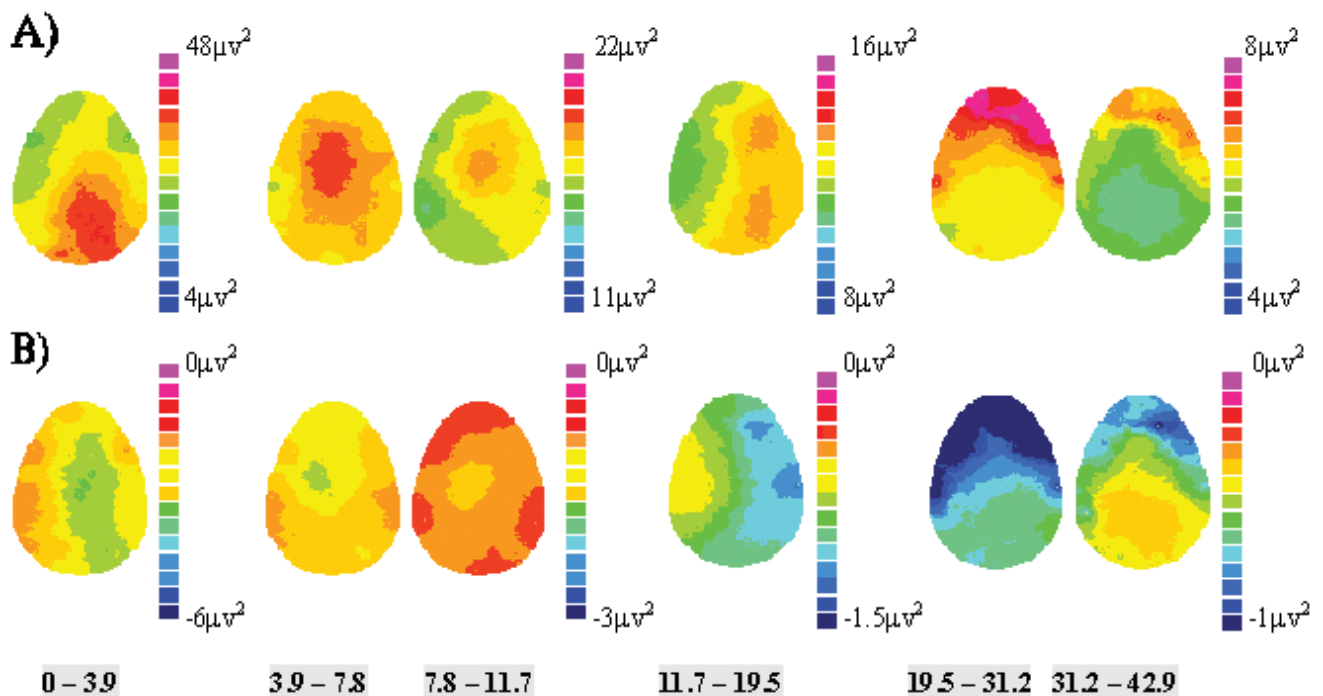

Fig. 3. (A) Topographical maps of the power spectral density (PSD) during the baseline condition. The maps represent the collapsed PSD value for the frequency range indicated: the classical frequency bands of delta (0-3.9), theta (3.9-7.8), theta and low-alpha (7.8-11.7), high-alpha and low-beta (11.7-19.5), high beta (19.5-31.2) and gamma (31.2-42.9) Hz. Note the central midline distribution of theta, the posterior topography of alpha and delta, and the anterior distribution of beta and gamma. More-detailed maps with narrower frequency ranges confirmed these topographies (maps not shown). (B) Topographical maps of the subtraction of baseline PSD values from those during the expectancy condition. Note that the topographies are similar to those of the corresponding maps in Fig. 1, but show negative values, indicating that PSD values were higher in the baseline than in the expectancy condition.

Table I

ANOVA results of the comparison between the EEG activity in baseline (pre-warning) vs. the expectancy period (pre-target) EEG activity

Frequency Hz	F (D.F.=1,12)	P	Reduction %
0 - 3.9	7.975	0.015	9.37
3.9 - 7.8	11.932	0.005	8.02
7.8 - 11.7	8.807	0.012	9.74
11.7 - 19.5	13.054	0.004	6.65
19.5 - 31.2	8.829	0.012	8.89
31.2 - 42.9	8.881	0.011	8.80

The F value, statistical significance (P), and the percentage of reduction collapsed for the four electrodes considered are presented.

DISCUSSION

The present report shows that during expectancy-to-stimulus periods, in addition to the presence of a CNV component (Brunia 1999, Gómez et al. 2001) there is a spectral power reduction for all the EEG bands from 0 to 42.9 Hz of around 8% of spectral power in the expectancy condition with respect to the baseline condition (Table I). The fact that the different frequency ranges were unequal should not affect the reliability of the results, given the general effect on the reduction of EEG power obtained in the expectancy condition with respect to baseline. The use of dynamic techniques such as ERD would improve the time resolution of the already obtained EEG power reduction.

In the case of the alpha band, specific and non-specific reactivity has been described for different frequencies. The lower range (7-10 Hz) occurs during a great variety of tasks, is topographically widespread, and is probably related to attentional task demands (Klimesch et al. 1990). On the other hand, the upper alpha ERD appears more often during the processing of sensory-semantic information, with a topography restricted to parietal-occipital areas (Klimesch et al. 1997). The motor-related ERD occurs in the upper alpha and lower beta bands, and is localised close to sensorial-motor areas (Stancák Jr. and Pfurtscheller 1996). In cases where an increase of alpha activity can be recorded during active cognitive processing, the increase of alpha oscilla-

Table II

Frequency Hz	r	D.F.	P
0 - 3.9	0.733	57	0.001
3.9 - 7.8	0.941	57	0.001
7.8 - 11.7	0.959	57	0.001
11.7 - 19.5	0.815	57	0.001
19.5 - 31.2	0.934	57	0.001
31.2 - 42.9	0.942	57	0.001

(r) Pearson correlation coefficient of PSD values in the 58 electrodes when comparing the baseline condition with respect the modulatory effect of the expectancy period, (D.F.) degrees of freedom, (P) statistical significance.

tions could be interpreted as triggered oscillations related to processing information (Basar et al. 1989), but – particularly in the case of sustained oscillations in the alpha range – it could be interpreted as an electrophysiological marker of "idling cortical areas" (Chase and Harper 1971).

In the present report, there is a more generalised reduction of spectral power, including the previously described alpha and beta band ERD, but the reduction is extended to delta, theta, and gamma bands. The high correlation between the PSD values of the baseline condition and those of the modulatory effect of expectancy, suggest that during the preparatory period there is a reduction of brain rhythm amplitude in the areas that are oscillating during the baseline period. This kind of argument − modulation of voltage amplitude but maintenance of topography similar to that of the baseline condition − has been used in the literature of event-related potentials to support sensory gain control as a neural mechanism for selective attention (Hillyard et al. 1998). The present experiment extends previous results that showed a reduction of EEG power from 0.2 to 40 Hz with respect to baseline during visual stimulation with a rotating disc of 26 cm for 10 seconds (Vijn et al. 1991). That result was obtained with a visual stimulus to drive visual neurons much stronger than the one used in the present experiment, and was not directly related to expectancy. Moreover, no references to topographies are provided in that report (Vijn et al. 1991).

In the particular case of alpha oscillations, alpha power decreased in the present experiment. A decrease in alpha oscillations has also been obtained during the

expectancy for visual feedback (Bastiaansen and Brunia 2001, Bastiaansen et al. 1999). Present results are at variance with others obtained in a similar paradigm, where cueing for an auditory target increased alpha values (Fu et al. 2001), and visuospatial cueing increased alpha to the ipsilateral side (Worden et al. 2000). Those results were interpreted as suggesting that an increase of alpha is used to deactivate the brain areas that are not needed for the task. In the present experiment, a decrease in alpha was obtained, but it must be remarked that as the subjects do not know the location of the next target, it would be more convenient – for the processing of the next visual stimulus – to decrease alpha power in left and right visual cortices. In this sense, decreasing alpha activity has already been reported during different kinds of cognitive operations on visual images, such as attention (Gómez et al. 1998, Vázquez et al. 2001), visual imagination (Michel et al. 1994), and the phasic REM period (Cantero et al. 1999). The decrease of alpha during the pre-target period obtained in the present experiment could be interpreted as an active preparation of the visual cortex to process the next incoming stimulus.

In the case of beta, an increase of beta power has been reported during attentional expectancy in cats (Bekisz and Wróbel 2003, Wróbel 2000) and during post-target attentional processing in humans (Gómez et al. 1998, Vázquez et al. 2001). In the present experiment, a decrease in the beta PSD power has been obtained in the late phase of the expectancy period. It must be remarked that recent wavelet analysis (Ghazaryan et al. 2003) has shown an increase of beta activity followed by a progressive decline along the expectancy to visual stimulus period. The latter result is similar to present results.

There are at least two different ways in which neurons could process an incoming sensory stimulus: (i) through phasic activity in response to the incoming stimulus – this operational mode being reflected as ERPs ; and (ii) increasing already present oscillatory activity – this operational mode being reflected as event-related oscillatory activity, and increased brain area coherence in specific frequency ranges (Basar et al. 1989). The latter way of functioning would imply that certain areas of the cortex have a natural resonant frequency, such as alpha for occipital, theta for midline frontal (Ishii et al. 1999), or beta for frontal areas. Both phasic and oscillatory modes of operation would benefit from a reduced oscillatory activity over the whole scalp for a broad frequency range during the expectancy period, given that the following stimulus would be able to produce a neural response (oscillatory or phasic) that was more salient when background activity was reduced.

This reduced background activity in the different cortical areas producing brain rhythms would facilitate the attentive processing of the imperative stimulus. However, it must be remarked that scalp recordings cannot distinguish between reduced voltages at the sources and a reduced EEG power caused by desynchronization of cortical columns producing an out-of-phase averaging in the electrode due to the superposition principle. However, the very extensive EEG power reduction obtained over the whole cortex would favour the idea of a reduction of intracranial current sources in the very late phase of the expectancy period for the different frequency bands tested in the present experiment. How the obtained EEG power reduction is dependent on the particular stimulatory contingencies used in the present experiment remains to be determined.

A broad reduction of spectral power in the EEG after consumption of caffeine (a noradrenergic agonist that increases the subject's vigilance) has been described (Gilbert et al. 2000). The role of increasing the signal-to-noise ratio of the relevant stimulus has been proposed for the noradrenergic system. These results allow speculation that the locus coeruleus (Aston-Jones et al. 1999) and/or other noradrenergic nuclei would facilitate the attentive processing of cognitive information by reducing the EEG power in the late phase of the expectancy period.

CONCLUSION

The late phase of an expectancy situation produces a reduction of the spectral power of the EEG in the different brain rhythms, which could facilitate the processing of an incoming stimulus.

APPENDIX (Author: C. M. Gómez)

The present report uses a correlational method to propose that the topographies of the voltage maps of the baseline and the modulation by expectancy are similar. Its validity is presented in this appendix. The central assumption is that a high correlation between two voltage maps implies that the same generators are active in both conditions. The demonstration is valid only for generators that modulate the amplitude by the same constant, while maintaining source position and orientation.

The electrical fields in the brain follow the superposition law (Scherg et al. 1990). This law implies that the potential at electrode k is the linear sum of the contributions from all sources.

$$U_k(t) = \sum_{i=1}^{NS} C_{kt} S_i(t) \qquad (1)$$

($U_k(t)$) Voltage at electrode k, (NS) number of sources, (C_{kt}) transmission coefficients (the value depends on the position and directionality of the source – if these parameters remain steady, then this coefficient is constant), ($S_i(t)$) amplitude of the source.

Thus, to obtain the voltage (U'_k) in the same set of electrodes but modulating the amplitude of the source by a constant (1/h):

$$U'_k(t) = \sum_{i=1}^{NS} C_{kt} S_i(t)\, 1/h \qquad (2)$$

As 1/h is a constant

$$U'_k(t) = 1/h \sum_{i=1}^{NS} C_{kt} S_i(t) = 1/h\, U_k(t) \qquad (3)$$

Our aim is to prove that if the sources are modulated by a constant (1/h), then the new set of voltages in the electrodes ($U'_k(t)$), when correlated with the initial voltage in the electrodes (U_k), should have a correlation of 1. As voltages are measured in a given time, the time will not be included. Pearson's correlation coefficient (r_{xy}) equation is:

$$r_{xy} = \frac{\sum_{i=1}^{n}\left[\left(X_i - \overline{X}\right)\left(Y_i - \overline{Y}\right)\right]}{\sqrt{\sum\left(X_i - \overline{X}\right)^2}\sqrt{\sum\left(Y_i - \overline{Y}\right)^2}} \qquad (4)$$

Applied to our set of data, to compute the correlation coefficient between the voltages in the same electrodes in two different conditions:

$$R_{w'} = \frac{\sum_{k=1}^{n}\left[\left(U_k - \overline{U_k}\right)\left(U'_k - \overline{U'_k}\right)\right]}{\sqrt{\sum\left(U_k - \overline{U_k}\right)^2}\sqrt{\sum\left(U'_k - \overline{U'_k}\right)^2}} \qquad (5)$$

Substituting (3):

$$R_{w'} = \frac{\sum_{k=1}^{n}\left[\left(U_k - \overline{U_k}\right)\left(1/hU_k - 1/h\overline{U_k}\right)\right]}{\sqrt{\sum\left(U_k - \overline{U_k}\right)^2}\sqrt{\sum\left(1/hU_k - 1/h\overline{U_k}\right)^2}} \qquad (6)$$

Given than 1/h is a constant:

$$R_{w'} = \frac{1/h\sum_{k=1}^{n}\left[\left(\overline{U_k} - U_k\right)\left(U_k - \overline{U_k}\right)\right]}{\sqrt{\sum\left(U_k - \overline{U_k}\right)^2}\,1/h\sqrt{\sum\left(U_k - \overline{U_k}\right)^2}} \qquad (7)$$

$$r_{w'} = \frac{\sum_{k=1}^{n}\left(U_k - \overline{U_k}\right)^2}{\sum_{k=1}^{n}\left(U_k - \overline{U_k}\right)^2} = 1 \qquad (8)$$

The obtained result implies that modulating the amplitude of the brain sources, keeping orientation and position of the sources constant would produce a Pearson's correlation of 1 between the empirical voltages in the two conditions. The correlation obtained in the experimental part is close to –1 for all the frequency ranges, suggesting that the negative modulation in amplitude obtained in the expectancy period with respect to baseline corresponds to an amplitude modulation of the sources already present during the baseline period.

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