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Research Report

Wavelet analysis of the EEG during the neurocognitive evaluation of invalidly cued targets

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ABSTRACT

In a spatial central cueing paradigm, positions in the horizontal meridian were cued to evaluate the neurocognitive processing of validly (V) and invalidly cued (I) targets. ERPs were obtained from 20 EEG channel recordings. Complex Morlet wavelets were applied for computing event-related spectral power (ERSP) modulations and inter-trial phase coherence (ITC). P3a and P3b responses were increased in a statistically significant manner in I targets with regard to V targets. This increase seems to be generated only by phase resetting without enhancement of spectral power. Comparing ERSP modulations between I and V target trials we found a major effect centred in the alpha range. The following results were obtained for invalid condition in relation to valid condition: 6–12Hz ERSP decrease topographically widespread over the scalp, starting around 450ms and peaking around 650ms; 10–14Hz ERSP increase peaking around 200ms at fronto-central electrodes; and 10–14Hz ERSP decrease occurring from 400 to 600ms at posterior electrodes. Therefore, the invalidity effect indeed produces salient changes in the stimulus related and ongoing neuronal activity leading to a brain state of comparative higher activity both excitatory and inhibitory with respect to the validly cued target processing.

1. Introduction

In daily life humans continuously monitor actions in order to act according to the environment. In a broad perspective, this "perception–action cycle" has been introduced by Fuster (2003) to highlight the continuous interplay and outcome evaluation between perceptual and executive networks. However, a continuous expectancy bias occurs for certain stimuli and action driving the former perception–action cycle to a preparation–perception–action cycle. The existence of a neural signature indicating the adequacy among preparation, perception and action might help validating the hypothesis of a complete preparation–perception–action cycle.

In the central cue Posner paradigm a central cue can validly (V trials) or invalidly (I trials) indicate the spatial position of the upcoming target. As a consequence of preparation there is a speeded response (benefit) when the spatial position of targets is validly predicted and a delayed response (cost) if the spatial position of the target is invalidly predicted (Posner, 1980). This spatial cueing effect on the attentional sensory processing has also been evaluated by analyzing the modulation of the ERPs to targets which have been validly or invalidly

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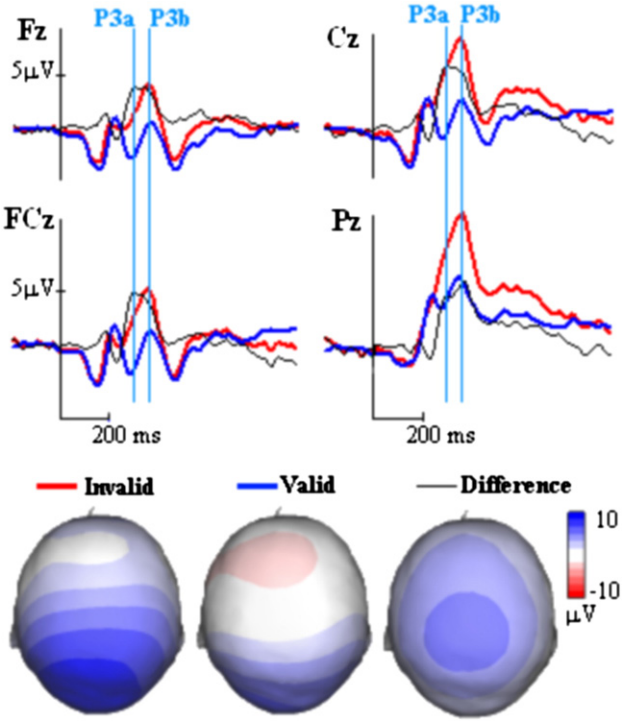

Fig. 1 – ERPs grand average (μV) of invalid and valid conditions and the difference wave. The vertical lines are crossing the mean point of the time windows used for the statistic analyses, which were 260–320 ms for P3a and 320–380 ms for P3b. At 290 ms the difference wave presents its peak at Fz and FCz electrodes. The P3b response peaks at 350 ms for invalid condition at Pz. Voltage maps (μV) of invalid and valid conditions and the difference wave are also displayed in the time window of 260 to 320 ms. Notice the more anterior extension of the posterior positivity (P3) to anterior sites in the invalid condition with respect to the valid condition. The voltage map of the difference wave remarks the P3a component at fronto-central electrodes.

cued (Eimer, 1993; Mangun and Hillyard, 1991; Perchet and García-Larrea, 2000; Perchet et al., 2001). The general result obtained was an increase of the P1 and N1 and a decrease of posterior P3 components in valid cued trials with respect to invalid ones (Luck et al., 1990; Mangun and Hillyard, 1991; Mangun et al., 1993; Anllo-Vento, 1995; Coull, 1998). An increase at the latency of the P3 component for I trials with respect to V trials has also been suggested, indicating that subjective expectancy induced by the cue (Mangun and Hillyard, 1991) was not accomplished in I trials. Furthermore, the possibility of dissociating a frontal late positive component from the posterior positive component has been observed (Eimer, 1993), but not further explored.

This late positive component is considered to be composed by subcomponents generated in different subcortical regions. P3a and P3b are known subcomponents of P3 which are associated with different neurocognitive processes and have distinct topographic amplitude distributions (Polich, 2007). They can be elicited by a variety of stimuli, including those that are defined as targets as well as novels, and can also co-occur within the same ERP waveform. The P3a is frontally oriented, whereas the P3b is localized at posterior scalp (Friedman et al., 2001).

Although the exact nature of P3a and P3b is not fully understood, the context updating theory proposes that the amplitude of P3 increases inversely to its appearance probability (Duncan-Jhonson and Donchin, 1977). Moreover, and more related with the present experiment, Duncan-Johnson and Donchin (1982) showed that the P3 to low probability target letters (indicated by the type of cue) presented in the centre of the screen showed an increased P3 when compared with the high probability target letters. They interpreted the latter result as a clear indication that P300 is related to the updating of self-hypothesis about the information provided by the environment after an event with a low subjective probability. In the same line of reasoning, we suggested (Gómez et al., 2008) that invalidly cued targets are processed in a similar manner to low frequency targets in odd-ball paradigms, as indicated by the presence of an increased P3a and P3b in I trials. The increased P3a (Friedman et al., 2001; Dien et al., 2003) would index the processing of the invalidly cue target as a novel stimulus, while according to Donchin and Coles (1988), increase of P3b in I trials would represent the context updating operation and subsequent memory storage (Polich, 2007).

The goal of the present study is to determine if there is a neural signature modulated by the adequacy of the preparation and the presented target stimulus. To reach this goal, we studied the characteristics of the P3a and P3b elicited when the invalidity of the spatial prediction induced by the cue is detected after the arrival of the S2. We will focus our attention in the modulation of brain rhythms involved in the generation of these evoked potential, especially changes in power and phase resetting. In recent years these two measures have been revealed as crucial in understanding the nature and behaviour of brain electrical activity. Hence, increase/decrease of power and inter-trial coherence would reflect the state of neuronal groups involved in certain task (David et al., 2005). In addition, phase locking of trials at certain frequency to stimulus presentation or events can provide relevant information on cognitive processes (Sauseng and Klimesch, 2008). It has been proposed that there are certain cognitive processes that

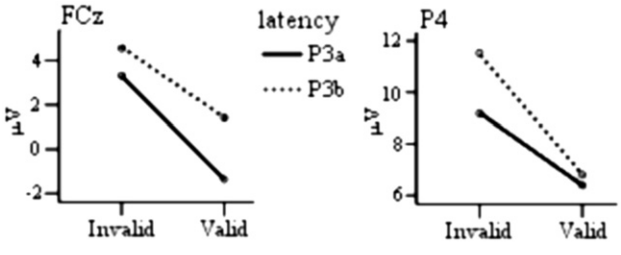

Fig. 2 – Graphics of mean voltage (μV) × condition for P3a and P3b latencies at electrodes FCz and P4. The difference in the slopes of the two lines can be observed, at FCz the slope of P3a latency line is greater than the slope of P3b latency line, at P4 the slope of P3b latency line is greater than the slope of P3a latency line.

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cannot be dissociated by using traditional physiological measures (such as ERPs latency and amplitude), but might be disentangled by using phase resetting (see i.e. Klimesch et al., 2004; Fuentemilla et al., 2008). In addition it has also been shown that some ERPs can be generated only by the phase resetting paradigm without concomitant increase of power (Makeig et al., 2002; Fuentemilla et al., 2006), or by a combination of both processes (Fuentemilla et al., 2006). Therefore to understand the characteristics of brain responses associated to a cognitive process is necessary for the study of their spectral measures (i.e. power modulation and phase resetting).

The proposed time-frequency approach will complement previous results on P3a and P3b increment during invalidly cued target. Particularly, it would assess (i) the contribution of phase resetting and power to the invalidity effect, and (ii) possible frequency effects which are neglected in ERPs. Finally, this type of analysis would allow to assess the possible oscillatory mechanisms underneath the genesis of the ERPs. Particularly, given the strong endogenous character of the P3 component it is possible to expect the dependency on phase resetting of the P3 spatial invalidity effect. These approaches would allow to evaluate how the brain process the spatial invalidity occurring at the end of the preparation–perception–action cycle.

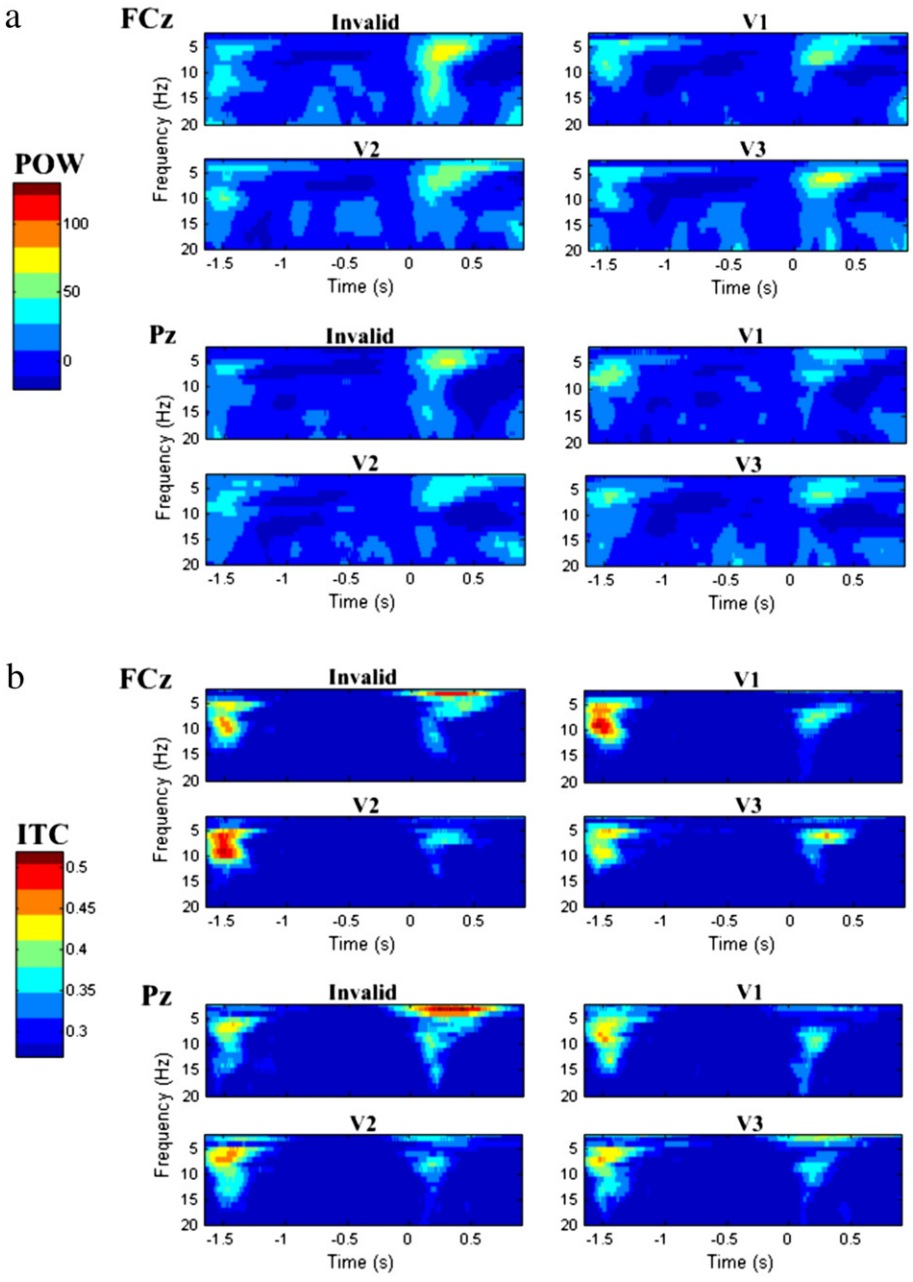

Fig. 3 – ERSP (a) and ITC (b) average over all subjects considering the target stimulus (S2) as trigger. Results for invalid and the 3 cases of valid conditions are presented in the frequency range of 2–20 Hz for electrodes FCz and Pz. Figures also depict responses to S1 stimulus around 1.5 s before S2.

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2. Results

2.1. Behavioural data

For the valid condition the mean RT was 349.7ms (SD = 85.5) and the percentage of errors was 5.7%. For the invalid condition the mean RT was 399.3ms (SD=90.1) and the percentage of errors was 9.8%. Comparison between the mean RT for both conditions (V vs I) using paired Student's *t* test for paired samples resulted in $t(15) = -4.9$, $P < 0.001$, indicating the benefit-cost pattern of the responses. There were also significant differences in the error rate between valid and invalid conditions ($t(15) = -2.3$, $P < 0.05$).

2.2. Event-related potentials

The invalid condition elicited a greater amplitude at all the recorded electrodes studied at P3a and P3b latencies (Fig. 1). A significant difference condition × component × electrode was observed when considering the 14 electrodes ($F(13, 195) = 6.9$, $P < 0.004$). A significant effect of condition ($F(1, 15) = 38.8$, $P < 0.001$) was also found, indicating the above mentioned greater amplitude of invalid condition. In addition, component factor was also significant ($F(1, 15) = 5.9$, $P < 0.03$).

These results confirm a statistically different topographic distribution of the mean voltage values at P3a and P3b latencies. As it can be appreciated in Fig. 2, comparing the slopes of the two lines, at electrode FCz the voltage increase in the invalid condition in relation to the valid is greater for P3a latency ($t(15) = 6.3$, $P < 0.001$) than for P3b ($t(15) = 2.7$, $P < 0.05$), although the interaction is only marginal significant (component × condition $F(1, 15) = 2.9$, $P = 0.1$). At electrode P4 the reverse is observed (Fig. 2b), the voltage increase in the invalid condition in relation to the valid is greater for P3b latency than for P3a (component × condition $F(1, 15) = 14.9$, $P < 0.05$, I vs V at P3b latency $t(15) = 4.9$, $P < 0.001$, I vs V at P3a latency $t(15) = 4.5$, $P < 0.001$).

2.3. Event-related spectral power change and event-locked phase coherency

Time frequency analysis revealed changes in spectral power and an increase in inter-trial coherence with respect to baseline at latencies related to stimuli S1 and S2 for all conditions at frequencies inside a frequency range from 2 to 20Hz (Fig. 3). No appreciable effects were observed from 20 to 40Hz.

In order to check the reliability of the data obtained through the wavelet analysis, statistical analyses were applied to a latency post S1 in which effects could be observed in Fig. 3. It was expected no statistical significant differences between invalid and valid trials, since at the appearance of the cue the subject doesn't know what stimulus will come next, therefore the neurophysiological processing of S1 should be similar for all trials. Results were consistent for both spectral power changes and ITC for all 3 comparisons: I vs V1, I vs V2, I vs V3 (being V1, V2 and V3 the three independent pools of valid trials randomly selected including the same number of trials as the invalid condition for each individual subject).

The ANOVA showed statistically significant event-related spectral power modulations (condition × electrode) for comparisons between invalid and valid conditions at latencies post S2. The results presented at Table 1 are the principal effects of the condition factor at two different electrode configurations: fronto-central (*F3/4, Fz, FCz, C3/4, Cz*) and parieto-ocipital (*P3/4, Pz, P7/8, O1/2*). Both significant increments and decrements of spectral power resulted from the analyses. In theta range, from 5 to 6Hz, ERSP(I) was greater than ERPS(V1) and ERPS(V3) at parieto occipital sites, from 100 to 250ms. In high alpha range, from 10 to 15Hz, an increment in ERSP(I) appeared with respect to ERSP(V1, V2, V3), from 150 to 300ms, in fronto-central electrodes. A deactivation in power for invalid condition was observed at late latencies. High theta and low alpha range (6 to 12Hz), from 600 to 800ms showed a significant decrement in ERSP(I) over the scalp in comparison with ERPS(V1) and ERPS(V2). The ERSP(I) deactivation was also statistically significant with respect to ERPS(V3), but only from 700 to 800ms at fronto-central sites. At 10 to 14Hz, from 400 to 600ms, a significant decrease in ERSP(I) with respect to ERSP (V1, V2, V3) appeared at posterior sites. Fig. 4 shows the temporal evolution for 10–14Hz ERSP and 6–12Hz ERSP from 0.2s before S2 to 1s after S2 at three electrodes selected from the fronto-central set and three electrodes from the parieto-posterior set used in the statistical analyses.

Statistical analyses for ITC yielded to significant difference between valid and invalid conditions with the invalid presenting a greater phase-locked effect than the valid at P3a and P3b response latencies, from 250 to 450ms post S2, at low frequencies (Table 2, Fig. 5). Since the pair comparisons were made using always the ITC at the same electrode for the invalid and valid conditions, ex. ITC(F3.Invalid) compared with ITC(F3.Valid), and never between different electrodes, ex. ITC (F3.Invalid) compared with ITC(FCz.Valid), the multiple comparisons are orthogonal and therefore independent. In this case, there is no need to use a correction like Bonferroni (see

Table 1 – Repeated-measures ANOVA (Condition× Electrode) results for the factor condition on comparison of event-related spectral power (ERSP) between invalid condition (I) and each of the 3 valid conditions (V1, V2, and V3)

ERSP (I) > ERSP (V)		V1: $F(1,15)$	V2: $F(1,15)$	V3: $F(1,15)$
P3/4, Pz, P7/8, O1/2				
5–6Hz	100–250ms	11.84^{++}	*n.s.*	5.00^{+}
F3/4, Fz, FCz, C3/4, Cz				
10–15Hz	150–300ms	13.48^{++}	7.12^{+}	6.05^{+}
ERSP (I) < ERSP (V)				
P3/4, Pz, P7/8, O1/2				
10–14Hz	400–600ms	9.39^{++}	9.74^{++}	5.94^{+}
6–12Hz	600–700ms	10.84^{++}	11.54^{++}	*n.s.*
6–12Hz	700–800ms	7.97^{+}	8.68^{++}	*n.s.*
F3/4, Fz, FCz, C3/4, Cz				
6–12Hz	600–700ms	4.92^{+}	15.88^{+++}	*n.s.*
6–12Hz	700–800ms	8.70^{++}	22.95^{+++}	6.40^{+}

$^{+++}P < 0.001$, $^{++}P < 0.01$, $^{+}P < 0.05$ and $^{-}P > 0.01$.

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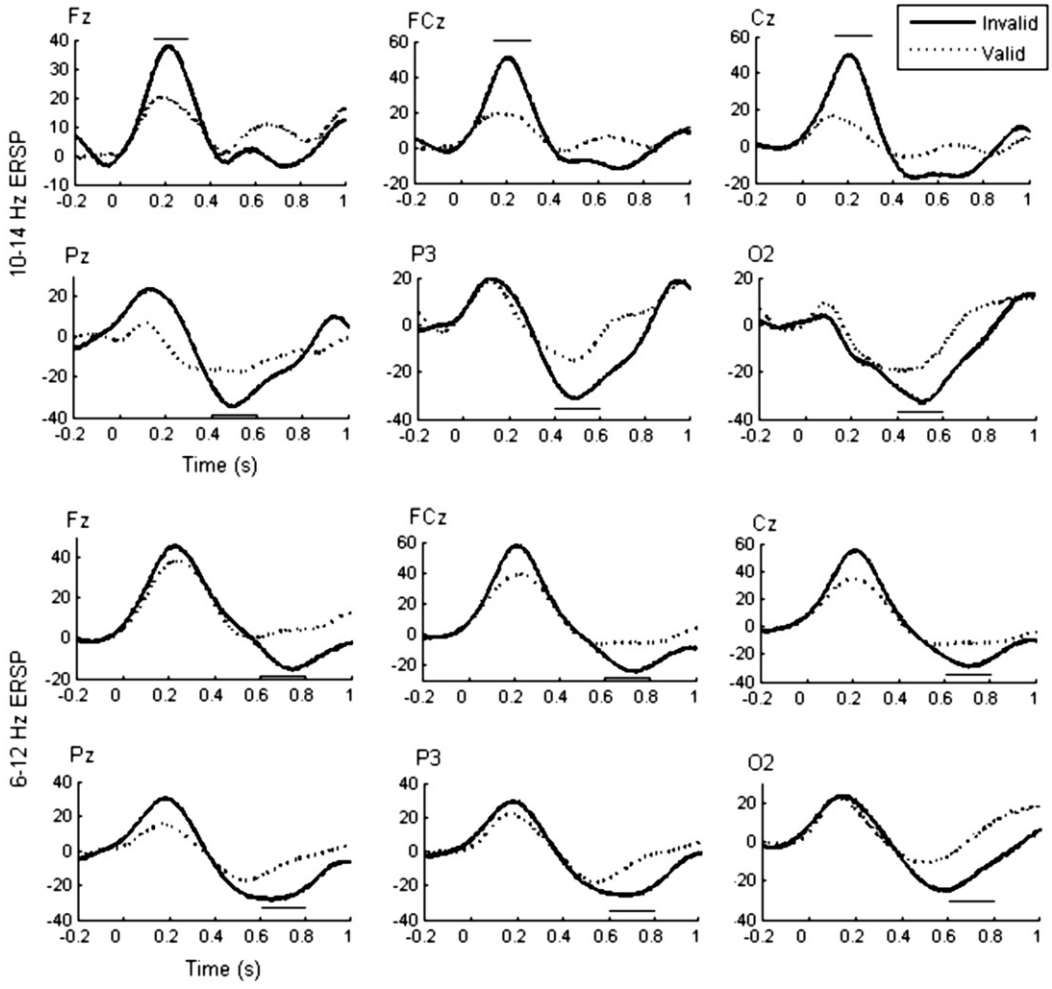

Fig. 4 – ERSP temporal evolution from 0.2 s before to 1 s after S2 for frequency ranges from 10 to 14 Hz and from 6 to 12 Hz at electrodes Fz, FCz, Cz, Pz, P3 and O2. The horizontal lines indicate the time window used for statistical analyses when the results yielded a statistical difference between conditions invalid and valid.

Keppel and Zedeck, 1989; Kirk, 1995). Also, since at low frequencies the spread effect caused by the wavelet length is greater, so the temporal definition is less exact than for higher frequencies, we chose for the statistical analysis the latency in which the effect was greater and ignored earlier or later latencies. Therefore we performed the analysis only from 250 to 450ms.

At 2Hz, in centro parietal electrodes, the increment in ITC (I) started. At 3Hz, the effect consolidates for all the 3 valid cases showing a significant difference in electrodes F3, Fz, F4, FCz, C3, Cz, C4, P7, P3, Pz, P4, P8. At 4Hz, there was a consistency among the 3 comparisons (V1 vs I, V2 vs I, V3 vs I) for parietal electrodes. For fronto-central electrodes not all differences were significant and the significances were

Table 2 – Wilcoxon matched-pairs signed-rank test results on comparison of inter-trial coherence (ITC) between invalid condition (I) and each of the 3 cases of valid conditions (V1, V2, and V3) at P3a and P3b response latency peaks (250–450 ms)

		F3	Fz	F4	FCz	C3	Cz	C4	P7	P3	Pz	P4	P8	O1	O2
2 Hz	V1	–	–	–	–	+	+	+	++	+	+	+	–	+	–
	V2	–	–	–	–	+	+	+	++	+	+	++	++	–	+
	V3	–	–	–	–	+	–	–	–	–	–	–	–	–	–
3 Hz	V1	++	+++	+++	+++	+++	+++	+++	++	++	+++	+++	++	–	–
	V2	+++	+++	+++	+++	+++	+++	+++	++	+++	+++	+++	++	+	+
	V3	++	+++	+++	+++	++	+++	+++	+	++	+++	++	+++	–	+
4 Hz	V1	+	++	+++	++	+++	++	++	+	+++	+++	++	+++	–	–
	V2	++	+	+	–	++	–	+	++	+++	++	+++	+++	–	–
	V3	–	+	++	+	+++	++	+++	++	+++	+++	+++	+++	–	–

ITC (I)>ITC (V1, V2, and V3). $^{+++}P<0.001$, $^{++}P<0.01$, $^{+}P<0.05$ and $^{-}P>0.01$).

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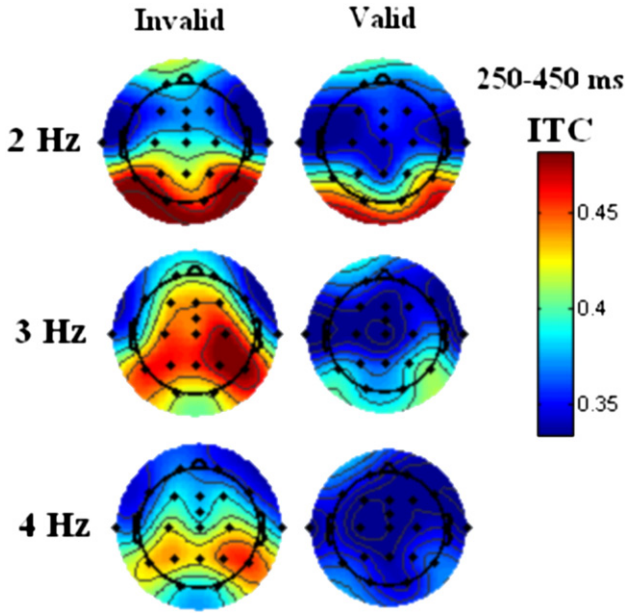

Fig. 5 – Maps of ITC topographic distribution for invalid and valid (V1) conditions at 2, 3 and 4 Hz from 250 to 450 ms.

generally weaker than at 3Hz, as if the effect in this topography was fading away. Interestingly, there were no significant effects of increase of power at these low frequencies for the invalid condition compared to the valid ones, as can be see in Fig. 6. This result seem to suggest that the P3 increase found in the invalid condition is produced by phase resetting only, without concomitant increase of power. In addition, no significant differences were found for 6, 7 and 8Hz from 250 to 450ms. The time-frequency windows 5–6Hz and 7–10Hz from 100 to 200ms, and 10–15Hz from 150 to 250ms were also analyzed but no significant effects were found.

3. Discussion

Present results showing a P3a and P3b modulation by invalidly cued targets complement a previous study (Gómez et al., 2008) evaluating the neurocognitive processing of invalidly cued targets. In accordance with this study we found an increased P3a and P3b response for the invalidly cued targets and, moreover, we obtained significant differences of ERSP and ITC between invalid and valid conditions for several ranges of frequencies in specific time windows.

Comparing the ERPs elicited by the invalidly(I) and validly (V) cued targets, the effect of the I targets upon endogenous late ERP components was clear. The difference wave topography for the early P3 and the interaction among the ANOVA factors Condition, Component and Electrode suggest the presence of a P3a component in I trials followed by a late increase of P3b at posterior electrodes. These results reinforce the ideas presented before by Gómez et al. (2008) suggesting that the subjective bias induced by the cueing creates expectancies for the cued location and induce the subject to treat I targets in a similar manner to low frequency targets in odd-ball paradigms. The increased P3a would index the processing of the I target as a novel stimulus (Friedman et al., 2001; Dien et al., 2003) and the increased P3b would represent the context updating of the working memory (Donchin and Coles, 1988), in this case the updating of the S1–S2 contingencies.

Although "traditional" measures (i.e. amplitude and latency) in the study of brain electrical activity are important, several studies have shown that spectral analysis, especially single trial time-frequency wavelet based, can provide key measures in understanding brain electrical activity. Therefore the increase/decrease of power and inter-trial phase alignment at certain frequencies have been revealed as key parameters in the ERPs description. In this line, it has been proposed that some ERPs could be generated, not by a fixed latency amplitude response (classical view), but as a result of changes in the oncoming EEG (see, i.e., Makeig et al., 2002; Fuentemilla et al., 2006). Hence, ERPs would be characterized by increased inter-trial phase coherence (ITC) at certain frequencies, but not necessarily by increase in the power (Fuentemilla et al., 2006). It has also been proposed that the modulation of both mechanisms (increase/decrease of power and ITC) can reflect different neuronal states (David et al., 2005). ERPs presenting only phase resetting without increase of power might arise from stimulus related ongoing activity, whereas low ITC activity might generate the ERPs in the basis of the classical view. Therefore, the finding of ERPs generated only by phase resetting would indicate an increase in the stimulus related or ongoing neural activity. In a recent work, Fuentemilla et al. (2006) showed that certain ERPs could arise exclusively by reorganization of the phase of the ongoing scalp activity. The authors found that the N1 ERP in response to the repeated auditory stimuli was generated by a partial phase resetting in the theta/alpha band without concomitant increase of power. Moreover, Fuentemilla et al. (2008) proposed that the temporal component of the MMN was generated in bases of phase resetting, while the frontal component also presented increase in power at the same frequency band. In addition, Cheron et al. (2007) found that

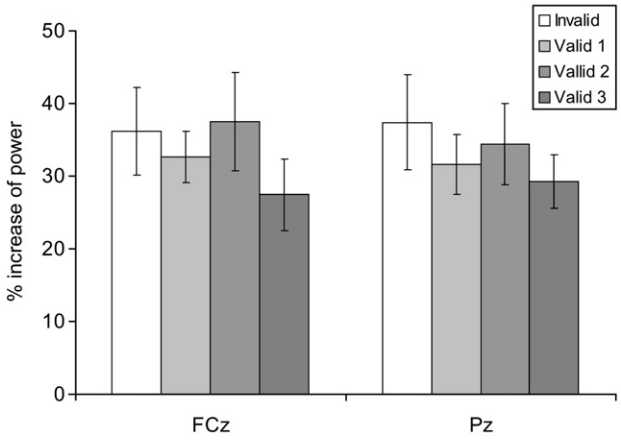

Fig. 6 – Increase of power for the invalid and the three pools of valid conditions in the 2–4 Hz frequency range between 250 and 450 ms for FCz and Pz. None of the invalid vs valid comparisons yielded significant results.

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large proportions of the trials generating the N30 ERP were generated by pure phase locking.

Present results suggest that the increased P3 response in I trials was formed exclusively by phase resetting since the statistical analyses yielded to significant differences between I and V trials at P3 latency for ITC but not for ERSP. The 2-factor ANOVA carried out between ERSP for invalid and each of the 3 valid conditions, for the 2 sets of electrodes, from 250 to 450 at low frequencies, produced very similar results indicating the high reliability of this result. Considering that the P3a responses are fronto-central and the P3b are parietal (Polich, 2007), the ITC topography suggest that P3a responses are centred around 3Hz while P3b responses might involve a wider range of low frequencies, from 2 to 4Hz. This result would again support a critical role of the phase resetting (indexed by ITC) in the generation of the ERPs. Following David et al. (2005), the increase in the phase resetting without increase in the power would arise from nonlinear interactions in the neural networks characteristics of the neural networks generating the EEG due to a high level of stimulus related or ongoing activity. Therefore, two main mechanisms seem to take part in the genesis of the P3 response: an extra neuronal contribution superimposed to the background EEG activity as reflected by the power enhancement; and an increase in phase concentration of EEG ongoing oscillations as revealed by the increased low frequency ITC at P3 latency. Nevertheless, the effect caused by the invalidity of the cue seems to only increase the phase resetting without enhancing the spectral power as we discussed above. Shah et al. (2004) suggested that top-down influences would appear in the ERPs as a consequence of phase resetting rather than by power modulation They proposed that this mechanism would be particularly suited for P3 generation. In the same sense Digiacomo et al. (2007) showed a correlation between theta amplitude and the P3 component. Therefore, present results suggest a key role of phase realignment of EEG ongoing activity in the generation of late cognitive ERP components, as in the case of P3 component.

The spatial cueing effects on the attentional sensory processing have been previously evaluated by analyzing the modulation of the ERPs to valid and invalidly cued target (Mangun and Hillyard, 1991; Eimer, 1993; Perchet and García-Larrea, 2000; Perchet et al., 2001). The general result obtained was an increase of the P1 and N1 and a decrease of posterior P3 components in valid cued trials with respect to invalid ones. In the present experiment, given the low number of invalid trials and although some P1 valid effects were visually observed they did not reached statistical significance. The wavelet technique is more sensitive than ERPs, given that it does not cancel the induced activity. The early increased posterior ERSP obtained in posterior sites for invalidly cued targets in the theta range might be interpreted as an increased processing of invalid targets. This processing would include the necessary reorienting attention needed to fully visually process the previously unattended target and might have been undetected by the ERP techniques if the brain response to uncued targets is not tightly linked to the stimulus.

ERSP increased modulation in invalid targets with respect to valid, presented a major effect centred in the theta and alpha ranges in latencies around 100–300ms, for both theta (5–6Hz) at posterior sites and high alpha (10–14Hz) in anterior sites. On the other hand, late latency alpha presented a power decrease at posterior sites from 400 to 800ms period. In frontal electrodes the alpha decrease occurred delayed with respect to posterior sites (from 600 to 800ms). Klimesch et al. (2007) suggested that alpha oscillatory activity is induced by inhibitory cells and reflects rhythmic changes between phases of maximal and minimal inhibition. Therefore, desynchronized alpha activity reflects a state of comparatively high excitability, whereas synchronized alpha activity reflects a state of inhibition. Hence, alpha ERD may not primarily reflect bottom-up sensory processing but may be triggered by top-down processes. In present experiment, the increase in 10–14 ERSP(I) at fronto-central electrodes around 200ms may reflect a top-down inhibitory cognitive-motor process to hold the incorrect response prepared during the CNV period, which may reflect a state of reduced information processing over brain areas which are not task-relevant or where a learned response must be withhold in order to perform a task (Klimesch et al., 2007). Hummel et al. (2002) observed ERS in the upper alpha range (11–13Hz), reflecting the mu rhythm, when acquired motor programmes needed to be inhibited. Therefore, the increased high alpha at fronto-central sites would be related to inhibition of the response activated during the preparatory period (Eimer, 1993; Gómez et al., 2004).

Low alpha showed a significant decrease in the invalid condition ERSP compared to valid condition ERSP starting at 500ms and peaking around 700ms at fronto-central electrodes, and starting at 400ms and peaking around 600ms for posterior electrodes. The high alpha range presented an increase in invalid condition ERSP peaking around 200ms followed by a decrease starting at 400ms at fronto-central electrodes. According to Klimesch et al. (2007), alpha ERD is not a unitary phenomenon, but presents distinct types of task-related reactivity with different frequency bands and topographies. Although its functionality is not completely understood, it has been associated with directed attention and stimulus classification (Klimesch et al., 1993; Sterman, 1996), resource allocation (Sergeant et al., 1987; Dujardin et al., 1995; Spencer and Polich, 1999), memory retrieval and recall (Dujardin et al., 1995; Krause et al., 1995), or unspecific arousal reactions (Boiten et al., 1992). In addition, posterior alpha suppression has been found during attentional visual paradigms involving expectancy and spatial attention (Gómezet al., 1998, 2006; Vázquez Marrufo et al., 2001; Digiacomo et al., 2007). Interestingly, Yordanova et al. (2000) found a greater alpha ERD for the odd target stimuli compared to the frequent non-target stimuli. In addition, they suggested that alpha ERD may index a subsequent processing stage guided by stimulus relevance. Our similar results comparing validly and invalidly cued targets reinforce the idea that the invalidly cued targets are processed in a similar way to the odd target stimuli.

In conclusion, time-frequency analysis applying complex wavelet yielded interesting results in the analysis of the neuronal responses elicited by the central cue Posner paradigm. In summary, we found that the P3 difference in invalid condition was generated by an increase in the ITC. In addition, we found a complex alpha power modulation pattern in the imperative stimulus related to the invalidity of the cue. Further analysis has to be done in order to better understand

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the role of these responses in the preparation–perception–action cycle.

4. Experimental procedures

4.1. Subjects

Sixteen participants (9 women, 15 right-handed) between 19 and 23years old (mean age 20.6years old, SD = 1.03) took part in the experiment. All participants were undergraduate university students. Experiments were conducted with the informed and written consent of each participant following the Helsinki protocol.

4.2. Stimuli and behavioural paradigm

The stimulus presentation was computer-controlled by the software EEVOKE (ANT, Enschede, The Netherlands). Participants were seated 0.50m away from a computer screen. A white fixation square was on during the whole experiment. The complete trial period included a central directional cue (S1) lasting 300ms, an attentive waiting period of 1360ms, and a subsequent peripheral target (S2) lasting 300ms and subtending a visual angle of 4.56° situated 3.66° eccentrically in the horizontal meridian (Fig. 7). The targets were squared cartoon figures. A different figure was presented in each block of trials. The whole experiment consisted of 240 trials divided in 5 blocks, with an inter-trial interval of 1860ms, including 10 training trials. Since the central cue could indicate the correct or the incorrect direction in which the target would appear, two different conditions arose: validly cued targets (V) (82.1% of trials) and invalidly cued targets (I) (17.9% of trials). The targets appear randomly with a probability of 0.5 in the right or left hemifield for both conditions. The subjects used the right index finger to respond to right targets by pressing a joystick button, and the left index finger to respond to left targets.

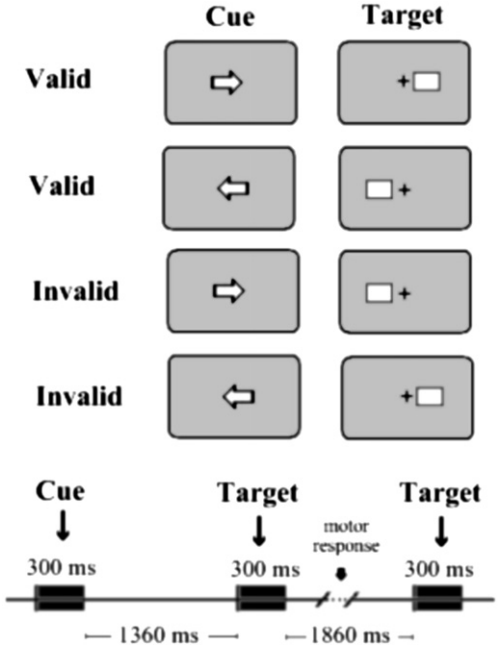

Fig. 7 – Experimental paradigm. ERPs, ERSP and ITC were obtained from the target (S2) stimulus.

4.3. Electrophysiological recording

The EEG was recorded from 20 scalp sites of the International 10–20 system (Fp1/2, F3/4, F7/8, Fz, FCz, T7/8, C3/4, Cz, P7/8, P3/4, Pz, O1/2), using tin electrodes mounted in an electrode cap (EASYCAP, Herrsching-Breitbrunn, Germany) with two additional electrodes (M1/2). Ocular movements (EOG) were recorded from two electrodes at the outer canthus of each eye for horizontal movements and one electrode under the left eye for vertical movements. EOG correction (ASA software, ANT) was applied off-line to the EEG recordings. All the electrodes were referred to the left mastoid (M1). Impedance was maintained below 5kΩ. Data were recorded in DC at 512Hz, with a 20,000 amplification gain using a commercial AD acquisition and analysis board (ANT).

4.4. Event-related potential data analysis

ERPs were obtained off-line by averaging EEG epochs from 0.2s before S2 (baseline period) to 1s after S2. An off-line Fast Fourier Transform 20Hz low-pass filter with an edge width of 0.2Hz (ASA software) was applied to the data. All the epochs for which the EEG exceeded ± 100 microvolts in any channel were automatically discarded. A total of 2683 valid trials (85.2%) and 565 invalid trials (82.60%) were accepted for the analysis. Three-factor repeated measures ANOVAs with Condition (Valid/Invalid), Component (260–320ms and 320–380ms corresponding respectively to P3a and P3b) and Electrode as within subject factors were performed on the mean voltage. The factor Electrode had 14 levels: F3/4, Fz, FCz, C3/4, Cz, P7/8, P3/4, Pz, O1/2. P values were calculated by using the Greenhouse–Geisser correction when appropriate.

4.5. Event-related spectral power modulation and inter-trial phase coherence analysis

To study time-frequency behavior of the electrical activity elicited by the invalid and valid targets, single trials (from 2.5s before to 1.5s after S2) were convoluted time locked to the target stimuli using a complex Morlet wavelet:

$$w(t,f) = \left(2\pi\sigma_t^2\right)^{-1/2} e^{\frac{-t^2}{2\sigma_t^2}} e^{2i\pi ft}$$

The relation f/σ_f (where $\sigma_f = 1 / (2\pi\sigma_t)$) was set to 6.7 (Tallon-Baudry et al., 1997).

Changes in time varying energy (square of the convolution between wavelet and signal) in the studied frequencies (from 1Hz to 40Hz; linear increase) were computed for each trial

$$SP_k(f,t) = |w(t,f) \otimes S_k(t)|^2$$

(where $s_k(t)$ is the EEG signal at the kth trial, || represents the complex norm and ⊗ is the complex convolution). Event-related spectral power modulations (increase or decrease of power respect to baseline) was analyzed in relation to averaged spectral power in the baseline period of 0.2s before

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the target stimulus S2 after averaging the spectral power of all trials (SP(*f*,*t*)) as:

$$\mathrm{ERSP}(t,f) = \frac{\mathrm{SP}(f,t) - \mathrm{SP}(f,t)_{\mathrm{baseline}}}{\mathrm{SP}(f,t)_{\mathrm{baseline}}} \times 100$$

In addition, the inter-trial phase coherence (ITC) was also computed to study the degree of coincidence of the phase of the different trials:

$$\mathrm{ITC}(f,t) = \frac{1}{N}\left|\sum_{k=1}^{N}\frac{w(t,f)\otimes S_k(t)}{|w(t,f)\otimes S_k(t)|}\right|$$

being *N* the total number of trials. Time frequency computation were performed under Matlab© 7 (Mathworks, Natick, MA).

In order to compare ERSP and ITC between conditions valid and invalid, the number of valid trials for each subject used in the average was adjusted to be the same as in the invalid condition. A total of 506 invalid trials (74% of all invalid trials) were accepted for the analysis. Since the valid condition appeared in 82.1% of trials, and to avoid effects due to the different number of trials in the valid condition than in the invalid condition, three independent pools of valid trials (V1, V2, V3) were used. For each subject, the three pools containing the same number of trials than the invalid condition were created by randomly selecting trials from the valid condition. With this method we ensure that the number of trials of the valid and invalid condition was the same and that the effects found could not be due to an effect of the size of the samples. In addition, the existence of three independent randomly selected pools guarantees that results found are robust and not dependent on the random trials selected.

Two-factor repeated measures ANOVA, with the condition (valid–invalid) and electrode (fronto-central including F3/4, Fz, FCz, C3/4, Cz and parieto-posterior including P7/8, P3/4, Pz, O1/2) as factors was used two establish the differences between the invalid and valid conditions (Kiebel et al., 2005). Comparisons were performed with the three pools of data with the valid conditions containing the same number of trials as the invalid one. Time and frequency windows chosen for the analyses were selected based on ERPS time-frequency figures for each selected electrode (see in Results Fig. 3a).

For ITC statistical analysis the non-parametric Wilcoxon matched-pairs signed-rank test was used to avoid false assumptions about data distribution. Since ITC is normalized its values range from 0 to 1, with values near to 1 meaning near-perfect EEG phase coherence across trials. Wilcoxon tests were applied on the mean ITC values in selected time and frequency windows between the invalid condition and each of the valid conditions for all electrodes. As for ERSP, the time and frequency windows were chosen based on ITC time-frequency figures for each selected electrode (see in Results Fig. 3b).

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