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Fast Track Report Emotional expressions modulate low α and β oscillations in a cortically blind patient $\stackrel{_{\sim}}{\approx}$

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ABSTRACT

Studies of cortical blindness have suggested that some residual visual function may persist without perceptual awareness, a condition known as blindsight. To investigate electrophysiological evidence of unconscious processing of emotional stimuli, we examined the event-related oscillations (EROs) in a 62 year-old male patient (TN) with affective blindsight during random stimulation of three facial expressions (fearful, happy and neutral). Spectral power analysis in response to the different emotions revealed significant differences between fearful and happy faces over the right frontal regions at 7–8 Hz (low α), and between emotional and neutral faces over the left frontal sites at 12–13 Hz (low β) in a time period between 100–400 ms after visual stimulus onset.

These results demonstrate that emotional face processing occurs very early in time in the absence of any functional striate cortex, and further reveals the existence of specific oscillatory frequencies that reflect unconscious processing of facial expressions in affective blindsight.

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1. Introduction

The capacity to discriminate visual stimuli without any perceptual awareness following lesions in the primary visual cortex (V1) has been called "blindsight" (for overviews Kentridge et al., 1999; Weiskrantz, 1986, 1996, 1997, 2001, 2003). Previous experiments with a hemianopic blindsight patient described a specific phenomenon called affective blindsight, in which patients retain the capacity to correctly guess the expression on a face presented to their blind field (De Gelder et al., 1999, 2001). An ERP study in a blindsight patient, GY, revealed that P1 and N1 components for emotional faces were smaller and delayed on mesial-occipital electrodes placed over the impaired hemisphere, compared to those placed over the healthy hemisphere (De Gelder et al., 1999). However, no difference was reported for emotional expressions presented to the blind visual half field.

By contrast, the first fMRI investigation of a bilateral blindsight patient (TN) showed that, despite the absence of any conscious visual processing by the patient, a strong right amygdala activation occurred in response to emotional compared to neutral faces (Pegna et al., 2005). This finding was in line with the study of patient GY (Morris et al., 2001), although in this case, the patient suffered from a hemianopic defect, and bilateral amygdala activation was observed for fearful faces presented in his blind field.

Electrophysiological data was subsequently recorded in patient TN and multivariate pattern analysis was applied to the source-localisation algorithms computed from these data (Gonzalez Andino et al., 2009). This investigation, using passive viewing of different emotional faces, suggested the possibility of an emotion-specific response (fear vs. neutral, happy and angry face) beginning at 120 ms after stimulus onset in the right anterior cerebral areas which was followed by right amygdala activation at around 200 ms in response to emotional faces (e.g. happy, angry, fearful vs. neutral faces). To date, this is the only published study on a patient with complete cortical blindness that specifically compared categories of emotional faces.

Recently, electrophysiological findings in healthy controls (Smith, 2011) showed that emotional faces produce an early modulation at around 170 ms. Combining event-related potential analysis (ERPs) with time-frequency analysis of event-related oscillations (EROs), Zhang et al. (2012) also found a greater N170 for fearful compared to neutral faces that increased with awareness of the stimuli. This effect was significantly correlated with a theta power (4–8 Hz) increase within 250 ms of stimulus presentation. They claimed that the parallel modulation of the N170 component and theta synchronization over occipital parietal areas may reflect awareness of rapidly presented fearful faces.

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This report further showed a high interindividual variability for consciousness during backward masking and suggested a relatively early (<200 ms) neural response to unconscious emotional faces. By contrast, a similar study of Balconi and Mazza (2009) with seven facial expressions reported alpha (8–15 Hz) brain modulation in response to masked emotional facial expressions. Specifically, a decrease (desynchronization) in alpha activity was found in response to: i) negative (angry and surprised) facial expressions over the right-frontal region; ii) facial stimuli over anterior frontal compared with central and posterior sites; and iii) positive emotions (happiness) over the left hemisphere. These findings were interpreted as reflecting the interplay between inhibition and activation brain responses in the context of approach vs. withdrawal behaviour towards emotional stimuli.

EEG rhythms are therefore clearly modulated by the presentation of emotional stimuli and alpha oscillatory activity appears to play a fundamental role in the cognitive processing during attentional as well as memory tasks (for a review see Başar and Güntekin, 2012). However, the EEG oscillatory response to emotional faces has not yet been studied in patients with affective blindsight.

Here we present an EEG study with patient TN, which aimed at establishing the electrophysiological oscillatory activity underlying the processing of three facial expressions (positive, negative and neutral faces) in blindsight using an active task. We aimed to investigate the time course of the ERO activity underlying non-conscious processing of facial expressions using high-density EEG recordings. Since gamma is linked to visual awareness according to several studies involving healthy controls (Tallon-Baudry et al., 1997; Zhang et al., 2012) and in patients with hemianopia (Schurger et al., 2006, 2008), our analysis focused on theta, alpha and beta frequencies.

2. Materials & methods

Patient TN is a 62 year old male who suffered two consecutive strokes at the age of 52, the first in the left parietal-temporal-occipital cerebral areas, and the second in the right occipital lobe producing a loss of the remaining left visual field (for anatomical details see paragraph 1 in "Supplementary Material" and Buetti et al., 2013). Clinically, TN was completely blind and unable to detect colours or geometric shapes or even the presence of light source at the time of testing. Interestingly, despite this lack of conscious vision, we observed that he could guess above chance the emotional expression on a face (Pegna et al., 2005), point to a visual stimulus with an above-chance probability (Buetti et al., 2013), guess whether a face was making eye-contact (Burra et al., 2013; De Gelder et al., 2008; Pegna et al., 2005) or even navigate down a path while avoiding obstacles (De Gelder et al., 2008). These residual capacities occurred without any awareness.

The current study was approved by our local Ethics Committee. Prior to testing, the consent form was read to the patient in the presence of his wife who verified its content before signature. A visual perimetry performed on the day of the experiment confirmed that TN was unable to report the presence of any visual stimulus confirming his cortical blindness. Clinically, no auditory deficit was observed, nor was any reported by the patient.

2.1. Stimuli and procedure

Visual cue-stimuli consisted of 60 pictures of emotional cropped faces divided into 3 categories: fear (F), happiness (H) and neutral (N), with 20 different models by category (half females). As the patient was unwilling to guess at the time of the test, we used an attentional visual cuing paradigm rather than a task requiring explicit guessing. In this paradigm, this emotional face was presented in the centre, left or right side of the screen for 1000 ms, followed by a random interval ranging between 100 and 400 ms. This was followed by a 500 Hz acoustic stimulus of 250 ms duration that was delivered to the left or right ear (50% on each side) by means of a pair of headphones, to which the patient had to

respond (for details, see Fig. S1a in Supplementary Material). The following trial was initiated 1500 ms after the target. To monitor the gaze fixation of the patient, we used a webcam placed on the monitor in front of the participant. Additionally, an experimenter was located under the screen during all experimental session.

The investigation was performed according to the Declaration of Helsinki on Biomedical Research Involving Human Subjects. The protocol was approved by the local Ethics Committee and informed consent of the patient was obtained.

2.1.1. EEG recording parameter

Electroencephalogram (EEG) was continuously recorded during the entire experimental session using a 128 scalp electrodes cap at a sampling rate of 2048 Hz (BioSemi Active Two, Amsterdam, Netherlands; for more details see Supplementary Material, Fig. S1b).

2.2. Time frequency-domain analysis

EEG data analysis was performed using BrainVision Analyzer 2 software (Brain Products GmbH). Continuous EEG data were down sampled to 512 Hz and corrected for eye movement artefacts through an independent component analysis (ICA) (Jung et al., 2000). For EEG power analysis, data were segmented into cue-locked epochs of 4000 ms, starting 1000 ms before cue onset. The epochs were visually inspected for artefacts and only artefact-free EEG trials corresponding to correct responses were subsequently analysed, totalling 281 trials for fearful faces, 272 trials for happy faces, and 265 trials for neutral faces.

We performed a time-frequency (TF) analysis based on a continuous wavelet transform of the signal (complex Morlet's wavelets) between 4 and 30 Hz in 1 Hz step (e.g. Deiber et al., 2012; Tallon-Baudry et al., 1998), corresponding to theta, alpha and beta frequency bands. The mean power of the pre-cue stimulus interval (-650 ms to -150 ms before cue onset) was considered as a baseline level independently for each frequency (for details see "Methods" in "Supplementary Material").

2.3. Statistical analyses

2.3.1. EEG data

We performed an initial exploratory time-point by time-point *t*-test analysis, in order to determine the range of frequencies to be investigated (Michel et al., 2004). The EEG oscillatory power of each of the 3 emotional conditions was computed at every time point in the 3000 ms epochs of all trials, time-locked to stimulus onset. This was performed at every frequency from 4 to 30 Hz on all 128 electrodes. Facial expressions were then contrasted using the running *t*-test which compared the conditions 2 by 2 at every consecutive time-point. Using a threshold at p = .01 (uncorrected), 4 frequencies emerged as possible frequencies of interest: 7 Hz, 8 Hz, 12 Hz and 13 Hz (see Supplementary Figure S2).

In order to explore interactions across the different factors, we carried out an ANOVA analysis restricted to the four frequencies. We defined 9 regions of interest (ROIs) covering the main scalp regions (Fig. S1b in Supplementary data), and divided the epoch into six 50 ms-timewindows from 100 ms after cue onset (i.e., from 100 to 400 ms). The differences in oscillatory power across emotional conditions were established by entering the power of the four sensitive frequencies (7 Hz, 8 Hz, 12 Hz and 13 Hz) in every trial into a repeated measures ANOVA using $3 \times 6 \times 3 \times 3$ factors: 3 facial emotions (E), 6 time windows (T), 3 regions along the left-right axis (Hemisphere, H: right, central and left) and 3 regions along the antero-posterior axis (AP: frontal, central, and occipital). Thus, the number of repetitions in each category was determined by the number of trials retained. Greenhouse-Geisser corrections and post-hoc Tukey HSD comparisons, with a statistical threshold of p < .05, were used in both behavioural and electrophysiological analysis.

3. Results

We calculated TN's individual alpha frequency (IAF) over contiguous epochs of 4s (independently of triggers and responses) and used this as a cut-off point for lower and upper alpha bands. We obtained a value of IAF = 8.5 Hz. According to the bandwidth definition by Klimesch (1999), TN's alpha frequency thus ranged from 4.5 to 10.5 Hz, defined as follows: lower 1 alpha from 4.5 to 6.5 Hz; lower 2 alpha from 6.5 to 8.5 Hz; upper alpha from 8.5 Hz to 10.5 Hz. Thus, 7 Hz and 8 Hz frequencies belong to α -band while 12 Hz and 13 Hz frequencies belong to β -band.

At 7 Hz and 8 Hz, the three-way interaction of " $E \times H \times AP$ " was significant ($F_{[8, 2112]} = 3.58$, MSE = 10.49, $\epsilon = .47$ p < .008 and $F_{[8, 2112]} = 2.49$, MSE = 12.24, $\varepsilon = .56$ p < .036 respectively), denoting a decrease of power for fearful compared to happy faces only over the right frontal regions. Data confirmed the significant difference between fearful and happy faces at both frequencies (7 Hz: p < .03; 8 Hz: p < .002Tukey HSD post-hoc test), but not between fear and neutral (p = .077) or between happy and neutral (p = 1) faces (Fig. 1a and b). Both frequencies decreased significantly over frontal compared to central and occipital regions [AP factor: 7 Hz ($F_{[2, 528]} = 31.38$, MSE = 24.36, $\epsilon = .79~p < .0000)$ and 8 Hz (F_{[2, 528]} = 67.45, MSE = 24.54, $\epsilon = .87$ p < .0000] and over right compared to left and central areas [H factor: 7 Hz ($F_{12, 5281} = 30.54$, MSE = 24.87, $\varepsilon = .97 \text{ p} < .0001$) and 8 Hz $(F_{12, 5281} = 3.69, MSE = 36.30, \epsilon = .996 p < .026)]$. At 8 Hz level the three-way interaction of " $E \times T \times AP$ " ($F_{120,52801} = 4.003$, MSE = 3.25, $\epsilon = .31 \text{ p} < .0005$) showed that the maximum decrease in power for fearful faces (compared to happy) occurred in the time range between 250-300 ms over the frontal areas (post-hoc comparison: p < .0001).

At 12 Hz, the three-way interaction of " $E \times H \times AP$ " ($F_{(8, 2112)} = 2.67$, MSE = 12.27, $\varepsilon = .59 \text{ p} < .023$) was also significant, revealing a greater power for neutral compared to fearful and happy faces. As shown by the Tukey HSD post-hoc tests, the difference between fearful and neutral, as well as between happy and neutral faces were significant over central (both comparisons: p < .001) and left frontal regions (fear vs. neutral p < .02; happiness vs. neutral p < .001). In contrast, there was no difference between fearful and happy faces over these areas (p = 1).

At 13 Hz no significant effects were found in four-way ANOVAs. Consequently, we computed three-way ANOVAs on every time window separately. This revealed a specific effect across emotions (E factor per se: $F_{12, 5281} = 3.39$, MSE = 2.881, $\varepsilon = .97 \text{ p} < .036$) only in the time range between 200 ms and 250 ms after the onset, in which the power was lower for fearful and happy faces when compared to neutral faces (fear vs. neutral: p < .02; happiness vs. neutral: p < .05; fear vs. happiness: p = .65; Fig. 1a and b).

4. Discussion

The present investigation explored the event-related oscillations in a cortically blind patient with affective blindsight during the presentation of positive and negative as well as neutral facial expressions.

Although the patient could not report seeing the stimuli, oscillatory modulations were found in low alpha (7–8 Hz) and low beta power (12–13 Hz) over frontal leads in relation to different facial expressions. Fearful (compared to happy) faces decreased the power of frequencies at 7 and 8 Hz over the right frontal lobes between 100 and 400 ms after cue onset. Additionally, both happy and fearful (vs. neutral) expressions decreased the power of the frequencies at 12 and 13 Hz over the left frontal lobes reaching a maximum between 200 and 250 ms, especially for 13 Hz.

To date, there are no studies investigating oscillatory activity of emotional face processing and their specific frequencies in bilateral blindsight patients. In healthy controls, spectral power modulations have been found in response to emotional faces in several studies although no consensus has emerged regarding the exact frequency bands concerned. For instance, Schutter et al. (2001) observed a strong asymmetry between right and left parietal leads in the beta (15–30 Hz) frequency range during selective attention for angry faces. Other reports (Balconi and Lucchiari, 2006; Balconi and Pozzoli, 2009) have suggested



Fig. 1. a) Grand mean time-frequency plots displaying the relative change in the spectral power of EEG signal between 4 and 16 Hz at frontal electrodes, C4 (right) and D4 (left), for each emotional condition. Time 0 denotes cue onset. Black boxes correspond to periods and frequencies that differed significantly in the *t*-test. b) Topographic maps of relative frequency bands power (7, 8, 12, 13 Hz) averaged for each emotional condition in a time window between 100 and 400 ms for 7 and 8Hz, between 190 and 290 ms for 12 and 13 Hz after the cue onset. 7 and 8 Hz ERD are globally larger in fear than happiness over the right dorso-ventral areas, whereas over left dorso-ventral 12 and 13 Hz ERS are larger in fearful and happy than neutral faces.

that the emotion expressed by the face affects theta power (4–8 Hz) between 150 and 250 ms over right frontal areas. In yet another study, Güntekin and Basar (2007) reported high posterior alpha (9-13 Hz) and left frontal beta (15-24 Hz) responses for angry compared with happy faces. These authors later investigated lower frequency bands (Güntekin and Basar, 2009) but failed to observe any significant difference across emotional categories in delta (0.5–3.5 Hz) or theta (5-8.5Hz) power. Our current findings confirm the observations that emotional faces give rise to specific modulations of spectral power in the low alpha range and emphasize the role of the right anterior regions in differentiating emotional valence, even in the absence of a functional primary visual cortex and of any visual awareness. It is noteworthy that, in our study, the analysis of every single frequency and the computation of the IAF allowed a greater degree of precision in terms of frequency bands, as we did not rely on boundaries determined by group data but on individual frequency values. With this in mind, our results are in agreement with the literature in terms of the oscillatory frequencies involved (Güntekin and Basar, 2007) as well as the importance of anterior regions (Balconi and Pozzoli, 2009; Balconi and Mazza, 2009; Güntekin and Basar, 2007).

In healthy controls, a decrease of alpha power (desynchronization) is commonly observed over posterior brain regions in tasks involving attention (for a detailed review see Klimesch, 2012). Thalamo-cortical structures, as well as the lateral geniculate nucleus and the pulvinar, are known to affect the modulation of alpha rhythm and play an important role in the synchrony between cortical areas as a function of attentional demands (Saalmann et al., 2012). Alpha power is task and stimulus dependent and reflects information control processing during internally directed (Cooper et al., 2003) as well as externally directed attention (e.g. Klimesch, 1999), with larger responsiveness under difficult perceptual conditions, such as backward-masked shortduration presentations (e.g. Balconi and Mazza, 2009). The lower alpha component has been hypothesized to reflect general task demands (long range interactions) in contrast with the upper alpha/low beta component (local interactions), which is involved in more specific encoding tasks (Klimesch et al., 1997; Pfurtscheller and Lopes da Silva, 1999). More specifically, desynchronization of low alpha is thought to be associated to alertness and vigilance, medium alpha to expectancy (external attention processes), and upper alpha/low beta to enhanced cognitive processing (Klimesch et al., 1998). Thus, it could well be that even in this patient, who lacks any visual awareness, the low alpha desynchronization arises due to an increased state of alertness produced by the unconscious processing of negative (compared to positive) faces, although the mechanism producing low beta desynchronization appears less clear

The anterior location of alpha activity observed in TN may seem at first view surprising. However, the frontal lobes may also be involved in the generation of alpha activity, as demonstrated, for instance, in comatose patients who are reported to present alpha activity produced by alternative generators and pathways than those of healthy controls (Abusleme and Chen, 2009). In addition, a few studies have described alpha power modulation in congenital blindness (Birbaumer, 1970; Noebels et al., 1978), reporting more pronounced changes over frontal than occipital areas in a scalp distribution pattern opposite to healthy subjects (Birbaumer, 1970; Noebels et al., 1978). The frontal lobes also play a crucial role in emotional processes as well as in the resting alpha asymmetry (Davidson, 2004). Magnetoencephalography studies demonstrated the existence of ongoing α -frequency band oscillations in human sensorimotor cortex as well as in frontoparietal regions known to be relevant for consciousness (e.g. Dehaene et al., 2006; Rees et al., 2002). In patient TN, V1 is completely destroyed and, as noted above, previous fMRI evidence has shown that the amygdala nevertheless responds to emotional expressions presented visually. Although the current data cannot provide an answer to the cortical and subcortical generators of the oscillatory activity of this patient, we would hypothesize that the modulations observed over anterior regions must reflect the response of the remaining functional network to emotions that operates without visual awareness.

To conclude, the EEG time frequency analysis yields results that are consistent with a rapid transmission of information (by ~150 ms) as well as left vs. right differences in frontal lobe activations. The findings further suggest that the processing taking place at these frequencies reflects the activation of networks that do not involve consciousness and thus constitute one of the many indicators of affective blindsight.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx. doi.org/10.1016/j.ijpsycho.2013.10.007.

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