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Effects of stimulus location on automatic detection of changes in motion direction in the human brain

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Abstract

We extended the results of a previous report by further exploring the underlying mechanisms of an electrophysiological index of attention-free memory-based detection mechanism to motion-direction changes in the human visual system. By means of presenting bilateral, right- and left-hemifield stimulation in separate conditions, we tried to assess whether the location of the stimuli within the peripheral visual field affected the processing of motion-direction deviations, and to identify brain regions involved in the detection of unattended infrequent changes of motion direction using low-resolution brain electromagnetic tomography (LORETA). Results indicated that the ERP component related to visual change was not affected by stimulus location, and that bilateral temporal and medial regions were activated during this deviance-related response regardless of the hemifield stimulated. The bilateral activation foci observed in this study suggest that brain generators of this deviance-related component could be located at the vicinity of motion processing areas.
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In audition, any change in stimulation, even in the absence of attention, produces a negative event-related potential termed mismatch negativity (MMN) which is maximally distributed over frontocentral regions. MMN has a supratemporal bilateral generator located in the auditory cortex and an additional source located in the right frontal lobe [7,9,10,13], among others.

In the visual system, some recent studies have suggested the existence of a visual ERP component, the visual MMN (vMMN), which could reflect an analogous detection mechanism for changes in colour [6], spatial frequency [14], or spatial position [3]. Recently, we found that infrequent motion-direction changes elicited a negative ERP displacement at the N2 latency range at occipitotemporal sites that showed some of the main MMN characteristics [22]. This response was observed to be independent of processing load or exogenous effects. Moreover, we also proved that this deviance-related component (vMMN) was not related to a differential refractory state of neurons responding to the characteristics of stimuli, but presumably due to a process which compares visual inputs to templates of previous visual sequences.

The study of the topographical characteristics and cerebral generators of MMN has contributed to understand the functional role of this component in auditory sensory processing. Therefore, we aimed this study to extend the results of the previous report and to further explore the underlying mechanisms of this vMMN component to motion-direction changes. By presenting bilateral, right- and left-hemifield stimulation in separate conditions, we tried to assess whether the location of the stimuli within the peripheral visual field affected the processing of motion-direction deviations, and to identify brain regions involved in the detection of unattended infrequent changes of motion direction using low-resolution brain electromagnetic tomography (LORETA).

Twelve healthy subjects (seven females, age 22.50 ± 4.21 years, range 18–32) with normal or corrected-to-normal

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vision participated in the experiment. All were right handed and had no history of neurological disorder. Subjects gave informed consent to participate in this study.

To assess the effects of presentation side on the automatic processing of infrequent changes in the direction of motion, we presented either bilateral, left- or right-hemifield stimulation in separate conditions. Stimuli consisted in sinusoidal gratings differing in the direction of motion placed in the periphery (10.7°) of the visual field. The gratings (20% contrast) had 0.7 c/degree of spatial frequency and subtended 4.13° of visual angle. These stimuli were presented in oddball sequences of repetitive upward (standard or frequent, p = 0.8) and downward-drifting gratings (deviant or infrequent, p = 0.2). Gratings drifted with a speed of 1.95°/s for 133 ms and were followed by a blank screen interstimulus interval of 665 ms. Frequent and infrequent stimuli were presented randomly with the restriction that at least one standard motion direction would occur before each deviant motion direction.

Subjects were requested to ignore the peripheral gratings and to keep their eyes in a small fixation cross placed at the centre of the visual field. Over this fixation point one of nine possible digits (i.e., 1 to 9) was equiprobably presented for 40 ms. Subjects were required to press the left button of a standard mouse with their left hand in response to odd numbers (with the exception of nine), and the right button with their right hand in response to even numbers, as rapidly and accurately as possible. Assignment of response keys and the order of conditions were counterbalanced across subjects. Each experimental block consisted of 770 trials, from which 500 trials corresponded to task-irrelevant gratings and 270 to task-relevant digits. All stimuli were presented with a stimulus onset asynchrony (SOA) of 798 ms.

The electroencephalogram (EEG) was recorded with a NeuroScan system using scalp electrocaps (ECI, Inc.) with electrodes placed at FP1, FPz, FP2, F3, Fz, F4, F7, F8, FCz, C3, Cz, C4, CP3, CPz, CP4, P3, Pz, P4, T3, T4, T5, T6, PO3, POz, PO4, O1, Oz, O2 (10/20 International System). Two extra electrodes were fixed to the scalp, located halfway between O1 and T5 (OL), and O2 and T6 (OR). The active electrodes were referred to the nose-tip and grounded with an electrode at the nasion. Electrical activity elicited to vertical and horizontal eye movements was monitored by EOG recorded bipolarly from above and below the left eye and from the outer canthi of both eyes.

EEG was acquired as continuous signals digitised at a rate of 500 Hz and filtered on-line with an analogue bandpass of 0.05–100 Hz. Trials with eyeblinks, horizontal eye movements, or exceeding ±100 μV were excluded from analyses. For each electrode EEG epochs consisting of 500 ms post-stimulus and 50 ms pre-stimulus were obtained off-line and averaged for peripheral gratings in each subject and condition separately.

The N2 peak amplitudes and latencies relative to baseline were measured individually for each subject and condition from the deviant and standard stimulus ERPs. Difference waveforms (vMMN) were obtained for each subject and condition by subtracting the ERPs elicited to standard from those elicited to deviant stimuli. In the resulting subtraction waveforms, mean amplitude values were calculated separately across six consecutive latency windows within a 100–225 ms latency range. Analyses were restricted to the N2 latency range and occipitotemporal locations. This decision was based on previous results [22] showing that the reliable deviance-related response was located at these time ranges and scalp derivations.

The hemispheric differences in scalp distribution of N2 and vMMN were analysed using repeated-measures ANOVAs with the within-subject factors of condition (bilateral, unilateral right, unilateral left stimulation) and right–left hemisphere (right: O2, OR, T6; left: O1, OL, T6). Separate statistical analyses were run for each component. When appropriate, degrees of freedom were corrected using the Greenhouse–Geisser estimate. Post hoc comparisons were performed using the Bonferroni adjustment for multiple comparisons.

Reaction times (RTs) were on-line recorded for each trial, and hit rates were defined as the percentage of correct responses to target digits with RTs no longer than 798 ms. RTs were analysed for hits only. Hit rates and mean RTs for each participant were compared across conditions using repeated-measures ANOVA.

To locate the possible brain areas activated by unattended motion-direction changes, we analysed the intensity of the current source activities using low-resolution electromagnetic tomography (LORETA) [20,21].

Statistical significance for each pair of deviant-motion versus standard-motion topographic ERP maps was assessed nonparametrically with a randomisation test for each condition separately. The topographic analysis of variance (TANOVA) [23] computes the exact probability of dissimilarity between two maps. To establish the sources responsible for the deviant-related response, LORETA instant images of averaged mismatch potential maps were obtained at the latency defined by the maximum of global field power (GFP). To assess significantly activated areas in relation to baseline statistical non-parametric mapping tests (t-test for single mean value zero) were performed separately for each condition over the LORETA xyz tomographies obtained from averaged subtraction maps.

Differences in visual deviance-related maps among conditions were also analysed with a TANOVA.

Mean hit rates and corresponding RTs of the central visual task are shown in Table 1. Repeated-measures ANOVA revealed no significant differences in the RTs ($F(2, 22) = 0.931$, $p = 0.409$) or hit rates ($F(2, 22) = 0.118$, $p = 0.889$) among conditions.

Peripheral gratings elicited P1, N2 and P2 deflections that were more prominent at posterior sites. Statistical analyses showed that, irrespective of the hemifield stimulated, contralateral N2 peaked earlier than ipsilateral N2, as reflected by a significant "Stimulation × Hemisphere" interaction ($F(2, 22) = 15.060$, $p = 0.001$, $\varepsilon = 0.624$). No differences

Table 1
Behavioural measures: mean RTs and hit rates, standard deviation in parentheses, for each stimulation condition

	Bilateral	Right	Left
Reaction time (ms)	554.14 (51.94)	561.55 (56.93)	549.33 (49.02)
Hits (% correct responses)	87 (11.28)	86.21 (10.88)	87.04 (10.91)

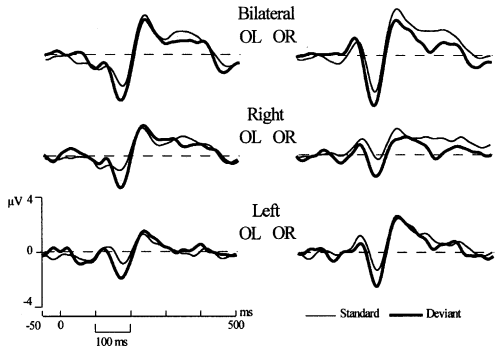

Fig. 1. Group average (N = 12) ERPs to deviant (thick line) and standard (thin line) stimuli during bilateral, right- and left-hemifield stimulation at OL/OR derivations.

between hemispheres in N2 latency were observed during bilateral visual field stimulation ($F(1, 11) = 0.060$, $p = 0.810$). However, N2 amplitude was larger during bilateral stimulation (−3.738 μV) than when unilateral right (−2.006 μV) or left (−2.322 μV) motion stimuli were presented ($F(2, 22) = 8.877$, $p = 0.001$). ANOVA also revealed that the amplitude of this component was larger on the hemisphere contralateral to the hemifield stimulated ($F(2, 22) = 6.896$, $p = 0.005$).

The ERPs elicited to deviants were negatively displaced in relation to ERPs elicited to standards at posterior locations around N2 latency range (see Fig. 1).

Mean amplitudes of the difference waveforms (vMMN) recorded at the right hemisphere were slightly larger than that recorded at the left hemisphere, regardless of the stimulated hemifield.

However, these interhemispheric differences were restricted to the 100–145 ms latency windows (all $p < 0.019$). Neither the main effect of stimulation condition nor the interaction "Stimulation × Hemisphere" were significant across the later latency windows assessed (145–225 ms) (see Fig. 2).

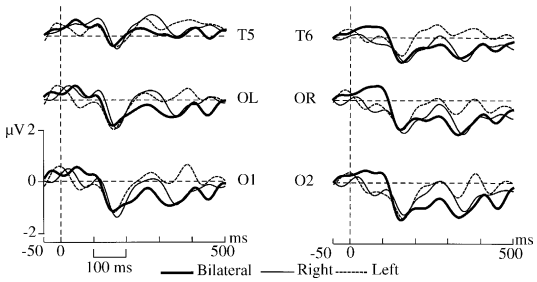

Fig. 2. Difference waveforms (deviant minus standard ERPs) for bilateral, right- and left-hemifield stimulation conditions. The electrodes where they showed largest amplitudes are represented.

In summary, N2 peaked earlier and was of larger amplitude when recorded at the hemisphere contralateral to the visual field stimulated. However, there were no significant amplitude differences across sides of presentation in the mean amplitudes of the vMMN at the latencies where it has shown to be a genuine automatic detection index.

TANOVAs revealed that the time periods where standard-motion and deviant-motion maps showed continuous and consistent differences ranged 158–262 ms for bilateral, 148–238 ms for right, and 124–190 ms for left-hemifield stimulation. These ranges included the GFP peak for the averaged visual difference waveforms (158 ms for bilateral, 168 ms for right, and 166 ms for left-hemifield stimulation).

LORETA sources of averaged mismatch responses at GPF latency ranges located the local maxima for the different conditions at temporal regions in both hemispheres, plus an additional activation in medial areas. Bilateral simultaneous stimulation revealed sources at posterior cingulate (BA 31, 23, 30), right and left superior temporal gyrus (BA 22, and BA 42, 22, respectively), right middle temporal gyrus (BA 37) and right inferior temporal gyrus (BA 20). Right- and left-hemifield stimulation showed similar local LORETA maxima again at bilateral temporal (left BA 42, 22 and right BA 22; BA 37, 20) and medial regions (BA 31, 23, 30). During right-hemifield stimulation an additional activation was also found at BA 31 and 23 corresponding to the posterior cingulate. Detailed results are summarized in Table 2.

Statistical non-parametric mapping tests (t-test for single mean value zero) performed over the LORETA xyz tomographies showed significant activation different from baseline at medial and temporal regions (see Fig. 3). Bilateral stimulation significantly activated the posterior cingulate at medial areas (BA 31, 23, 30) together with a right supratemporal activation (AB 22, 39). The left-hemifield activity located at the supratemporal gyrus almost reached significance. During right-hemifield stimulation significant activity was found at medial areas (posterior cingulate, BA 31, 23, 30; cingulate gyrus, BA 31; precuneus, BA 7) and at the left hemisphere (left superior temporal gyrus, BA 42, 22; postcentral gyrus, BA 40). The right superior temporal gyrus source (BA 39, 22) almost reached significance. Left-hemifield stimulation yielded a significant medial activation (posterior cingulate, BA 31, 23, 30), while areas located at the right (BA 39, 22, supratemporal gyrus) and left (BA 22) hemispheres were near to the limit of the level of significance. These statistical non-parametric mapping tests did not revealed a significant activity at the medial temporal gyrus (BA 37), however p-values were below 0.1 in all conditions.

Table 2
Brain active areas revealed by LORETA sources of averaged mismatch responses at GPF latency ranges. Columns show activation in brain areas in the right- and left-hemisphere, and in medial areas. Bold numbers denote Brodmann areas. Numbers in parentheses represent the Talairach coordinates (in mm) for *x*-, *y*-, and *z*-axis

Stimulation condition	Left-hemisphere activation	Right-hemisphere activation	Medial areas activation
Bilateral	**42**, **22** (−59, −32, 8)	**22** (60, −39, 15); **37**, **20** (53, −53, −13)	**31**, **23**, **30** (4, −67, 15)
Right	**42**, **22** (−59, −32, 8)	**22** (60, −39, 15); **37**, **20** (53, −53, −13)	**31**, **23**, **30** (4, −67, 15); **31** (−3, −46, 43); **31**, **23** (4, −60, 22)
Left	**42**, **22** (−59, −32, 8)	**22** (60, −39, 15); **37**, **20** (53, −53, −13)	**31**, **23**, **30** (4, −67, 15)

Finally, TANOVA analyses comparing deviance-related maps elicited during bilateral and hemifield stimulation conditions did not reveal significant differences among them.

The analysis of amplitudes and latencies of the N2 component elicited by motion gratings showed effects of spatial presentation of stimuli. N2 peaked earlier and was of larger amplitude when recorded over the hemisphere contralateral to the stimulated hemifield. These effects are in agreement with pattern and motion VEP studies, where amplitude and latency differences between hemispheres were found [11]. However, the hemispheric differences are less pronounced for motion stimuli, since extrastriate motion areas in the dominant hemisphere receive motion stimulation inputs from both the ipsilateral and contralateral visual fields [26].

All peripheral gratings elicited this P1–N2–P2 complex, but deviants (infrequent motion direction) elicited a more negative N2 component especially at occipitotemporal locations.

The N2 component of the motion-onset VEPs has been identified as a motion-related component which also matches motion perception in its susceptibility to adaptation [2]. The adaptation effects are to a larger extent global, since the amplitude reduction of N2 is non-direction specific, however when a stimulus shares the same direction with the adapting stimulus the amplitude of N2 can be reduced up to 28% more [16]. These reductions are also present even with very low duty cycles (i.e., the relation of motion to total presentation time) such as those used in the present study [15]. Therefore, one could argue that the shortened amplitude of N2 elicited to standards could be due to the direction-specific additional reduction of this component elicited by frequent directions of motion. However, in a previous study [22] we provided evidence precluding that the effects could be due to a differential sensory adaptation. In that study we presented an additional control block which consisted in the presentation of equiprobably gratings ($p = 0.2$) drifting in five different directions. We found that the control stimuli that shared the same low probability and physical features with deviant stimuli elicited an N2 component with similar amplitude to that elicited by standard stimuli. That is, the responses elicited to oddball-motion

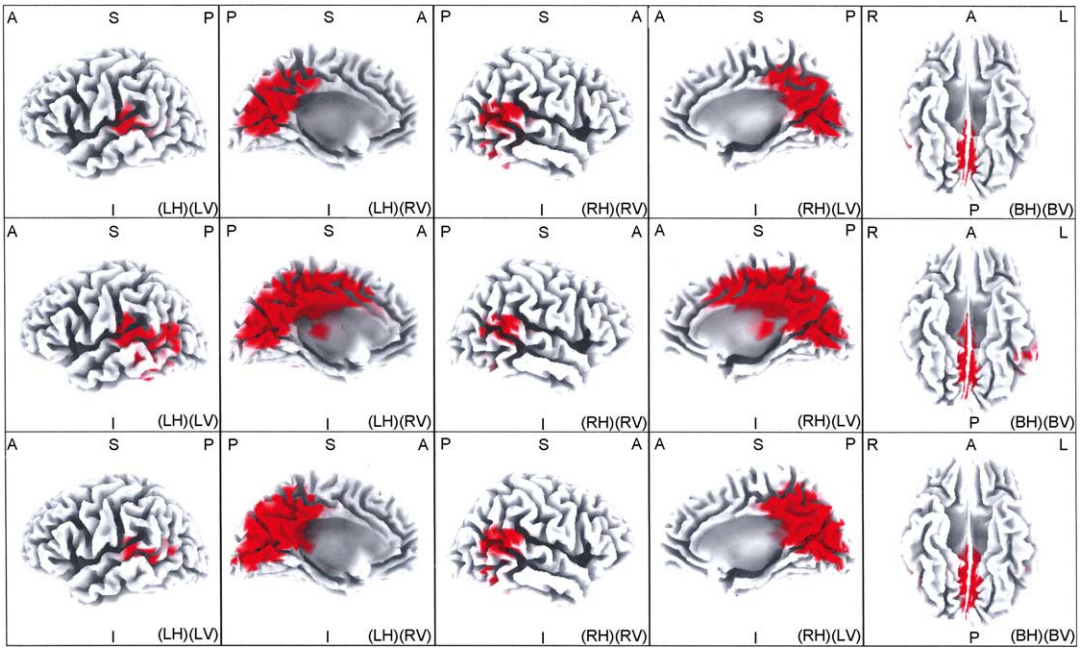

Fig. 3. LORETA-based statistical nonparametric maps (SnPM) derived from averaged mismatch LORETA *xyz* tomographies showing activation areas at GFP latencies for each stimulation condition. First row: 158 ms post-stimulus latency, bilateral stimulation. Second row: 168 ms right-hemifield stimulation. Third row: 166 ms left-hemifield stimulation. A, anterior; P, posterior; S, superior; I, inferior; LH, left hemisphere; RH, right hemisphere; BH, both hemispheres; LV, left view; RV, right view; BV, bottom view.

deviants were significantly more negative than both oddball-standards and controls. This result suggests that the larger negative displacement observed in deviants was not related to a differential sensory adaptation between frequent and infrequent directions of motion but to a mechanism dependent on a deviance from the stimulus context.

It could also be claimed that the enhanced negativity in response to deviants was due to an exogenous effect of the stimulus change. However, we also observed that reversing the stimuli that acted as infrequent and frequent directions of motion did not affect the vMMN. Therefore, this negative deviant-related response was not related to the direction of motion per se, but to a change in the direction of motion.

In the present study, the occipitotemporal distribution of the recorded motion vMMN component is in agreement with other visual studies that have reported deviant-related components distributed over posterior scalp locations consistent with a specific modality distribution [3,6,14]. Nevertheless, other studies have also reported additional positive components located in frontal regions [14].

We also observed that the deviance-related negativity (vMMN) did not show significant contralateralization or hemifield dominance at the latency ranges where it has been proved to be a genuine deviant-related process [22]. The lack of significant differences across sides of presentation was confirmed by additional topographic analyses of variance. Our results are in line with brain imaging studies that have shown that visual motion stimulation induces bilateral foci of activation in area V5-MT, since motion areas receive input from both ipsilateral and contralateral visual field [26]. In humans, it has been observed that V5-MT map invades the ipsilateral visual field up to, at least 10°–14° [25,26]. We presented the stimuli with an eccentricity of 10.7°, which could led us to conclude that the lack of contralateralization effects on vMMN are due to the used eccentricity. However, the contralateral effects observed on N2 suggest that the hemifield stimulation was effective with the employed eccentricity values.

Stimulus location effects on auditory MMN have been previously assessed. It has been observed that monaural versus binaural stimulation does not have effects on auditory MMN to changes in frequency or duration [18]. However, when right and left lateralized stimuli were used, mismatch dipole amplitudes to changes in source direction of complex non-language stimuli have been reported to be larger in the contralateral than the ipsilateral hemisphere. Moreover, a facilitated detection of auditory spatial deviances could occur in the right hemifield [17].

In the visual modality the effects of stimulus location on vMMN have been less explored. Recently, Czigler et al. [5] investigated the effects of upper and lower hemifield stimulation on vMMN to changes in colour. They found that infrequent coloured patterns elicited a posterior negative event-related potential component only in case of lower half-field stimulation. The authors considered this finding as indirect evidence that vMMN originated in retinotopically organized parts of the visual system such as the prestriate cortex. In contrast with the Czigler et al.'s [5] study, we found that the location of stimuli within the peripheral visual field did not affect the visual change related response. The lack of stimulus-location effects could be explained by the differential upper–lower versus left–right-hemifield stimulation used in both studies. Nevertheless, we could also consider this result as an indication that visual motion change detection sources are located in less retinotopically organized areas than those reported for colour vMMN.

Current density tomographies calculated over GPF latencies of subtraction maps showed bilateral temporal activations (supratemporal gyrus, BA 42, 22; medial temporal gyrus, BA 37) together with a medial region identified as the posterior cingulate (BA 31, 23, 30) for all bilateral, right- and left-hemifield conditions. Statistical non-parametric tests performed over the LORETA *xyz* tomographies suggest that the areas related to deviance more significantly activated are located in the posterior cingulate and the supratemporal gyrus of the temporal lobe.

The posterior active areas observed in the present study are consistent with results previously shown in motion neuroimaging studies. Most of them reported activation in middle temporal complex (hMT/V5+), human visual area V3a or superior temporal sulcus (STS). Visual hMT/V5+ area has been identified in fMRI and PET studies at the limits of Brodmann's areas 19 and 37 [1,26,27]. LORETA solutions calculated over sources of averaged mismatch responses are within the limits of hMT/V5+ and satellite areas.

One of the most active regions was located in the vicinity of the supratemporal gyrus (BA 22, 42). This area is close to other motion-related areas, such as the superior temporal sulcus [24], and the posterior insular cortical region [8]. An additional evidence for the role of the superior temporal region in motion processing comes from lesion studies that have observed that damage in this area leads to an impairment in direction and speed motion discrimination [12]. However, this visual feature is not crucial for its activation. The supratemporal region is considered as a polysensory area that also responds to other sensory modalities (i.e. audition) [4].

An additional medial source, the posterior cingulate, was active through all conditions. This area, which has been identified as a motion responsive region [24], has also been related to other processes, such as spatial attention [19].

In summary, we obtained a negative ERP component to changes in motion direction that was not significantly affected by the hemifield stimulated. LORETA sources of the deviance-related response were found at bilateral temporal and medial locations. The bilateral activation foci observed in this study could be related to brain sources located at modality specific areas for motion processing. Future research should confirm the neural sources of this mechanism by combining ERP recordings with more precise localization techniques such as MEG or fMRI.

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References

[1] S.J. Anderson, I.E. Holliday, K.D. Singh, G.F.A. Harding, Localization and functional analysis of human cortical area V5 using magnetoencephalography, Proc. R. Soc. Lond. [Biol.] 263 (1996) 423–431.
[2] M. Bach, D. Ullrich, Motion adaptation governs the shape of motion-evoked cortical potentials, Vision Res. 34 (1994) 1541–1547.
[3] S. Berti, E. Schröger, Distraction effects in vision: behavioral and event-related potential indices, Neuroreport 15 (2004) 665–669.
[4] R. Blake, R. Sekuler, E. Grossman, Motion processing in human visual cortex, in: J.H. Kaas, C. Collins (Eds.), Primate Visual System, CRC Press, Boca Raton, FL, 2003.
[5] I. Czigler, L. Balázs, L.G. Pató, Visual change detection: event-related potentials are dependent on stimulus location in humans, Neurosci. Lett. 364 (2004) 149–153.
[6] I. Czigler, L. Balázs, I. Winkler, Memory-based detection of task-irrelevant visual changes, Psychophysiology 39 (2002) 869–873.
[7] L.Y. Deouell, S. Bentin, M.H. Giard, Mismatch negativity in dichotic listening: evidence for interhemispheric differences and multiple generators, Psychophysiology 35 (1998) 355–365.
[8] P. Dupont, G.A. Orban, B. De Bruyn, A. Verbruggen, L. Mortelmans, Many areas in the human brain respond to visual motion, J. Neurophysiol. 72 (1994) 1420–1424.
[9] M.H. Giard, J. Lavikainen, K. Reinikainen, F. Perrin, O. Bertrand, M. Thevenet, J. Pernier, R. Näätänen, Separate representation of stimulus frequency, intensity, and duration in auditory sensory memory, J. Cogn. Neurosci. 7 (1995) 133–143.
[10] M.H. Giard, F. Perrin, J. Pernier, P. Bouchet, Brain generators implicated in the processing of auditory stimulus deviance: a topographic event-related potential study, Psychophysiology 27 (1990) 627–640.
[11] E. Gopfert, R. Muller, D. Breuer, M.W. Greenlee, Similarities and dissimilarities between pattern VEPs and motion VEPs, Doc. Ophthalmol. 97 (1999) 67–79.
[12] M.W. Greenlee, A.T. Smith, Detection and discrimination of first- and second-order motion in patients with unilateral brain damage, J. Neurosci. 17 (1997) 804–818.
[13] R. Hari, M. Hamalainen, R. Ilmoniemi, E. Kaukoranta, K. Reinikainen, J. Salminen, K. Alho, R. Näätänen, M. Sams, Responses of the primary auditory cortex to pitch changes in a sequence of tone pips: neuromagnetic recordings in man, Neurosci. Lett. 50 (1984) 127–132.
[14] D. Heslenfeld, Visual mismatch negativity, in: J. Polich (Ed.), Detection of Change: Event-related Potential and fMRI findings, Kluwer, Doordrecht, 2003, pp. 41–59.
[15] M. Hoffmann, T.J. Dorn, M. Bach, Time course of motion adaptation: motion-onset visual evoked potentials and subjective estimates, Vision Res. 39 (1999) 437–444.
[16] M. Hoffmann, A.S. Unsöld, M. Bach, Directional tuning of human motion adaptation as reflected by the motion VEP, Vision Res. 41 (2001) 2187–2194.
[17] J. Kaiser, W. Lutzenberger, N. Birbaumer, Simultaneous bilateral mismatch response to right- but not leftward sound lateralization, Neuroreport 11 (2000) 2889–2892.
[18] J. Lavikainen, H. Tiitinen, P. May, R. Näätänen, Binaural interaction in the human brain can be non-invasively accessed with long-latency event-related potentials, Neurosci. Lett. 222 (1997) 37–40.
[19] M.M. Mesulam, A.C. Nobre, Y.H. Kim, T.B. Parrish, D.R. Gitelman, Heterogeneity of cingulate contributions to spatial attention, Neuroimage 13 (2001) 1065–1072.
[20] R.D. Pascual-Marqui, Review of methods for solving the EEG inverse problem, Int. J. Bioelectromagnetism 1 (1999) 75–86.
[21] R.D. Pascual-Marqui, C.M. Michel, D. Lehmann, Low resolution electromagnetic tomography: a new method for localizing electrical activity in the brain, Int. J. Psychophysiol. 18 (1994) 49–65.
[22] P. Pazo-Álvarez, E. Amenedo, F. Cadaveira, Automatic detection of motion direction changes in the human brain, Eur. J. Neurosci. 19 (2004) 1978–1986.
[23] W.K. Strik, A.J. Fallgatter, D. Brandeis, R.D. Pascual-Marqui, Three dimensional tomography of event-related potentials during response inhibition: evidence for phasic frontal lobe activation, Electroencephalogr. Clin. Neurophysiol. 108 (1998) 406–413.
[24] S. Sunaert, P. Van Hecke, G. Marchal, G.A. Orban, Motion-responsive regions of the human brain, Exp. Brain Res. 127 (1999) 355–370.
[25] R.B. Tootell, J.D. Mendola, N.K. Hadjikhani, A.K. Liu, A.M. Dale, The representation of the ipsilateral visual field in human cerebral cortex, Proc. Natl. Acad. Sci. U.S.A. 95 (1998) 818–824.
[26] R.B. Tootell, J.B. Reppas, K.K. Kwong, R. Malach, R.T. Born, T.J. Brady, B.R. Rosen, J.W. Belliveau, Functional analysis of human MT and related visual cortical areas using magnetic resonance imaging, J. Neurosci. 15 (1995) 3215–3230.
[27] J.D. Watson, R. Myers, R.S. Frackowiak, J.V. Hajnal, R.P. Woods, J.C. Mazziotta, S. Shipp, S. Zeki, Area V5 of the human brain: evidence from a combined study using positron emission tomography and magnetic resonance imaging, Cereb. Cortex 3 (1993) 79–94.