PERGAMON

Vision Research 40 (2000) 2379–2385

Vision Research

www.elsevier.com/locate/visres

Visual motion detection in man is governed by non-retinal mechanisms

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Received 1 June 1999; received in revised form 30 September 1999

Abstract

It is generally assumed that there is no sizable proportion of motion detectors in the primate retina. To test this specifically for humans, visual evoked potentials (VEPs) and electroretinograms (ERGs) were recorded simultaneously to visual motion onset (9.3°/s) of an expanding or contracting 'dartboard'. The degree of motion-specific responses in cortex and retina was assessed by testing the direction specificity of motion adaptation with three conditions in a fully balanced paradigm: motion-onset potentials were measured after adaptation to: (1) a stationary pattern; (2) motion in the same direction as the test stimulus; and (3) motion in the opposite direction. Motion-onset responses in the VEP were dominated by the typical N2 at 150 ms, in the ERG by a positivity at 70 ms. Onset of contraction or expansion evoked virtually identical VEP and ERG responses ($P > 0.5$). Motion adaptation produced strong direction-specific effects in the VEP ($P < 0.05$), but not in the ERG ($P = 0.58$): In the adapting and non-adapting direction the VEP (N2) was reduced by 75 and 50% ($P < 0.001$), the ERG by 32 and 26% ($P < 0.01$ and 0.05), respectively. The striking difference of the direction-specificity of motion adaptation between cortex and retina suggests that in humans the vast majority of motion-specific processing occurs beyond the retinal ganglion cells.

Keywords: Motion; Adaptation; Human; Retina; Cortex; Electroretinogram; Visual evoked potentials

1. Introduction

Motion detection is an elementary aspect of visual perception (Nakayama, 1985) and in many vertebrates it is already accomplished at the retinal level (reviewed by Grüsser & Grüsser-Cornehls, 1973; Amthor & Grzywacz, 1993; e.g. frog: Maturana, Lettvin, McCullach & Pitts, 1960; turtle: Jensen & Devoe, 1983; birds: Maturana & Frenk, 1963; rabbit: Barlow & Hill, 1963a,b; Oyster, 1968; squirrel: Michael, 1968). In higher animals the proportion of direction selective units in the retina is diminished: for cats, Stone and Fabian (1966) report 1 out of 50 cells; for macaques, we did not find a single source. While Amthor and Grzywacz (1993) hint on evidence in Schiller and Malpeli (1977) and De Monasterio (1978), in the original reports no direction selective units are reported. In the macaque monkey a considerable degree of direction selectivity is found in cortical areas such as V1 (layer 4B), V2, V3, and V5 (reviewed by DeYoe & Van Essen, 1988). In accordance with other similarities of the human and the macaque monkey visual system (e.g. Zeki, Watson, Lück, Friston, Kennard & Frackowiak, 1991; Sereno, Dale, Reppas, Kwong, Belliveau, Brady et al., 1995; Tootell & Taylor, 1995; DeYoe, Carman, Bandettini, Glickman, Wieser, Cox et al., 1996; Tootell, Dale, Sereno & Malach, 1996; Engel, Glover & Wandell, 1997), there should also in the human retina only be a low, if any, degree of motion specific processing. This has not been tested so far.

Electroretinograms (ERGs) and visual evoked potentials (VEPs) are tools for the noninvasive electrophysiological investigation of the human visual

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0042-6989/00/$ - see front matter
PII: S0042-6989(00)00106-1

system. The ERG to pattern stimulation with no net luminance change can be attributed to the activity of retinal ganglion cells (Groneberg & Teping, 1980; Maffei & Fiorentini, 1981; Baker, Hess, Olsen & Zrenner, 1988; Zrenner, 1989; Bach, Gerling & Geiger, 1992), whereas the VEP is due to the activity of cortical neurons. While there are only few reports on ERG-recordings in humans applying motion stimuli (e.g. Korth, 1986; Dodt & Kuba, 1995; Korth, Rix & Sembritzki, 1997), cortical mechanisms of motion detection in humans have repeatedly been investigated with the motion VEP (e.g. MacKay & Rietveld, 1968; Clarke 1972, 1973a,b, 1974; Tyler & Kaitz, 1977; Andreassi & Juszczak, 1982; Göpfert, Müller, Markwardt & Schlykowa, 1983; Kubovà, Kuba, Hubàcek & Vit, 1990; Bach & Ullrich, 1994; Snowden, Ullrich & Bach, 1995). At occipital and occipito-temporal electrodes visual motion onset evokes a potential which is dominated by a positivity, P1 around 100–130 ms, and a negativity, N2 around 150–200 ms (reviewed by Niedeggen & Wist, 1998). By its velocity and contrast dependence N2 was identified as a motion related component, whereas P1 is more likely to be associated with pattern processing (Markwardt, Göpfert & Müller, 1988; Müller & Göpfert, 1988; Kubovà et al., 1990; Schlykowa, van Dijk & Ehrenstein, 1993; Kubovà, Kuba, Spekreijse & Blakemore, 1995; Bach & Ullrich, 1997). Additionally, N2 is very susceptible to motion adaptation (Göpfert et al., 1983; Göpfert, Müller & Hartwig, 1984; Müller, Göpfert & Hartwig, 1985; Schlykowa et al., 1993; Bach & Ullrich, 1994; Wist, Gross & Niedeggen, 1994) and matches human motion perception in its time-course of motion adaptation and recovery (Hoffmann, Dorn & Bach, 1999). Source analysis showed that N2 originates in or near area MT (Probst, Plendel, Paulus, Wist & Scherg, 1993). With direction-specific adaptation it could be shown that at least 30% of the N2 amplitude reflects activity of direction specific units (Bach, Hoffmann & Unsöld, 1999). Hence N2 can be regarded as a component that reflects motion specific cortical neuronal activity to a substantial degree.

Here we simultaneously recorded ERGs and VEPs to assess the degree of motion detection in the retina. In Experiment 1 we determined the 'optimal' stimulus velocity for our experiments in a combined psychophysical and electrophysiological approach. For the psychophysical measurements we used the illusory motion perceived after prolonged viewing of visual motion, the motion aftereffect (MAE; reviewed by Wade, 1994). In Experiment 2 we used the direction specificity of motion adaptation to test whether cortical motion adaptation might be traced back to the adaptation of retinal motion selective mechanisms. A preliminary account of this work has been presented by Hoffmann and Bach (1999).

2. Methods common to both experiments

2.1. Subjects

VEPs and ERGs were simultaneously recorded from six (Experiment 1) or ten (Experiment 2) human observers with normal or corrected to normal visual acuity (≥ 1.0). In Experiment 1 all, in Experiment 2 nine subjects were naive as to the experimental question and gave their written informed consent to participate in the experiment.

2.2. Stimuli

Pattern reversal potentials were evoked with a phase-reversing checkerboard pattern (2 rev/s, 0.8° check size, 98% contrast, 55 cd/m^2 luminance). Motion onset was generated with the abrupt onset of contraction or expansion of a 'dartboard'-pattern (34° diameter, 16 sectors, 15 elements (alternating black and white) per sector; 55 cd/m^2 mean lumimance; 98% contrast). The contraction/expansion type of movement avoids optokinetically induced eye movements, though small vergence eye movements to radial flow patterns cannot be excluded (Busettini, Masson & Miles, 1997).

A central disc (2°) with a centered cross served as fixation target. Stimuli were generated by a computer (Power Macintosh G3; Bach, 1999) and presented on a CRT with a frame rate of 75 Hz at a viewing distance of 57 cm. The pattern was surrounded by a circular gray mask (size: $44 \times 34°$; mean luminance 45 cd/m^2). Subjects stabilized their heads with a chinrest.

2.3. ERG and VEP recordings

We recorded ERGs binocularly with DTL electrodes (Dawson, Trick & Litzkow, 1979; Bach, 1998) referenced to the ipsilateral canthi and VEPs from three derivations, O_z, O_{tr}, and O_{tl} (5 cm right and left from O_z, respectively) referenced to linked ears. The ground electrode was attached to the right wrist. Signals were amplified, filtered (0.3–70 Hz, Toennies physiological amplifier), and digitized at a sampling rate of 1 kHz.

2.4. Data analysis (ERG and VEP)

Trials were analyzed off-line over the interval from 200 ms before to 500 ms after motion onset. Trials with blinks, detected with a threshold criterion of 100 μV, were discarded (number of sweeps left for analysis per stimulus and observer [median; range] — Experiment 1: 163; 58–209; Experiment 2: 153; 84–197). Averaged sweeps were digitally filtered (VEP: 0–40 Hz; ERG: 2–40 Hz). Baseline was defined as the mean value from 100 ms before to 30 ms after stimulus onset of the averaged trace and used as zero reference for peak measurements.

ERGs from both eyes of a subject were averaged. For VEPs the O_t^* derivation, covering recordings from the O_{tr} or O_{tl} derivation of a subject, was introduced for the following rationale: motion-onset potentials are often strongly lateralized, i.e. some subjects have more pronounced potentials at O_{tr}, others at O_{tl} (Andreassi & Juszczak, 1982). To maximize the signal to noise ratio for N2 amplitudes we evaluated the dominant O_t derivation, i.e. the one with maximal N2 amplitudes, of each subject for the grand mean of O_t^*. The dominant O_t derivation was the one with maximal N2 amplitudes as determined in Experiment 1 from the mean of the normalized N2-amplitudes to the five stimuli used and in Experiment 2 from the mean response to contraction and expansion during the baseline condition in Experiment 2. The degree of lateralization varied among subjects. N2 was maximal at the left derivation in three out of six subjects in Experiment 1 and in seven out of ten in Experiment 2.

For statistics and parametric plots of the results from Experiment 2 we normalized the ERG and VEP data with respect to each subject's baseline amplitude (mean of response to contraction and expansion) of P70 or N2, respectively, to minimize multiplicative intersubject variability.

2.5. Statistics

Statistical significance of experimental effects on the normalized peak amplitudes were evaluated with an ANOVA and tested post-hoc with Fisher's protected LSD test. Significance levels are indicated in the figures (* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$).

2.6. Specifics of Experiment 1: motion-onset VEP and ERG to different stimulus velocities

2.6.1. Stimuli

Motion onset was generated by the onset of contraction of a stationary 'dartboard' pattern. For VEPs and ERGs the following stimulus sequence was repeated: 2700 ms stationary, 300 ms test stimulus (motion or pattern reversal). To elicit a stronger MAE the following stimulus sequence was shown for the psychophysical rating procedure: 5400 ms stationary, 600 ms test stimulus. The short motion-adaptation epoch of 600 ms evoked a small, but sufficient MAE for ranking (see below). Four different velocities (4.7, 9.3, 22.2, and 36.2°/s) and pattern reversal onset (75 rps) were tested.

2.6.2. Procedure

A psychophysical ranking experiment was performed to determine the strength of the MAE from the different velocities. The five stimuli were randomly assigned to the digits 1–5. Subjects arbitrarily selected by keypress any of the five stimuli for display in order to judge the strength of the illusory expansion of the stationary pattern after each motion stimulus. Their task was to sort the five stimuli according to the strength of the MAE. They could select the stimuli as often as they wanted. Three rating measurements per subject were taken and the median for each subject was determined. This procedure took about 15 min.

For ERG and VEP recordings ten blocks of 100 randomly ordered stimuli were presented separated by short breaks. One session lasted about 2.5 h.

2.7. Specifics of Experiment 2: direction specific motion-adaptation

2.7.1. Stimuli

As stimuli we applied checkerboard-pattern reversal (2 rps) and motion-onset of a 'dartboard'-pattern. Motion onset was generated by the onset of contraction or expansion of the stationary dartboard-pattern at a velocity of 9.3°/s. Three conditions were tested: motion-onset potentials after adaptation to: (1) a stationary pattern ('baseline'); (2) motion in the same direction as the test stimulus ('same'); and (3) motion in the opposite direction ('opposite'). All three conditions were tested with both expanding and contracting dartboards. The following stimulus sequence was repeated: 300 ms motion, 2200 ms adaptation, 500 ms stationary pattern.

2.7.2. Procedure

Stimuli were presented in a fully balanced interleaved block design. Blocks of 100 stimuli were presented, each block containing test stimuli for contraction and expansion in random order. The following block sequence was applied: 2 × baseline, 2 × adaptation with contraction, 4 × adaptation with expansion, 2 × adaptation with contraction, 2 × baseline. After each change of adaptation direction and before baseline a 5 min break was introduced to reduce crosstalk of adaptation. One session lasted about 3 h.

3. Results

3.1. Experiment 1

ERG-responses to onset of motion or pattern reversal consisted of a positivity between 43 and 66 ms followed by a negative excursion (Fig. 1, left). They were most pronounced for the higher stimulus velocities and for pattern reversal onset. VEP responses were dominated by N2 around 160 ms (Fig. 1, middle) and were less influenced by stimulus velocity. Pattern reversal onset evoked the most pronounced N2. Perceptually direction specific adaptation was strongest for a stimulus velocity of 22.2°/s as determined with MAE rating (Fig. 1, right). Since the velocities 9°/s and 22°/s evoked

strong MAE, we chose the former, being closer to previous motion-VEP work, as adaptation velocity in Experiment 2.

3.2. Experiment 2

Motion-onset responses in the ERG consisted of a pattern-ERG like positivity at 70 ms followed by a negative excursion at 150 ms (Fig. 2a and c), in the VEP they were dominated by N2 at 160 ms (Fig. 2a). There was no significant difference in the response to the expanding versus contracting 'dartboard' (ERG: $P = 0.78$; VEP: $P = 0.56$). Motion adaptation produced strong effects in the VEP (Fig. 2a and b): N2 was reduced by 75% ($P < 0.001$) when testing in the adapting direction ('same') and by 50% ($P < 0.001$) in the non-adapting direction ('opposite'). The effect was specific for direction ($P < 0.05$). Smaller, not direction specific effects ($P = 0.58$) of motion adaptation were found in the motion ERG: P70 was reduced by 32% ($P < 0.01$) in the adapting direction and by 26% ($P < 0.05$) in the non-adapting direction.

4. Discussion

Distinct ERGs and VEPs were evoked by both motion onset and onset of pattern reversal in all subjects. Adaptation to motion markedly reduced both ERG (P70) and VEP (N2) amplitudes. In the VEP motion adaptation was specific for direction, confirming previous results (Bach et al., 1999), in the ERG it was not specific for direction. The striking difference of the direction-specificity of motion adaptation between cortical and retinal signals suggests that in humans the vast majority of motion detection occurs beyond the retinal ganglion cell level. Consequently the ERG evoked by motion onset (Korth, 1986; Dodt & Kuba, 1995; Korth et al., 1997; present study) does not represent motion-specific, i.e. direction specific, mechanisms.

Let us consider possible error sources which might affect our conclusions:

1. It is the adaptability of the motion detectors that forms the bases of our paradigm; but should retinal motion detectors be expected to adapt to motion? In species with retinal motion detectors motion adaptation has been demonstrated (Barlow & Hill, 1963b). Hence, if there were a substantial proportion of motion detectors in the human retina, we would expect them to be detected with the adaptation paradigm.
2. The present experiment does not exclude the possibility that there exists at least a small proportion of motion detectors in the human retina. However, the direction specific difference in the ERG-amplitude reduction is small (6%) and far from reaching significance with ten subjects. It would be intriguing to search for signals of a small proportion of retinal

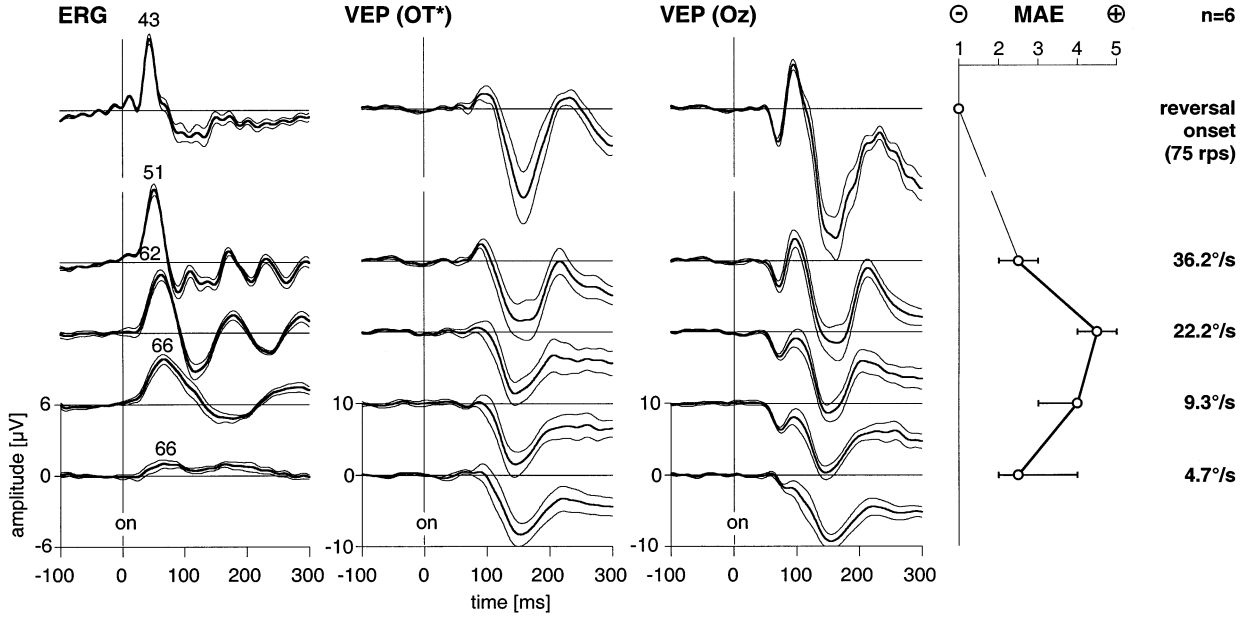

Fig. 1. Velocity dependence of ERG, VEP, and MAE. From left to right: ERG, two VEP derivations (grand mean ± SEM) and subjective rating of MAE (median ± quartile) to onset of motion with different velocities and to onset of pattern reversal. Velocity increases from bottom upwards, but the topmost condition was 'onset of reversal' (see numbers at right). Motion onset and rapid pattern reversal onset evoke an ERG with a positivity between 40 and 70 ms. Latencies are indicated next to the peaks. Motion onset and rapid pattern reversal onset evoke a VEP with N2. At Oz, there is an additional positivity (P1) for high velocities and pattern reversal. Motion aftereffect is strong after motion with 9–22°/s and weakest (or non-existent) after rapid pattern reversal. The stimulus velocity of 22.2°/s was selected for Experiment 2.

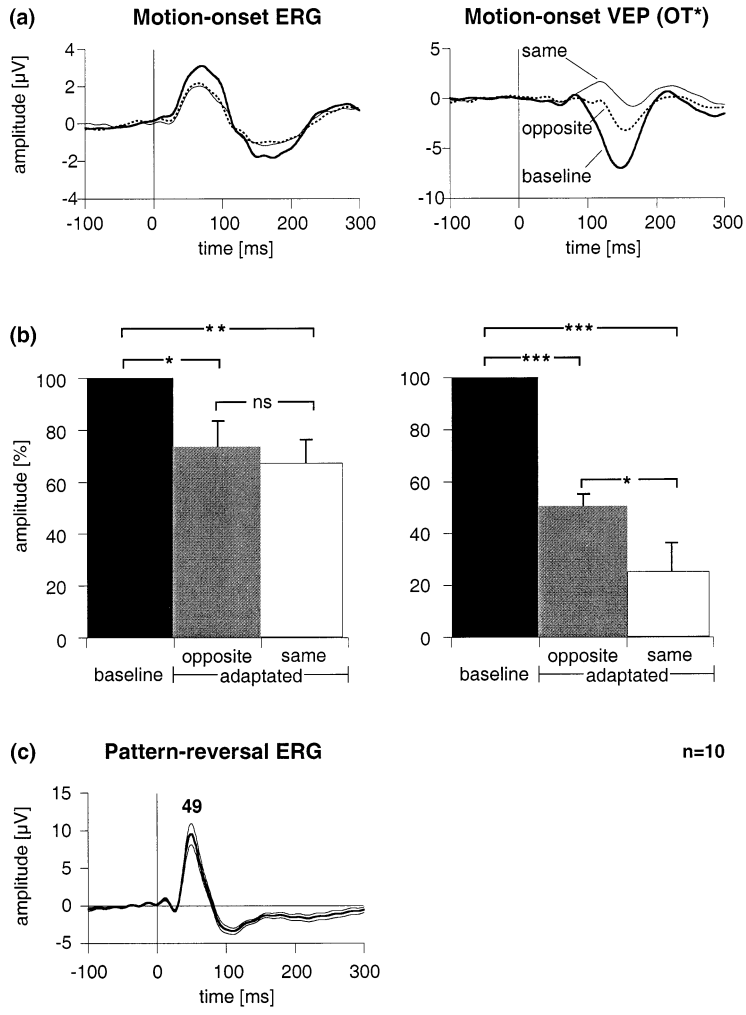

Fig. 2. Effect of test- and adaptation-direction on the amplitude of motion-onset-ERG and – VEP. Responses to contracting and expanding test stimuli are averaged. The test stimulus was either preceded by adaptation to a stationary pattern ('baseline'), adaptation to a motion stimulus of opposite direction ('opposite'), or adaptation to a motion stimulus of the same direction as the test stimulus ('same'). (a) Grand mean traces of ERG and VEP (SEMs omitted for clarity) (b) Average based on the evaluation of the individual peaks of each subject (normalized to baseline) ± SEM. Significance levels are indicated. The ERG-P70 is reduced after motion adaptation in a non direction-specific manner. The VEP-N2 is reduced after motion adaptation in a direction-specific manner. (c) Pattern reversal ERG (grand mean ± SEM) for comparison. P50 amplitude is almost three times the motion onset P70 amplitude.

motion detectors with low-contrast stimuli, since the signal of any motion system might be relatively enhanced using reduced stimulus contrast (Kaplan & Shapley, 1986). For such an experiment, however, it must be taken into consideration that lowered stimulus contrast strongly reduces ERG responses (Thompson & Drasdo, 1989; Zapf & Bach, 1999) thus deteriorating signal to noise ratio.

3. We tested only at a stimulus velocity of 9.3°/s. It is not a matter of course that the results can be generalized to all velocities. It has been shown that stimulus velocity can be of crucial importance in motion adaptation. For example, Verstraten, van der Smagt and van de Grind (1998) and Verstraten, van der Smagt, Fredericksen and van de Grind (1999) recently showed that the occurrence of MAE on static and dynamic test patterns heavily depends on the velocity of the adaptation stimulus. Still, we see no compelling reasons to expect qualitatively different results at different stimulus velocities.

How can we understand the non-direction specific adaptation of motion-onset ERG and VEP? This global adaptation effect is considerable in both ERG and VEP (30 and 50%, respectively). It appears plausible that it results from elements that adapt to temporal local luminance changes (Bach et al., 1999). Furthermore, the presented data strongly suggest that the global adaptation observed in the motion-onset VEP is to a high degree caused by retinal adaptation, whereas the direction specific adaptation is not caused by retinal adaptation.

Finally, why does the contribution of the retina to motion detection diminish when we ascend the phylogenetic ladder? As a basis for speculation, it is known that attention plays an important role in motion perception in higher animals and man (e.g. Corbetta, Miezin, Dobmeyer, Shulman & Petersen, 1990; Treue & Maunsell, 1996; Beauchamp, Cox & DeYoe, 1997; von Grünau, Bertone & Pakneshan, 1998). Modulatory influences cannot apply in the primate retina, as there is no appreciable efferent pathway (reviewed by Uchiyama, 1989). The two concepts, that motion processing needs to be modulated, and that there is no significant efferent motion pathway to the retina in higher animals, would push motion processing to a higher level than the retina. The question then remains why retinopetal projections diminish as the phylogenetic ladder is ascended (Uchiyama, 1989).

Here we present evidence that there is no substantial amount of motion detection in the human retina. This is in accordance with the common view of motion processing in humans and primates.

Acknowledgements

This work was supported by the Deutsche Forschungsgemeinschaft.

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