Increased Role of Peripheral Vision in Self-Induced Motion in Patients with Age-Related Macular Degeneration

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PURPOSE. The contribution of peripheral vision in inducing self-motion (vection) was investigated in people with bilateral age-related macular degeneration (AMD).

METHODS. Eleven patients with bilateral AMD and dense central scotomas with no islands of functional central retina and 12 age-matched control subjects were exposed to random-dot patterns projected on a large screen. The dots either moved from left to right, inducing linear vection, or rotated about the roll axis, inducing roll vection. Latency, total vection time, and objective and subjective measures of tilt were recorded.

RESULTS. The patients with AMD experienced shorter latencies than did the age-matched control participants, but the total vection time in both conditions and tilt during roll vection were the same in both groups. There was a positive correlation between objective tilt and subjective measures of tilt in the AMD, but not in the age-matched control group. There was a negative relationship between absolute scotoma size and latency.

CONCLUSIONS. Two main conclusions were drawn. First, the role of peripheral vision in inducing vection is enhanced in people with bilateral central vision loss. Second, people with bilateral AMD adapt successfully to a moving environment (they do not experience vection longer, nor do they tilt more) and are more aware of their postural position than are age-matched control subjects. (Invest Ophthalmol Vis Sci. 2008;49:3253–3258) DOI:10.1167/iovs.07-1290

Age-related macular degeneration (AMD)—the most prevalent ocular disease in the elderly population of developed countries—affects central vision by destroying the macula.1–3 Visual functions that depend heavily on intact macula (e.g., acuity) are weakened or permanently damaged,4,5 and the severity of the impairment usually depends on the scotoma’s size. In some patients with AMD, peripheral vision is minimally affected or not at all. It is recognized that, whereas central vision is responsible for resolving fine details, peripheral vision plays an essential role in motion detection, spatial orientation, and locomotion.6–8 These functions of peripheral vision are mediated by the magnocellular visual pathway, which transmits information from the large retinal ganglion cells (parasol cells) through the M divisions of the lateral geniculate nucleus (LGN) to the visual cortex and responds vigorously to sudden, fast fluctuations of light intensity within the cells’ receptive fields. These big variations are associated with large, fast-moving objects.9–11 Knowing that central and peripheral vision serve different purposes, it is of both theoretical and practical interest to understand how the visual system of patients with damaged central vision but with intact peripheral vision process motion—in particular, self-induced motion.

Studies in self-induced motion provide important information about the role of peripheral vision in detecting motion.12–14 The sensation of self-motion induced in a stationary observer exposed to large moving visual fields is called vection. Scenes moving vertically or horizontally in a flat plane induce a sensation of self-translation (linear vection), whereas rotating scenes induce a sensation of self-rotation (circular vection). Depending on the axis about which the scenes rotate, circular vection could be called roll, yaw, or pitch vection.15 It has been suggested that peripheral vision is more important than central vision in inducing vection.12 Vection depends on stimulus size: the larger the stimulus, the stronger it induces.12,16 Post17 also showed that peripheral and central vision are equally effective in inducing vection when controlling for stimulus area. However, in subsequent research it has been found that, when controlling for the stimulus area, peripheral vision is still more effective in inducing vection than is central vision in certain conditions—specifically, when stimuli have low spatial frequency, and the peripheral stimulus is perceived as background, or when no fixation stimulus is provided.13,14,18 Large stimuli, with or without the central 30° area, induce similar vection in persons with normal vision.12,14 It is tempting, then, to assume that patients who have lost central vision but have peripheral vision unaffected, as is the case of some patients with AMD, will experience vection similar to that of age-matched control subjects with intact vision when exposed to large, moving visual fields.

Indeed, this prediction is supported by an experimental study that examined vection in patients with AMD.19 In this study, a random-dot pattern was projected onto the inner walls of a hemispherical dome, inducing circular (yaw) vection in observers seated inside the dome. Vection latencies (the time between stimulus onset and the participant’s first response) were measured in patients with unilateral AMD and age-matched control subjects. The sizes of the absolute central scotomas of the affected eyes were well within 30° in diameter, but they produced severe acuity loss. Vection latency in the affected eye was compared with that in the unaffected eye in the patients with AMD, and left eye latency was compared with right eye latency in the age-matched control subjects. The
results showed that vection latency of the affected eye was not different from that of the unaffected eye. The latencies of the affected eye of the patients with AMD were only slightly lower than those of the control participants.

The prediction stated above, however, may not be valid in the case of people with bilateral AMD. Visual processing of people with bilateral central vision loss may differ from that of people with normal vision or those with unilateral AMD. There is evidence that some degree of plasticity of the visual system occurs in patients with a long history of bilateral AMD. A study using functional (f)MRI showed that the visual cortex, which normally responds to foveal stimulation, was vigorously activated by peripheral visual stimuli but not by foveal stimuli in patients with bilateral AMD. This was not the case in observers with normal vision: the foveal cortex was not activated by peripheral stimuli. Thus, important reorganization of visual processing takes place in patients without functional foveae. Therefore, it is reasonable to assume that the function of peripheral vision, especially of the better-seeing eye, of the patients with bilateral AMD would be enhanced. This process may not occur in patients with unilateral AMD, since the unaffected eye has a functional fovea and can compensate for the visual loss produced by the diseased eye.

The primary purpose of the present study was to examine the contribution of peripheral vision in the better-seeing eye of patients with bilateral AMD in inducing self-motion. The performance of patients with AMD was compared with that of age-matched control subjects. Two types of vision were used: circular (roll) and linear. Vection latency and total vection time were measured in both conditions. The roll vection condition provided additional information about subjective and objective measures of body tilt. We hypothesized that vection induced in persons with bilateral AMD (better-eye tested) would be stronger than that induced in the age-matched control subjects.

**METHOD**

**Participants**

Eleven patients (mean age, 79.3 ± 8.3 [SD] years) with a confirmed diagnosis of AMD participated in the study. Only the better-seeing eye was tested. The patients’ demographic characteristics and visual profiles are summarized in Table 1. The retinal microperimetry of the better-seeing eye of each patient was recorded with a microperimeter (MP-1; Nidek Technologies Srl, Vigonza, PD, Italy), using a static semiautomatic perimetry test. Only patients with a measurable absolute central scotoma without islands of functional central retina and a lute central scotoma without islands of functional central retina were included in the study.

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**TABLE 1. Visual Profiles of the Patients with AMD**

Initially, 15 age-matched control participants were included in the study; however, three were excluded because they did not experience vection in any of the conditions. Thus, the age-matched control group consisted of 12 volunteers (6 men, and 6 women, mean age, 74.3 ± 6.4 [SD] years) who had visual acuity ranging from 0 to 0.3 logMAR. Both the patients and the control volunteers were recruited from referrals to the Eye Clinic at the Toronto Western Hospital and from two private clinics. They had no history of neurologic or vestibular diseases, cognitive impairment, or other significant ocular diseases with the exception of mild cataract. Informed consent was obtained from all participants. The research was conducted in the Ocular Motor Laboratory at the Toronto Western Hospital, approved by the University Health Network Research Ethics Board, and conducted in accordance with the tenets of the Declaration of Helsinki.

**Apparatus and Stimuli**

Stimuli were random-dot patterns with a dot size of 2°, presented on a 192° × 146° projecting area, at a viewing distance of 40 cm. The dot density was 0.022 dot/cm² (300 dots over the whole area). The dots were white with a luminance of 59 cd/m², and the background was black with a luminance of 0.82 cd/m². The Michelson contrast ratio of the pattern was 0.97 (Fig. 1). The stimuli were generated with graphics and psychophysical testing software (VPixx Technologies, Inc., Montréal, QC, Canada) and rear-projected on a large flat screen. There were two conditions: the linear vection condition and the roll vection condition. In the linear vection condition, the dots moved from left to right with a velocity of 60°/s. Once the dots appeared from the left of the testing area, they were continuously present until they disappeared on the right. In the roll vection condition, the dots rotated clockwise about a horizontal axis perpendicular to the spinal axis, in the sagittal plane of the head (the roll axis) with an angular speed of 45°/s. For both conditions, the dot density was constant at all times.

**Measurements**

For both conditions we measured vection latency (the time between stimulus onset and the participant’s first response) and total vection time (the total time self-motion was perceived during each condition). These measurements were performed with a USB button-response box connected to a laptop computer. Four additional measurements were recorded in the roll vection condition: the perceived tilt angle, the
they experienced vection (total time measure). At the end of the roll moving (latency measure) and to keep the button pressed as long as they perceived themselves to be sick, vertigo, or habituation. The participants were to press the button on the response box as soon as they perceived they had tilted by moving the stick mounted on a protractor.

**Data Analysis**

For each condition, the performance of the AMD group was compared with that of the age-matched control group by using independent-samples t-tests. The strength of the relationships between variables was assessed with Pearson r correlations and with Kendall’s τb rank correlation. The α level was set at 0.05.

**RESULTS**

**Linear Vection**

All participants in both groups experienced induced motion in the linear vection condition. The vection latency of the AMD group (mean, 17.6 ± 12.1 [SD] seconds) was compared with that of the control group (mean, 29.1 ± 15.9 [SD] seconds). The difference between the two means was large (mean difference, 11.5 [SD] seconds), but it only marginally surpassed the significance level of 0.05 (t_{21} = 1.9, P = 0.06). A lack of power due to small sample sizes could account for the result of the t-test; yet, the mean latency of the AMD group fell outside the 95% confidence interval (CI) of the age-matched control group (95% CI = 19.00–39.20) and 7 of 11 patients in the AMD group fell below the lower limit of the 95% CI of the age-matched controls. The comparison between the two groups is illustrated in Figure 2. The mean total vection time of the AMD group (mean, 60.2 ± 36.2 seconds) was not significantly different from that of the age-matched control group (mean, 71.2 ± 32.1 seconds).

**Roll Vection**

All participants in the control group, but only 8 of 11 patients with AMD experienced induced motion in the roll vection condition. The mean latency of the AMD group (mean, 9.0 ± 7.1 seconds) was significantly smaller (t_{18} = 2.3; P < 0.05) than that of the age-matched control group (mean, 22.6 ± 15.7 seconds). The results are shown in Figure 3. Similar to the linear vection condition, the mean roll vection total time of the AMD group (mean, 53.1 ± 34.0 seconds) was not significantly different from that of the age-matched control group (mean, 62.5 ± 26.1 seconds).

**Tilt Measures**

**Mean Tilt Angle.** This angle represented the mean tilt value recorded during the 2 minutes of testing. The mean tilt...
angle was almost identical in the two groups: 8.2 ± 5.3° in the age-matched control group and 8.4 ± 2.7° in the AMD group.

**Maximum Tilt Angle.** This angle measured the maximum tilt during roll vection. There was no difference between the two groups. The maximum tilt angle of the age-matched control group ranged from 4.2° to 26.4° (mean, 13.4 ± 8.2° [SD]) and that of the AMD group ranged from 8.6° to 25.1° (mean, 14.6 ± 5.2°).

**Minimum Tilt Angle.** The mean and maximum tilt angles may have been affected if the participants had tried to correct the posture and lean in the opposite direction of tilt. The minimum tilt angle measured the angle in the opposite direction of tilt. Minimum angles of the age-matched control group ranged from 0° to −1.06° (mean, −0.3 ± 0.3°), and that of the AMD group ranged from −0.1° to −2.1° (mean, −0.7 ± 0.6°). These values were considered too small to have an impact on the mean and maximum angles.

**Perceived Tilt Angle.** This angle represented a self-reported measurement of maximum tilt, and it had a very large variability. The estimated tilt angle of the age-matched control group (mean, 16.8 ± 15.5°) was smaller, but not significantly, than that of the AMD group (mean, 24.5 ± 21.3°). We also looked at the relationship between the maximum tilt angle (objective measure) and the perceived tilt angle (subjective measure) within each group. There was no correlation between the two measures in the age-matched control group (Pearson $r_{10} = -0.18$, $P = 0.58$), but there was a very strong correlation in the AMD group (Pearson $r_{6} = 0.81$, $P < 0.05$). The results are plotted in Figure 4.

The bottom panel of Figure 4 shows that the strong correlation could have been the effect of an outlier. Further analysis showed that the point defined by maximum tilt of 25° and estimated tilt of 65° is not a far outlier. Tabachnick and Fidell recommend assigning the outlying case a raw score that is one unit larger than the next most extreme score in the distribution. Therefore, this case comes closer to the mean but maintains its rank order. The correlation still reached a significant level after this procedure (Pearson $r_{6} = 0.61$, $P = 0.05$). This transformation is illustrated in Figure 4.

**Relationship between Absolute Scotoma Area and Vection.** The MP-1 microperimeter has a feature that allows the measurement of the scotoma area in square millimeters. However, according to the user manual, this feature is recommended for clinical use only, because there could be large variations in the linear measurements. Since an exact measurement of the scotoma area was not possible, we decided to estimate the scotoma as a disc with a diameter equal to the average of the horizontal and vertical sizes of the absolute scotoma.

Because there was large variability in the scotoma area of the patients with AMD and because the sample size was too small to afford deleting the outlying cases, a nonparametric Kendall $\tau$ rank correlation test was used to evaluate the strength of association between the size of the absolute scotoma and vection.

There was a negative relationship between the estimated scotoma area and the latency in the linear vection condition (Kendall’s $\tau_{b10} = -0.527$, $P < 0.05$, two tails) and in the roll vection condition (Kendall’s $\tau_{b10} = -0.50$, $P < 0.05$, one tail). There was no relationship between area and total vection time in either of the conditions.

**Discussion**

The purpose of this study was to investigate the role of peripheral vision of the better-seeing eye of people with bilateral central vision loss in inducting vection. The results of the present study confirm our prediction that vection is enhanced in these patients. We hypothesized that the visual processing of people with bilateral central vision loss may differ from that of people with normal vision or from those with unilateral AMD. The loss of bilateral central vision results in an enhanced role of the peripheral vision, presumably due to the plasticity of the visual system. There is evidence of reorganization from an MRI study involving patients with a long history of bilateral AMD. However, patients with unilateral AMD may not benefit from the same degree of plasticity in the visual system since the unaffected eye usually compensates for the visual loss produced by the diseased eye.

Our results show that patients with bilateral central vision loss experience vection sooner than do age-matched control subjects. Vection latency in the roll vection condition was significantly shorter for the AMD group than for the control group. In the linear vection condition, 7 of the 11 patients in the AMD group as well as the mean of this group fell outside of the 95% CI for the control subjects. A larger sample size would probably have accentuated these differences and provided greater power. It was, however, unexpectedly difficult to find patients with AMD who met our criteria: localized, dense scotoma in the better-seeing eye with no healthy islands of central vision and a peripheral retina unaffected by the disease. Yet, the effect was strong enough to conclude that, despite their loss in visual function, patients with bilateral AMD who rely only on their peripheral vision experience vection sooner than people of similar age with normal vision.
The size of the absolute scotoma area correlated negatively with vection latency, but not with total vection time. That is, the larger the scotoma size, the shorter the latency. The size of the stimulus was much larger than that of the absolute scotoma area, so the results cannot be attributed to the loss of stimulus area. Studies have shown that masking the central part of large moving stimuli with black disks of different sizes has little influence on vection in people with normal vision.12,14 That is, large moving stimuli, with and without the central part blacked out induce similar vection. However, this does not mean that masking the central visual field with a black disc suppresses the central vision of people with an intact fovea. Moreover, it has been shown that vection latency in people with unilateral AMD is slightly, but not significantly diminished.15 Our findings suggest that, when there is no visual input from central vision as is the case of patients with bilateral AMD and dense central scotomas, vection is enhanced.

Of interest, despite the differences in vection latencies, the two groups did not differ in terms of how long they experienced vection in both conditions. That is, patients with AMD experienced vection sooner, but not longer, than the age-matched control subjects. It has been suggested that vection occurs because movements of large visual scenes, relative to a stationary observer, produce the same physiological response as stimulation of the vestibular system arising from body rotation and translation.15 The interpretation of the visual motion of the scenes as self-movement was canceled in the absence of vestibular inputs after a similar amount of time in both groups, perhaps because the participants from the two groups differed only in visual performance; their vestibular systems were equally aged, but unaffected by any vestibular diseases. Thus, it took the same amount of time to solve the conflict correctly between visual and vestibular inputs.

The objective measures of the tilt angles during the roll vection condition were practically identical in the two groups. The mean angle during roll vection was approximately 8° in the two groups, and the average maximum angle was approximately 14°. Held et al.4 found that tilt during roll vection increases with the velocity of stimulus rotation up to 40°/s, but is independent of the eccentricity of the display for normally sighted observers. Their data also show that at a stimulus speed of rotation of 45°/s (as in our case), the tilt was between 9° and 13° (p. 359, Fig. 3 in Ref. 6). It has been documented that the effect of the illusion on body tilt is limited by gravity, which elicits responses from the otolith organs and proprioceptive responses that will induce compensatory postural reactions.15,22 Thus, it is not surprising that the participants in the two groups experienced the same tilt.

There was a difference in how well the participants in the two groups estimated their tilt during the roll vection condition. Although there was no correlation between the perceived angle and the maximum tilt angle, as measured with the tilt sensor for the age-matched control group, there was a significant relationship between the two measures in the AMD group. This difference in how accurate the people in the two groups were in estimating the tilt was surprising. Why would people who rely only on visual input coming from the peripheral retina be more aware of tilt than would people with normal vision? Perhaps their visual loss has made these patients pay closer attention to their postural position in the environment, or the function of their peripheral vision, known to play an important role in orientation and locomotion, is enhanced after the loss of central vision. If this is the case, these results represent further evidence of plasticity in the visual system and highlight an adaptive aspect of these patients, making them more attuned to the moving environment.

It is important to acknowledge two limitations of the present work. First, we think that a larger sample would have provided an even stronger evidence of the enhanced role of peripheral vision in inducing self-motion in patients with bilateral AMD. Second, our measurement of scotoma size was based on static microperimetry, whereas the vection stimuli were large and in motion. It is known that stimulus size and motion influence visibility,7 and there is a possibility that the vection stimuli could have been faintly detected in central vision because they were more visible than the ones used in the static microperimetry. A kinetic microperimetry would have been more appropriate for this study, although it is very difficult to perform kinetic microperimetry on patients with such large scotomas, because their fixation is typically very unstable.23,24

In conclusion, the study of the role of peripheral vision in inducing vection in patients with bilateral AMD proved to have not only theoretical, but also practical implications. First, our results showed that people with central vision loss experience vection sooner than age-matched control subjects, suggesting that peripheral vision may play a more important role in inducing vection and that some degree of plasticity in the visual system takes place. Second, these patients adapt to the moving environment (they do not experience vection longer or tilt more) and are more aware of their postural position than are people with healthy vision. This observation implies that patients with bilateral AMD are more attuned to large moving fields, an adaptive aspect that may assist them in guidance and orientation in their environments.

Acknowledgments

The authors thank Teodor Tarita-Nistor for building the button-response box and the tilt-measuring device.

References


