

# Monocular optokinetic nystagmus in humans with age-related maculopathy

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## Abstract

**Aim**—To investigate full field monocular optokinetic nystagmus (OKN) in patients with age-related maculopathy (ARM) and relative central scotoma.

**Methods**—Six patients aged 59–88 years with bilateral ARM and an aged-matched control group of six patients aged 54–83 years were examined. Visual fields were assessed with a Humphrey field analyser using the threshold 30-1 routine. Monocular full field horizontal optokinetic stimuli were presented on a hemicylindrical screen subtending 172° horizontally and 50° vertically. The stimulus was a projected random dot pattern and three stimulus velocities were used, 30, 50, and 70°/s in both nasalward and temporalward directions. Each trial lasted between 30 and 40 seconds and eye movements were monitored using infrared oculography.

**Results**—The ARM patients had relative central scotomas with an average depth of 10 dB. Neither the ARM nor the age-matched groups displayed any directional preponderance or a buildup of the slow phase eye velocity with time. No statistically significant difference in the gain was found between the two groups ( $p>0.05$ ).

**Conclusions**—Marked central field loss in ARM does not significantly impair OKN gain. This supports the view that complete central retinal integrity is by no means essential and that the peripheral retina provides an important input to the generation of OKN.

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Optokinetic nystagmus (OKN) is a rhythmic involuntary conjugate oscillation of the eyes in response to a large moving visual scene.<sup>1–3</sup> The nystagmus is made up of slow phases in the direction of stimulus movement which are interrupted at regular intervals by fast phases (saccades) in the opposite direction. A measure of the effectiveness of the optokinetic response

in reducing retinal image slip is the gain which is defined as the ratio of the slow phase eye velocity to the stimulus velocity.

Over the past 20 years a number of researchers have examined the effect of stimulus size and position on the gain of OKN.<sup>4–10</sup> When selective stimulation of the central retina has been compared with peripheral retinal stimulation (by masking off portions of the central regions of the visual field), the OKN gains have been found to be reduced. Typically the OKN gain for a full field stimulus moving at a velocity of 40°/s is reduced by a factor of 2 when the central 12.5° of the field is masked off.<sup>11</sup> These studies illustrate that, although the gain of the OKN is significantly higher for combined central and peripheral retinal image slip at low stimulus velocities, the peripheral retina is still capable of giving a reasonable response.

The effect of central visual field loss from pathological central scotomas has also been investigated. In 1982 Yee and his colleagues<sup>12</sup> published a very brief report that six elderly patients, with age-related maculopathy (ARM) and unilateral central scotomas exhibited a slight lowering of the horizontal OKN gain to a unidirectional motion of vertical stripes. The size of the scotomas ranged from 5° to 15° but no indication as to their depth was given.

The purpose of this study was, therefore, to extend the work of Yee and his colleagues. With the inclusion of a control group we have examined in greater detail whether the presence of a relative central scotoma influences the gain of monocular OKN and, further, whether there is any disturbance in the symmetry of the OKN response in patients with ARM.

## Patients and methods

### SUBJECTS

Six patients (age range 59–88 years; mean 72.3 (SD 10.1)) with bilateral ARM participated in the experiments. Ophthalmic investigations revealed that each patient exhibited a dry type of ARM.<sup>13–15</sup> Monocular visual acuities, with a

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Table 1 Clinical details of the subjects with age-related maculopathy

Subject	Age (years)	Sex	Visual acuity (logMAR)		Visual field defect (size and average depth)	
			RE	LE	RE	LE
1	88	M	0.85	0.95	± 6°; 10 dB	± 12°; 13 dB
2	79	F	1.17	1.01	± 8°; —	± 8°; —
3	70	M	0.45	0.47	± 6°; 3 dB	± 6°; 6 dB
4	59	M	0.07	0.61	± 1°; 8 dB	± 6°; 9 dB
5	72	F	0.60	HM	± 12°; 16 dB	—
6	66	M	0.86	1.17	± 12°; 20 dB	8°SN; IN; 22 dB 6°ST; 3 dB

dB = decibels. HM = hand movements. IN = inferior nasal. SN = superior nasal. ST = superior temporal.

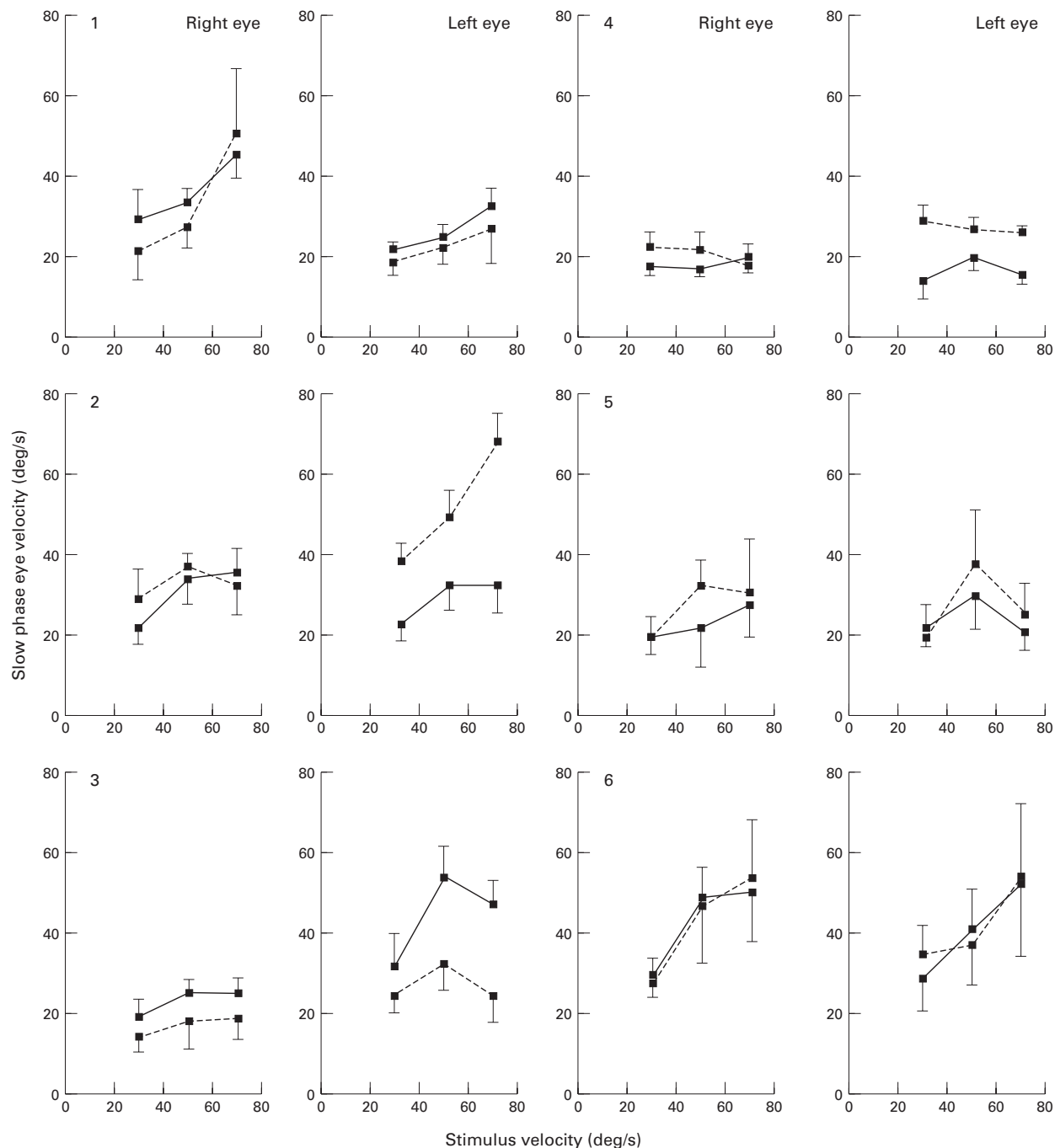


Figure 1 The relation between slow phase velocity and stimulus velocity for the right and left eye of each of six age-matched control patients for unidirectional optokinetic stimulation. Three stimulus velocities are illustrated: 30, 50, and 70°/s. The direction of the stimulus was either nasal-temporal (solid line) or temporal-nasal (broken line). The vertical bars indicate the standard deviation of the mean of 10 slow phases.

96% contrast Regan chart, ranged from 1.17 to 0.07 logMAR, with one eye only perceiving hand movements. The aged-matched control group consisted of six patients (age range 54–83 years; mean 70.3 (SD 9.7)). They had no history of ocular disease, showed no signs of ocular pathology, and monocular visual acuities ranged from 0.21 to  $-0.19$  logMAR. None of the patients in any of the three groups had a history or sign of strabismus.

#### VISUAL FIELDS

Monocular central visual fields were assessed with a Humphrey field analyser (model 630)

using the threshold 30-1 routine.<sup>16 17</sup> Each session commenced with a 5 minute adaptation period during which the patients were given instructions on the perimetric task. Incremental target intensities were presented in 72 positions over 30° of the central field using a target size of 0.25 mm<sup>2</sup> and a background luminance of 45 lux. For foveal threshold assessment, the fixation point was replaced by a diamond pattern of four yellow light spots and the test target was presented at the centre of the visual field. The duration of the test stimulus was set at 200 ms and presented in random order. Natural pupils and the appropriate refractive

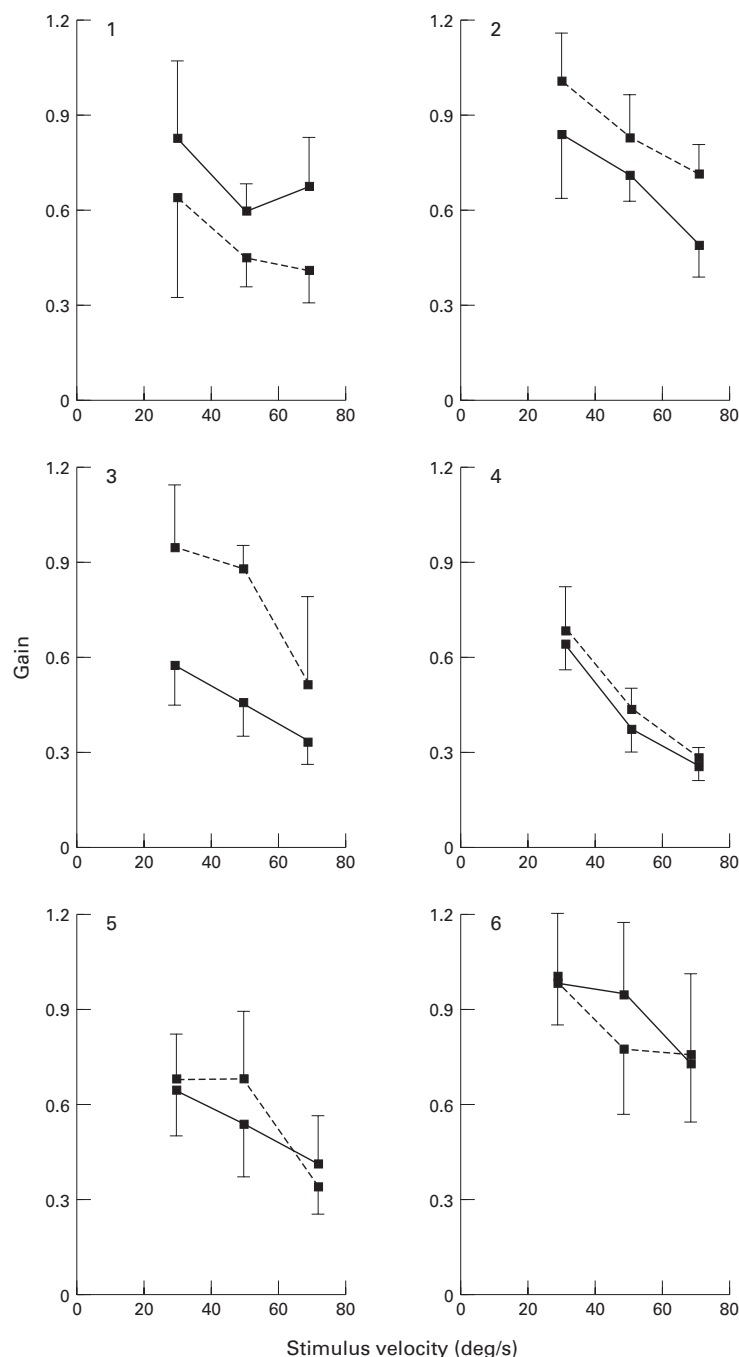


Figure 2 The relation between optokinetic gain and stimulus velocity for each of the six age-matched control patients. The direction of the stimulus was either nasal-temporal or temporal-nasal and the data of the optokinetic responses, elicited under both conditions, have been averaged for each patient's right (solid line) and left eye (broken line). Vertical bars represent the standard deviation of the mean of 20 slow phases (10 measurements for each of the two directions).

correction for the 33 cm viewing distance were used. Amsler charts were also used to indicate areas of field distortion and/or field loss.

OPTOKINETIC STIMULI

Full field horizontal optokinetic stimuli were presented on a hemicylindrical white screen subtending 172° horizontally and 50° vertically at a distance of 1.3 metres from the subject. The stimulus consisted of a projected random dot pattern (contrast 85%), each dot subtending a diameter between 1.5° and 2.5°, giving a

dot density on the screen of 46 dots/m<sup>2</sup>. Three stimulus velocities were used, 30, 50, and 70°/s, in both rightward and leftward directions and the duration of each trial run was between 30 and 40 seconds.

EYE MOVEMENT RECORDING

Monocular horizontal eye movements were recorded for both eyes separately using infrared oculography (resolution 10<sup>1</sup> arc) and displayed on a four channel strip chart recorder. Head movements were minimised with a chin cup and forehead rest and the screen was viewed monocularly. All subjects were instructed to gaze passively at the centre of the screen and to keep the stimulus as clear as possible. A calibration procedure of plus or minus 8° horizontal saccades to a 0.5° circular white target projected by a mirror galvanometer was undertaken at the start of the experimental run and repeated before each change in either stimulus velocity or direction. The stimulus was made to move either temporalwards (nasal to temporal, N-T) or nasalwards (temporal to nasal, T-N). At least 5 seconds after stimulus onset the mean velocity of 10 consecutive slow phases was calculated for each stimulus velocity and stimulus direction. OKN slow phase velocity was determined from the gradient drawn through each slow phase.

The tenets of the Declaration of Helsinki were followed in this research. Informed consent was obtained from all subjects after the nature and the possible consequences of the study had been explained.

Results

VISUAL FIELDS

The age-matched control group exhibited no distortions for the Amsler grid assessment and incremental threshold testing showed no loss of sensitivity within 30° of the centre of the visual field. All the patients with ARM indicated the presence of distortion and missing areas of the Amsler grid. With the Humphrey field analyser all exhibited foveal and parafoveal relative scotoma with the field defect size varying from 1° to 12° from the centre. Generally the scotomas were regular with the average depth of defect being around 10 dB (see Table 1). It was not possible to carry out a quantitative visual field evaluation on patient No 5 but an Amsler chart assessment revealed a field loss of plus or minus 8°.

OPTOKINETIC RESPONSE

All the eye movement traces showed a characteristic OKN with no slow phase velocity buildup. The relation between slow eye velocity and stimulus velocity during monocular N-T and T-N target motion for each of the six age-matched control patients are shown in Figure 1. Generally asymmetries for the two stimulus motion directions were small. In order to give a clearer indication of the efficiency of the OKN, the gain was plotted against stimulus velocity for the right (solid lines) and left (broken lines) eyes (Fig 2). Typically gains were found to be between 0.9 and 0.6 for stimulus velocities of 30°/s but would drop by around 0.3 for

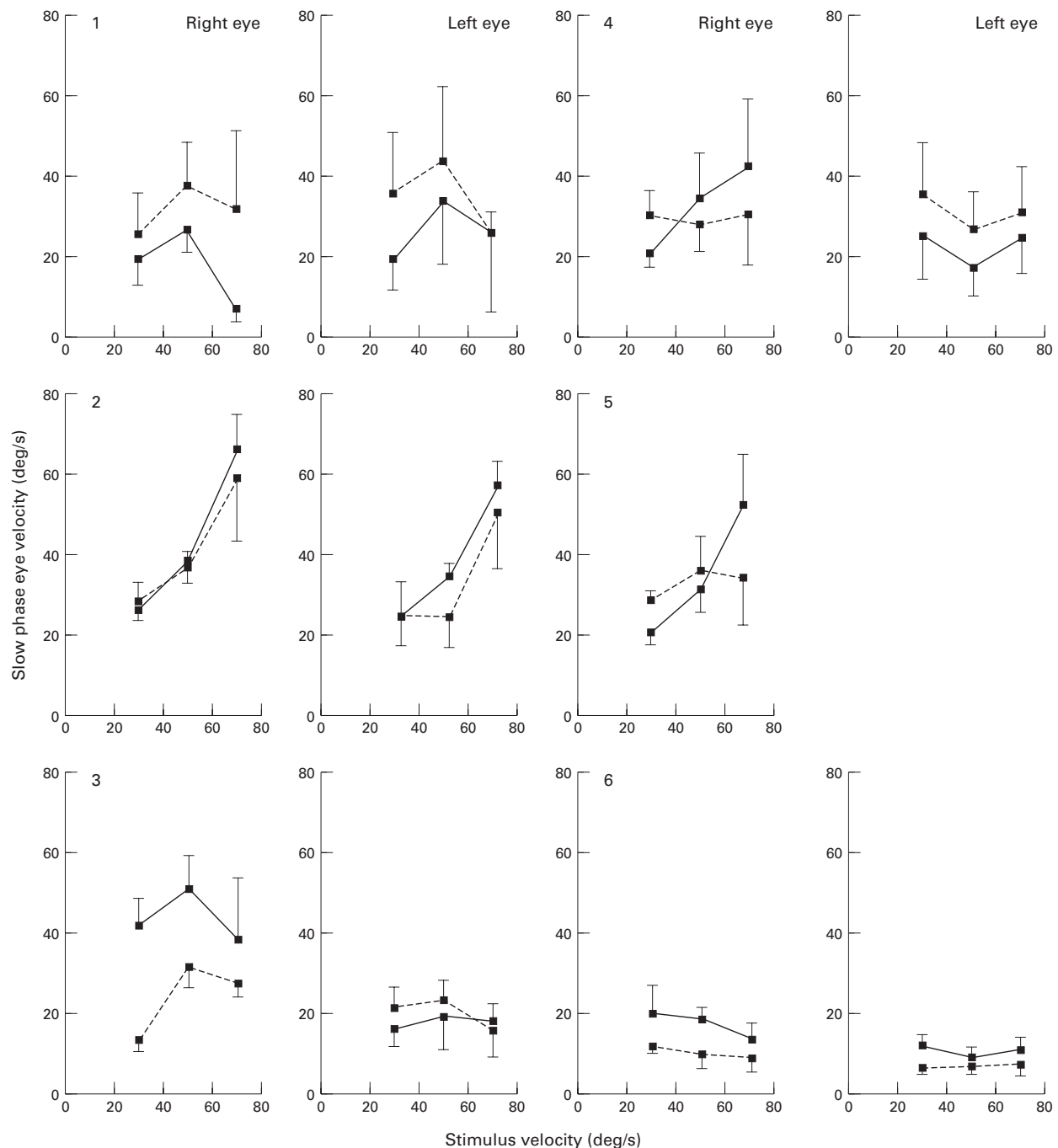


Figure 3 The relation between slow phase velocity and stimulus velocity for the right and left eye of each patient with ARM under unidirectional optokinetic stimulation. Three stimulus velocities are illustrated: 30, 50, and 70°/s. The direction of the stimulus was either nasal-temporal (solid line) or temporal-nasal (broken line). The vertical bars indicate the standard deviation of the mean of 10 slow phases. The left eye of patient No 5 was not examined.

velocities of 70°/s. The mean gains for the right and left eyes were found to be 0.75 (SD 0.15) and 0.82 (0.17) respectively and are a little lower than can be expected in a much younger control group.<sup>10,11</sup> No statistically significant directional asymmetries were apparent at any of the three stimulus velocities ( $p > 0.05$ , Student's paired  $t$  test).

Slow phase eye velocity and OKN gain plots for each of the six patients with ARM are illustrated in Figures 3 and 4, respectively. Gains were typically between 0.9 and 0.6. No statistically significant monocular directional asym-

metry in the mean slow phase eye velocity was found for any of the patients with ARM ( $p > 0.05$ ) at any of the three stimulus velocities (Fig 5). Finally, Figure 6 shows how the mean OKN gain is affected by the stimulus velocity for the two patient groups. There was no statistically significant difference between the two functions ( $p > 0.05$ ).

## Discussion

### MONOCULAR OKN GAIN

The aim of this study was to investigate the effect of relative central scotomas caused by

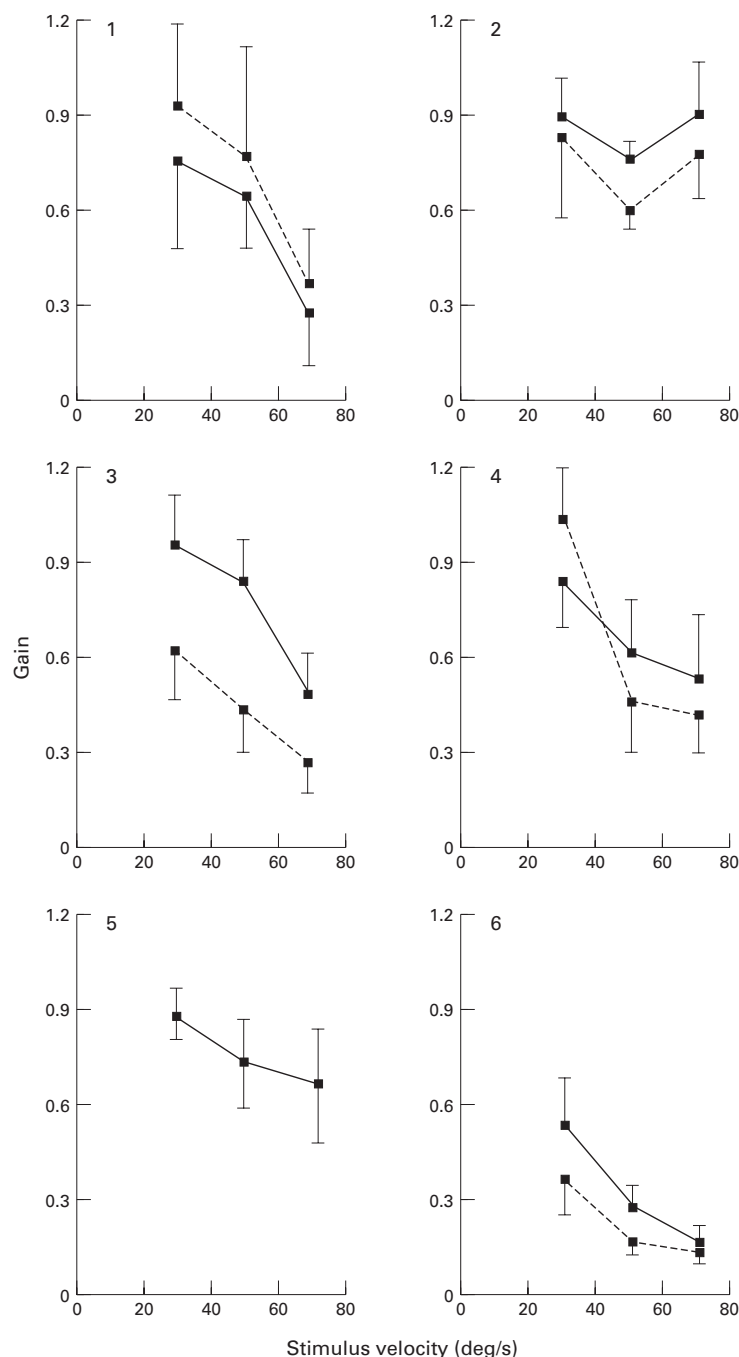


Figure 4 The relation between optokinetic gain and stimulus velocity for each of the six patients with ARM. The direction of the stimulus was either nasal-temporal or temporal-nasal and the data of the optokinetic responses, elicited under both conditions, have been averaged for each patient's right (solid line) and left eye (broken line). Vertical bars represent the standard deviation of the mean of 20 slow phases (10 measurements for each of the two directions) The left eye of patient No 5 was not examined.

ARM on the monocular horizontal optokinetic response. To examine the possibility of whether the age of the patient may influence the OKN gain an aged-matched control group was also investigated. The OKN gain of the aged-matched group was found to be consistent with the findings reported in the literature.<sup>19-22</sup> This decrease in OKN performance is not only restricted to patients above 60 years of age but reflects the continuous decline in OKN gain which affects younger patients from the age of 40 onwards. It has also been reported that both

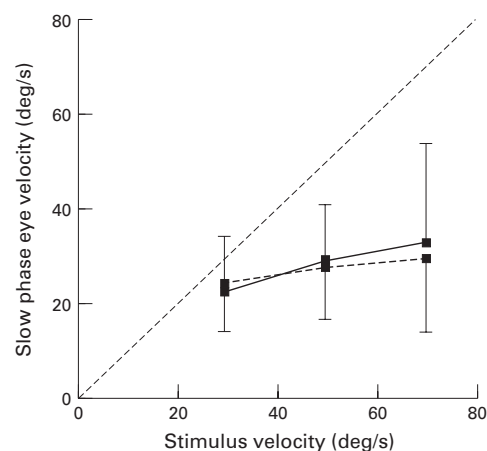


Figure 5 The relation between the mean slow phase eye velocity and stimulus velocity of the six patients with ARM, for nasal-temporal (solid line) and temporal-nasal (broken line) directions. Vertical bars indicate the standard deviation of the mean which is made up of the 11 ARM eyes tested. The line at 45° to the two coordinates represents a gain of unity.

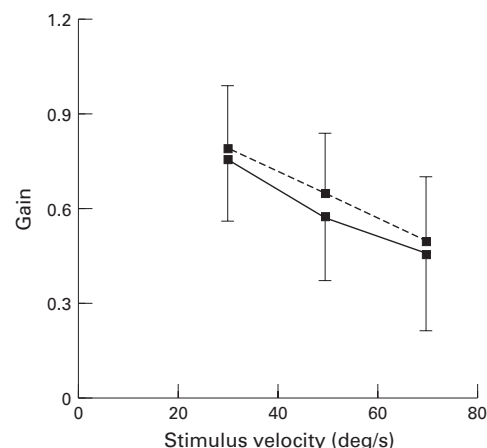


Figure 6 The effect of full field unidirectional stimulus motion on the averaged gain for the age-matched control (broken line) and age-related maculopathy (ARM) (solid line) patient groups. Vertical bars indicate the standard deviation of the mean for the control and ARM groups.

the early and late components of the OKN are affected suggesting that both the direct smooth pursuit element, and the indirect velocity storage mechanism have corresponding reductions in efficiency.<sup>19</sup> Not surprisingly, functional age-related changes in the gain of the visual tracking system have also been reported.<sup>23</sup>

Although Figure 6 illustrates that the mean gains for the control and ARM patient groups did not differ significantly there were, on occasions, large intrasubject OKN differences.<sup>24,25</sup> This variability is not unexpected in OKN studies since, although the experimental task is a passive one, the level of attention given to the stimulus during each experimental run can strongly influence the gain of the OKN.<sup>26-28</sup> It is pertinent to note that some investigators using artificial central field scotomas found large reductions in OKN gain.<sup>4,6,7,29</sup> However, it is likely that the nature of their experimental design may have been, in part, responsible for this discrepancy. In particular, the edges of the masks used to create the central field loss are

very likely to have contributed to the suppression of the OKN.<sup>8 11 25</sup>

Our present findings confirm those obtained by Yee and his colleagues<sup>12</sup> who also reported a modest but not significant lowering of OKN gain in ARM. Thus it appears that in patients with ARM a marked reduction of macular vision per se does not dramatically impair OKN gain nor reveal any increased preference for temporal to nasal stimuli. It thus appears that the remaining central retinal function together with the intact peripheral retina must provide an important drive to the optokinetic response. Support for this argument comes from an OKN study on patients with retinal lesions by Baloh and colleagues<sup>30</sup> which suggested that an overlap in signals derived from the fovea and peripheral retina may occur, since central and peripheral retinal lesions are not entirely selective in their effect on either the direct or indirect OKN pathway.

#### MONOCULAR OKN ASYMMETRY

There are now a number of reports that in the first few weeks after birth infants show a strong monocular OKN asymmetry with the T-N response being readily elicited while the N-T response is weak or absent. Thereafter, symmetrical responses are recorded at around 3 months.<sup>31-33</sup> These developmental changes in monocular OKN have been linked to cortical binocular development.<sup>34-36</sup> One possible suggestion for this is that the postnatal development of binocularly driven cortical projections to the nucleus of the optic tract supplement the innate direct inputs and thereby provide the N-T response.<sup>37 38</sup> An alternative proposal for the initially poor response to temporalward moving stimuli is that in newborn infants the visual cortex has a directional asymmetry to motion processing of nasalward and temporalward stimulus motion.<sup>39-41</sup>

In 1986, Van Die and Collewyn<sup>9</sup> reported larger monocular OKN gains for T-N compared with N-T field motion in three patients with central field scotoma secondary to retinal or neural disease. Long term loss of foveal function in patients with achromatopsia has also been reported to be responsible for a preferred T-N directional OKN sensitivity.<sup>42 43</sup> Similarly, monocular OKN asymmetries have been described in patients with long standing strabismic amblyopia.<sup>44-46</sup> It is clear, therefore, that directional disturbances of OKN are determined in part by the onset time of the foveal pathology as well as the position and depth of the scotoma in the visual field. This is compatible with the view that the development of a symmetrical monocular OKN requires an intact and functionally normal fovea during early infancy.<sup>32 47 48</sup> Moreover, the presence of a nystagmus often seen in patients with a congenital foveal pathology will undoubtedly modify the optokinetic response and can make OKN gain evaluations difficult.<sup>49-52</sup>

#### OKN AND CENTRAL SCOTOMAS

In conclusion, there is now much evidence to suggest that although the central retina strongly influences the human OKN, gains can

reach near normal levels with peripheral retinal stimulation.<sup>10 11</sup> It remains to be established whether the intact peripheral retina alone is capable of giving an appropriate feedback signal for the optokinetic response or whether residual inputs from the central retina such as a cortical filling in mechanism compensates for the absence of central vision. In support of the latter view, patients with a scotoma in their visual field often report that contours from the rest of the visual field fill in to occupy the scotoma.<sup>53</sup> Recently, Ramachandran and Gregory<sup>54</sup> concluded that spatial filling in is an active neural process that probably involves creating an actual neural representation of the surround rather than merely ignoring the absence of information from the scotoma. This situation could well occur in ARM, where a spatial temporal perceptual completion may have filled in the missing contours of the dot pattern of the optokinetic stimulus which fell within the area of the relative scotoma. Whatever the physiological mechanism, the conclusion drawn from this study provides evidence that complete central retinal integrity is by no means essential for the generation of high OKN gains. Thus the visual system of patients with functional macular loss acquired late in life is still capable of providing reasonable visually driven image stabilisation during whole field motion.

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