MONOCULAR PREFERRED RETINAL LOCI ARE INCONSISTENT WITH BINOCULAR VIEWING

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INTRODUCTION

Age-related macular degeneration is one of the leading causes of low vision. The functional consequence of macular degeneration is distortion or obstruction of foveal vision as a result of macular scotomas, or areas of reduced light sensivity. Patients with bilateral central visual field loss due to macular scotomas are known to develop an area of their retina which they use to perform visual tasks that the non-functioning fovea would normally accomplish. This «pseudo-fovea» has been named the preferred retinal locus, or PRL (Timberlake *et al.*, 1987).

Information about PRL position can be useful to clinicians planning treatment. For instance, photocoagulation requires information about residual vision at specific retinal locations and the actual use of these areas by patients with macular scotomas. While there have been studies investigating the properties of monocular PRLs, little is known about binocular PRLs because PRL information is customarily obtained monocularly. But information regarding monocular PRLs may not extrapolate to binocular viewing, which is how these patients function under most circumstances.

In binocular vision, coordinated operation of the two eyes is required, with the foveas acting as reference points and information from *corresponding points*[1] on the retinae integrated (Sullivan & Kertesz, 1979; Cooper *et al.*, 1992). Therefore when a PRL is used in one eye, the corresponding point in the other eye will be aimed at the target during binocular viewing. Assuming that PRLs develop just outside of central scotomas (Fletcher *et al.*, 1994), and that central scotoma size is similar in both eyes, if PRL position is similar across monocular and binocular tasks, then PRLs from both eyes will correspond even when tested monocularly.

Our study evaluates whether moncular PRLs from both eyes map to the same region in the visual field during binocular viewing.

METHODS

We reviewed the medical records of 93 patients [mean (M) visual acuity = 20/140 (6/42), M_{Age} = 72 years] with bilateral central scotomas (involving the central 10 degrees of visual angle [degVA]) and analyzed each patient's clinical monocular kinetic perimetry results.

[1] Corresponding points have the same angular distance both horizontally and vertically from the center of the fovea of each eye, and therefore exhibit zero binocular disparity (Tyler, 1991).

Clinical Procedure for Kinetic Perimetry

During kinetic perimetry, a perimeter is used to explore the visual field for scotomatous areas and to map their contours. The clinical kinetic perimetry results we analyzed were mapped using a Bausch & Lomb Auto-Plot which projects a movable illuminated test spot on a gray tangent screen and simultaneously tracks the position on a paper chart. While the patient continues to fixate (discussed below) monocularly a circle (subtending approximately 1 degVA) at the center of the tangent screen one meter away, the contours of both the central scotoma and the physiological scotoma (optic nerve head) are mapped. The technician moves a 6 mm test spot radially out from within the scotoma and marks on a recording chart where the patient reports seeing the light. The technician then draws a continuous contour connecting the markings on the recording chart. (A 1 mm test spot is similarly used to map relative scotoma, however we did not analyze these data.) Recording charts [Fig. 1a] depict the central 25 degVA of the visual field with both the fovea (centered) and physiological scotoma (15 degVA nasal to and 1 degVA below the fovea) preprinted. Scotomatous areas, or areas in which the patient reported not seeing the light target, are indicated by crosshatch patterns [Fig. 1b].

The patient is instructed to look at the circle at the center of the screen. If the patient is unable to fixate the circle at the center of the tangent screen, the circle is removed and the patient is instructed to look at the implied center of four thick black pericentral lines (suggestive of a large cross) on the screen, which help orient the eye even in the absence of central vision. It is unclear, however, whether the patient uses the PRL to fixate the circle or whether the patient directs the non-functioning fovea toward the fixation circle. Our analysis of kinetic perimetry results assumes that patients use their PRL to fixate the circle (or cross) at the center of the tangen screen.

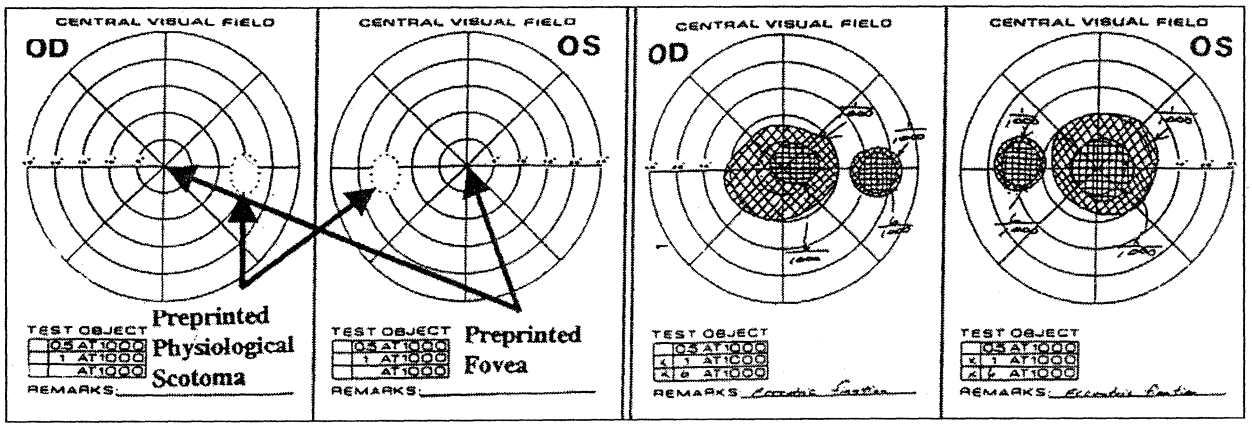

Figure 1. a: Kinetic perimetry recording charts for the right and left eyes. The central 25 degVA of visual field is depicted, with both the fovea (centered) and physiological scotoma (15 degVA nasal to and 1 degVA below the fovea) preprinted. b: Typical recording charts with both central and physiological scotoma (indicated by crosshatch patterns) each mapped with 1-and 6 mm test spots.

Quantifying Clinical Recording Sheets

If a patient uses an accentric PRL to fixate the center of the tangen screen, the mapped physiological scotoma will be displaced relative to the preprinted one (which presupposes foveal fixation). To determine the true location of the fovea, and hence the relative position of the mapped central scotoma, it was necessary to compensate for this shift. For each eye, we used the mapped position of the physiological scotoma to estimate the fovea and PRL positions relative to the central scotoma (Timberlake *et al.*, 1986). As shown in Figure 2a, we positioned a transparent copy of a preprinted recording chart (thick lines) over the mapped recording chart (thin lines) so that the mapped physiological scotoma and the transparency's preprinted physiological scotoma were superimposed. Assuming that patients used their PRL to fixate the center of the screen, the pre-

printed foveas on the transparency and the mapped recording chart in this arrangement indicate the estimated position of the fovea and the PRL (refer to arrows in Fig. 2a), respectively, relative to the central scotoma on the mapped recording chart. We also used the map to estimate the size of both the central scotoma and the physiological scotoma (by averaging their horizontal and vertical extents). Furthermore, we measured both the vertical and horizontal displacement of the central scotoma relative to the estimated fovea as well as the distance from the fovea to the center of the central scotoma (where the vertical and horizontal axes cross) for each eye.

RESULTS/DISCUSSION

Retinal periphery drives binocular fusion in subjects with central scotomas as it does in normal vision (Sullivan & Kertesz, 1979; Cooper *et. al.*, 1992), thereby yoking the eyes. Thus, when a PRL is chosen in one eye, the corresponding point of the other eye (which might be within the scotoma) will be aimed at the target during binocular viewing. Assuming that PRLs develop just outside of central scotomas, and that central scotoma size is similar in both eyes, if PRL position is similar across monocular and binocular tasks, then PRLs from both eyes will correspond even when tested monocularly.

Our data do not support this prediction. The two monocular PRLs are *not* at corresponding points, implying that PRL position differs across monocular and binocular tasks for at least one eye. We were able to estimate[2] monocular PRL position in both eyes for 60% (56) of these patients[3] [$M_{\text{Visual Acuity}}$ = 20/120 (6/36), M_{Age} = 71 years]. While mapped central scotoma size did not differ across eyes [M_{OD} = 10.4, M_{OS} = 11.0 degVA; $p > .1$] in these patients, PRL position did. The average distance between PRLs was 1.5 deg.VA. This disparity between monocular

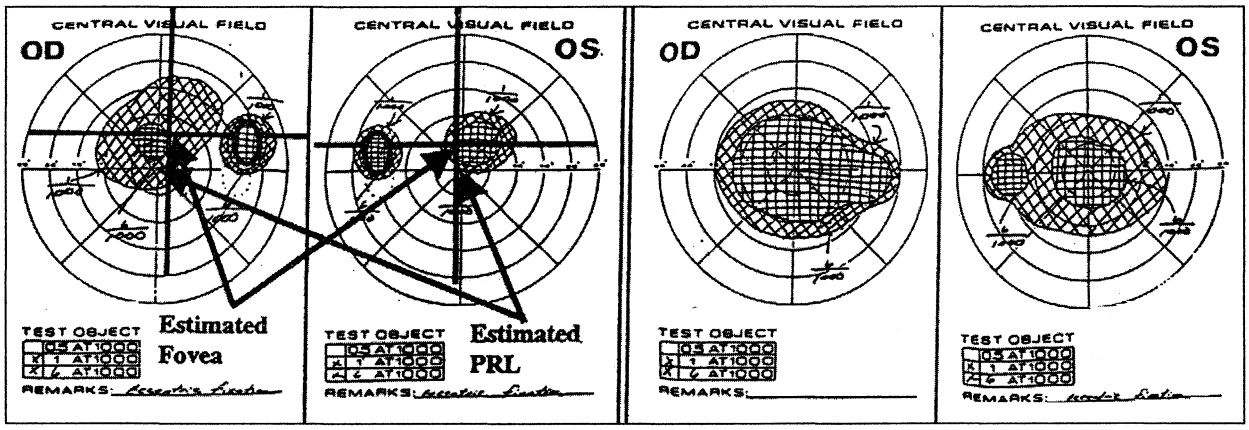

Figure 2. a: Depicts how the displacement of the mapped physiological scotoma relative to the preprinted position is used to determine the PRL. A transparent copy of a preprinted recording chart (thick lines) is positioned over the mapped recording chart (thin lines) so that the mapped physiological scotoma and the transparency's preprinted physiological scotoma are superimposed. In this arrangement, the positions of both the fovea and the PRL can be estimated relative to the central scotoma on the mapped recording chart. b: Recording charts with PRLs that coult not be estimated (OD) or were within scotoma (OS). For 31% (29) of the patients, PRL position of at least one eye could no be ascertained because the mapped scotoma was cecocentral. In 90% (101) of eccentrically fixating eyes the monocular PRL position was found within clinically mapped central scotoma (unlike the situation in Fig. 2.a).

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[2] The measurement error involved in quantifying the recording charts was negligible. For instance, ten repetitions of an author (AL) estimating a PRL position were all within 1 mm (horizontally and vertically) of each other on the recording charts (which corresponds to < 0.3 degVA).

[3] For 31% (29) of the patients PRL position of at least one eye could no be ascertained because the mapped scotoma was cecocentral (involving the physiological scotoma, the central 10 degVA, and the area in between) [Fig. 2b, OD]. For 9% (8) of the patients, the mapped physiological scotoma was centered over the preprinted physiological scotoma for at least one eye, indicating that the subject was not eccentrically fixating and could have some residual foveal vision.

PRLs in our patients agrees with recent data reported by Schuchard *et al.* (1995), in which only 40% of patients' monocular PRLs corresponded retinally (i.e., were less than 2.5 degVA apart, vertically and horizontally).

Using estimates from the literature regarding variability of fixation (defined as the average of the horizontal and of vertical standard deviations of eye position) at different eccentricities, we tested whether the disparity between PRLs could be due to increased fixation instability as eccentricity increased. We used data from three patients with central scotomas (Timberlake *et al.*, 1986) and six subjects with normal acuity (Sansbury *et al.* 1973; Bedell *et all.*, 1984) to construct a linear function expressing the relationship between eccentricity and variability of fixation [Fig. 3]. For each of our patients we used this function to estimate the variability of fixation at the patient's average PRL eccentricity. For 86% (48) of patients the distance between PRLs was more than 3x the estimated variability of fixation at their average PRL eccentricity. The disparity between monocular PRLs suggests that either the PRL of the dominant eye steers the other eye or a different PRL is used for binocular viewing in both eyes.

We also found that the mean distance between the PRL and fovea was 1.2 degVA. Since the average radius of the central scotoma was 5.3 degVA, and PRLs tipically form just outside of central scotomas, the relatively short[4] distance between the PRL and the fovea suggests (a) that central scotomas are not centered around the fovea, (b) that they are substantially smaller than mapped, or (c) a combination of these.

Since PRLs are usually on the edge of central scotomas, if the central scotomas are centered around the fovea, then the PRL eccentricity should increase as central scotoma size increases. We found no such correlation, suggesting that central scotomas were not centered around the fovea. This was corroborated by the fact that the average distance between the estimated fovea and the center of the central scotoma of each eye (0.6 degVA) is significantly different from zero [$t(95) = 13.16$, $p < 0.0001$].

We tested whether the decentration of central scotoma could be a result of an artifact. We measured the horizontal displacement of the central scotoma relative to the estimated fovea. A horizontal displacement of similar retinal direction and magnitude in both eyes might result from a difference in patient viewing distance or from an erronenous depiction of the relationship between the fovea and the physiological scotoma preprinted on the recording chart. But there was no consistent horizontal displacement across eyes: The mean displacement of the central scotoma in the left eye (0.19 degVA, temporal) was significantly different from zero [$t(55) = 3.22$, $p = .0022$], whereas the mean displacement in the right eye (0.10 degVA, temporal) was not [$t(55) = 1.67$, $p > 1$]. This difference across eyes suggests that the 0.6 degVA shift we found (from the center of the central scotoma to the estimated fovea) is nos due to patient viewing distance or the relationship between the fovea and physiological scotoma preprinted on the recording chart.

We further investigated this decentration of central scotoma by analyzing the vertical component of the shift. That is, we measured the vertical displacement of the central scotoma relative to the estimated fovea. We found that the mean vertical displacement for the right eye (0.15 degVA, inferior retina) was significantly different from zero [$t(55) = -2.39$, $p = .0203$], but the mean displacement for the left eye (0.03 degVA, inferior retina) did not differ from zero [$t(55) = -0.63$, $p > .5$]. We submit that this decentration of central scotoma toward inferior retina might be the result of a gravitational effect on the leakage caused by age-related macular degeneration.

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[4] Note that the distance between the PRL and fovea is more than 2x the 0.5 degVA radius of the foveola (Pirenne, 1967).

In 90% (101) of eccentrically fixating eyes the monocular PRL position was found within clinically mapped central scotoma [Fig. 2b, OS]. Although some patients may use a residual area of vision within the central scotoma to fixate (Fletcher *et al.*, 1994; Timberlake *et al.*, 1986), for other patients a more plausible explanation is that kinetic perimetry exaggerates scotoma size, a result of target speed and patient response time (Timberlake *et al.*, 1986). To further investigate this possibility, we timed the target speed that the technician used to map the scotomas. The technician moved the light target aproximately 2 degVA/sec when mapping the scotomas. Assuming that (a) the patient's response time was 1 sec, which is not unreasonable considering the age of our sample and that the stimulus is unpredictable and in peripheral vision (Boff & Lincoln, 1988), and that (b) the technician's response time to mark a position on the recording chart is at least 200 ms, then the mapped central scotomas would have a radius 2.4 degVA (1.2 sec x 2 degVA/sec) larger than that of the actual central scotomas. Our conjeture that central scotoma size is exaggerated by kinetic perimetry is further supported by the fact that mapped physiological scotomas are larger than the preprinted physiological scotoma.

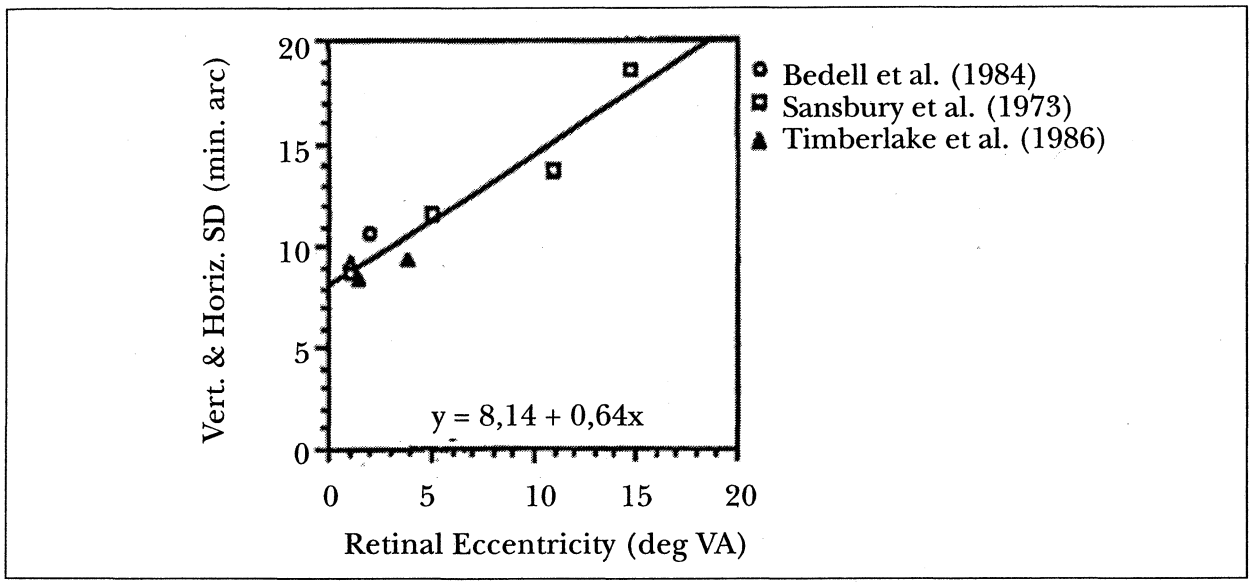

Figure 3. Estimates from the literature regarding variability of fixation at different eccentricities. Based on the estimated relationship between eccentricity and variability of fixation, we tested whether the disparity between PRLs could be due to increased fixation instability as eccentricity increased.

For each eye, we both measured and then averaged the vertical and horizontal extents of the mapped physiological scotoma in order to calculate the average diameter, and hence the radius, of the mapped physiological scotoma. The radius of the mapped physiological scotoma (4.05 degVA) differed significantly from the radius of the preprinted physiological scotoma (2.98 degVA) [$t(111) = 18.05$, $p < 0.0001$]. Note that this 1.1 degVA exaggeration of the physiological scotoma is only half of hour estimated of the exaggeration of central scotoma due to the kinetic perimetry procedure (2.4 degVA). We atribute this difference inmagnitude of exaggeration to a slower target speed for mapping physiological scotoma than for central scotoma. It is likely that the technician is biased to reduce the speed of the light targe when nearing the edge of the preprinted physiological scotoma.

Since the minimum actual central scotoma is estimated to be 1.2 degVA in radius (the distance from the fovea to the PRL), then the minimum size of the mapped central scotoma after taking into account the exaggeration of *central* scotoma size would be 3.6 degVA. The size of the mapped central scotoma might even be 0.6 degVA larger in radius (considering the scotoma are decentered relative to the fovea by that much), resulting in a mapped central scoto-

ma with a minimum radius of 4.2 degVA, comparable to that of the mapped central scotomas we analyzed (5.3 degVA). We conclude that central scotoma size is exaggerated by kinetic perimetry and that the distance from the fovea to the PRL may be a better estimate of the actual radius of the scotoma.

Our analysis of kinetic perimetry results assumes that patients use their PRL to fixate the circle (or cross) at the center of the tangent screen. An alternative interpretation presumes that patients direct their non-functioning fovea toward the fixation circle, and any discrepancy found between the preprinted and estimated foveas is due solely to fixation instability. However, this alternative is unlikely to explain our results. Data from Sansbury *et al.* (1973) suggest that even subjects with central scotomas 2x the size of our subjects' (i.e., 20 degVA in diameter) would have horizontal and vertical SDs less than 15 min arc, which is merely a fifth of the average distance we found between the PRL and the fovea.

CONCLUSIONS

Our data suggest that monocular PRLs may not be the ones used in binocular tasks. Furthermore, our data show that central scotomas are not centered around the fovea and imply that kinetic perimetry exaggerates central scotoma size. Future work will seek confirmation of these data from both Scanning Laser Opthalmoscope tests, which ascertain the retinal position of targets directly rather than by inference, and from binocular perimetry.

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