Contrast Sensitivity in Diabetic Retinopathy After Panretinal Photocoagulation

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ABSTRACT
Reports of changes in contrast sensitivity in proliferative diabetic retinopathy (PDR) patients after panretinal photocoagulation (PRP) have considered only relatively short-term results, and these have been conflicting. We evaluated contrast sensitivity changes in 30 eyes of 29 PDR patients after PRP. The patients were divided into two groups. One, group A, received PRP at one sitting, and the other, group B, at two sittings. Before and at regular intervals after PRP, all of the patients underwent a battery of macular function tests for best-corrected visual acuity, color vision, contrast sensitivity, and photostress. Contrast sensitivity was significantly affected (P < .001) in both groups immediately after PRP, but stabilized to prelaser levels by the end of 3 months. Color-vision-error scores also were significantly higher (P < .001) immediately after PRP. Best-corrected Snellen visual acuity, however, remained stable at prelaser levels. Contrast sensitivity appears to provide a more sensitive measurement of visual acuity than the Snellen chart for monitoring foveal integrity in patients undergoing PRP.

Panretinal photocoagulation (PRP) has been established as an effective treatment for proliferative diabetic retinopathy (PDR). The Diabetic Retinopathy Study1-2 clearly demonstrated PRP's long-term beneficial effects. Nonetheless, potential side effects remain a concern.2 Long-term effects such as field loss were expected and clearly have occurred in photocoagulated areas of the peripheral retina.2 However, in addition, there is increasing awareness that PRP may cause macular phototoxicity in both patients and operators.3,6

Short-term temporary loss of foveal contrast sensitivity attributable to PRP cannot be easily explained as a direct consequence of photocoagulation, since Snellen visual acuity seems to remain stable at prelaser levels (though patients have complained after treatment that their vision was less clear and bright in their treated as compared with their untreated eyes).6

The aim of the present study was to determine by a battery of macular function tests whether macular phototoxicity occurred due to PRP, with special attention directed at measuring short-term and long-term effects of PRP on central visual field function. Spatial contrast sensitivity measurements were used to supplement visual acuity testing, since previous research has shown it to be a more sensitive means of detecting subtle visual loss in diabetic as well as nondiabetic patients.7-11

METHODS
Selected for the study were patients with previously untreated PDR for whom PRP was indicated. We evaluated 29 patients with PDR who had new vessels on the optic disc or elsewhere (as determined by indirect ophthalmoscopy and fluorescein angiography) and severe pre-PRP. Inclusion criteria included a visual acuity of 20/60 or better with clear media and no clinically significant macular edema, as defined by the Early Treatment Diabetic Retinopathy Study report.12

Informed consent was obtained.
Patients were randomized into two groups by simple random sampling. Eighteen underwent PRP at one sitting (group A); the remaining 11, in two sittings, separated by a 1-week interval (group B). Group A was examined at 1 week and 3 months; group B at 1 week, 2 weeks, and 3 months.

Before treatment, all of the patients had a full ophthalmic examination, including slit-lamp biomicroscopy and indirect ophthalmoscopy. Also, fluorescein angiography and fundus color photography were performed. Snellen visual acuity, color vision, and contrast sensitivity were assessed in one eye of each of the patients before and at regular intervals after PDR. In addition, a photostress test was administered and fluorescein angiography performed at each of these points. The nonlasered fellow eye was not evaluated. Throughout the study, the tester was masked to previous results.

All of the patients underwent PRP using argon blue-green laser (Coherent Inc, Palo Alto, Calif) (group A, at one sitting; group B, at two sittings, separated by a week). Treatment was performed through a maximally dilated pupil using a panfunduscope Rodenstock lens. Fourteen hundred burns were applied to each eye, each with a spot size of 100 to 200 μm and a duration of 0.1 second. The power was adjusted to produce a mild to moderate retinal take. No additional laser treatment was given during the 3-month duration of the study. No region closer than 12° to 15° to the fovea was photocoagulated. Thus, all of the treatments were applied to retinal regions considerably peripheral to the 4 × 4-degree foveal region used for measuring spatial contrast sensitivity and visual acuity.

Biomicroscopy and fluorescein angiography were used to check for evidence of foveal edema and regression of neovascularization at each visit. Changes in the capillary-free zone were not evaluated, since such a determination was not included in our study design.

Best-corrected visual acuity was recorded on a projected Snellen chart at a distance of 6 m. Color vision was assessed with a Farnsworth-Munsell 100 Hue test. The error score was calculated from the sum differences between the number of each cap and its standard position.13 Color-vision-error scores for normal controls range from 97.75 ± 16.01; for diabetics without retinopathy, from 169.26 ± 16.42; for diabetics with retinopathy, from 195.33 ± 25.6; and for diabetics with maculopathy, from 246.8 ± 26.4.10 The photostress test was performed by exposing the patient monocularly to the maximally illuminated Welch Allyn ophthalmoscope at a distance of 15 cm in an untreated pupil for 30 seconds. The time taken for the acuity to return to the preexposure level was regarded as the macular recovery time.

We evaluated contrast sensitivity using the Cambridge low-contrast-sensitivity charts (obtained through the courtesy of Dr A.J. Wilkins, Medical Research Council Applied Psychology Unit, Cambridge, England9) (Fig 1). The charts consist of eight pairs of plates. One plate in each pair is blank and the other contains a square-wave grating. The gratings are generated by alternating matrices of dots with plain, unprinted paper. At a distance of 6 m, the individual dots can no longer be appreciated; thus, it is the variation in the contrast in the region containing the dots that is perceived by the observer. The contrast levels of the gratings in the eight plates are, from lowest to highest, 0.38%, 0.50%, 0.70%, 0.98%, 1.59%, 2.24%, 2.82%, and 9.63%. The gratings have a spatial frequency of 4 cycles/degree when viewed at a distance of 6 m. Although the charts measure the contrast sensitivity threshold at only one frequency, it is the frequency to which the visual system is most sensitive.9 Furthermore, although the contrast sensitivity of the square-wave gratings is 1.3 times that of a sine-wave grating of the same spatial frequency, square-wave gratings with a spatial frequency greater than 0.8 cycles/degree are not perceived as different from sine-wave gratings at threshold contrast.9
Each patient was shown the eight pairs of plates in random order three times, in a room with constant, diffuse lighting with a mean luminance of 250 lux at the test plates. The patient was asked to indicate which of the two plates contained the grating. The pair of plates with the lowest contrast that the patient judged correctly three times was regarded as the contrast sensitivity threshold for that patient. The contrast sensitivity threshold for normals has been established as 0.45%; for diabetics without retinopathy, 0.49%; for diabetics with retinopathy, 0.60%; and for diabetics with maculopathy, 1.57%.

RESULTS

Thirty eyes of 29 patients were included in this prospective, randomized study. The patients were split into two groups: group A, with 18 members; and group B, with 11 members. The mean age was 62.1 years; the sex ratio, 2.5:1; M:F. The age range and the mean prelaser postlaser visual acuities are shown in the Table. The majority of the patients in both groups were of non-insulin dependent diabetes mellitus of 10 to 15 years’ duration.

After laser, the mean best-corrected visual acuity remained stable at the prelaser level (20/60) in both groups, despite changes in spatial contrast sensitivity.

Group A

In group A, the mean contrast-sensitivity threshold of the 18 eyes before PRP was 1.987 (they all fell within a range of three plates, ie, 2.24% to 0.98%). Thus, they were already compromised as compared with age-matched normals. One week after PRP, the mean threshold increased from 2.24% to 2.82%, ie, an overall difference of one plate. At 3 months, it was, as might be expected, the same as the prelaser value, 1.987. One-way analysis of variance showed that the one-plate increase in the mean prelaser threshold 1 week postlaser was statistically significant (P < .001, Fig 2).

Group B

The mean prelaser contrast-sensitivity thresholds for these 11 eyes was 2.063 (most of the eyes had thresholds ranging between two plates, ie, between 2.24% and 1.59%). The increase in the mean threshold after laser was one plate (ie, 2.82% and 2.24%). The postlaser 1- and 2-week thresholds were 4.005. The mean postlaser threshold at 3 months, as expected, was the same as the prelaser value, 2.063. One-way analysis of variance showed that the one-plate increase in the mean prelaser threshold 1 and 2 weeks postlaser was statistically significant (P < .001, Fig 3).

The prelaser color-vision-error scores were high as compared with those of age-matched controls, with a mean error score of 446.056 in group A and 502.818 in group B. In group A, these scores increased at 1 week and at 3 months postlaser, to 478.000 and 479.556, respectively. In group B, the scores were higher than the mean prelaser score at all postlaser visits. Two-way analysis of variance showed that both these increases
were significant ($P < .001$ for group A and $P < .01$ for group B, Figs 4-5).

Photostress recovery in group A was increased over the prelaser value of 50.833 seconds, with a mean value of 52.778 seconds at the 1-week and 3-month intervals. One-way analysis of variance showed this increase was significant ($P < .001$). There was no statistically significant change in the photostress recovery time in group B.

**DISCUSSION**

These results are consistent with those of previous reports in concluding that the Snellen chart may not always reveal subtle visual loss.

Previous studies of contrast-sensitivity changes in diabetic retinopathy patients undergoing PRP focused on short-term consequences and reported conflicting results. Higgins and colleagues have studied four eyes with PDR undergoing PRP. In three, they found a temporary loss of contrast sensitivity in the high spatial frequency during closely-spaced treatments, even though Snellen visual acuity remained stable and there were no signs of macular edema. They attributed this loss to the effect of intraocular scattering of the laser beam on the macula. They also suggested that the laser energy may have disrupted the foveal receptor orientation by creating traction along the retinal surface.

Canning and colleagues, using Arden plates or Vistech data, found minimal changes in contrast sensitivity in patients who underwent PRP using lasers of various wavelengths (blue-green, yellow, and dye-orange). Khosla and colleagues, using Cambridge charts, reported that the contrast thresholds of normal controls were 0.45%; of diabetics without retinopathy, 0.49%; of diabetics with retinopathy, 0.60%, and of diabetics with severe retinopathy, 1.57%.

In our study, contrast-sensitivity thresholds increased temporarily in the first and second weeks after PRP, but returned to prelaser levels at 3 months. This temporary increase occurred in both groups. Our results are somewhat similar to those reported by Higgins et al. However, in our study, contrast sensitivity was evaluated using gratings with a spatial frequency of 4 cycles/degree in 30 eyes over a period of 3 months, with PRP administered in two settings in one group. The increases in contrast sensitivity after PRP in both groups were similar in magnitude, regardless of the number and spacing of the laser treatments. In our study, the loss of contrast sensitivity in the initial postoperative period may have been due to the intraocular scattering effect of the laser beam, foveal receptor disorientation, and/or retinal edema.

The color-vision-error scores significantly increased after PRP in both groups, possibly due to photoocoagulation-induced necrosis of the photoreceptors, which are already compromised by metabolic-induced cone damage. This postlaser deterioration is known to be related to the intensity and confluence of the laser spots.

Although the mechanism of this secondary but temporary depression of spatial frequency sensitivity is not known, there are several possibilities. First, PRP might have temporarily disrupted foveal vision by directly injuring the nerve fibers subserving the foveal receptors. However, this is unlikely, since PRP was not applied temporal to the optic disc, and, therefore, should not have disturbed any of the fibers of the papillomacular bundle.

A second possibility, indirect damage, cannot easily be ruled out. Zwick et al., Robbins et al., and Zwick and Beatrice have pointed out that direct foveal exposure to low-level laser irradiation can temporarily disrupt vision in monkeys. Also, intraocular scatter
from the laser beam may have contributed to the temporary depression we observed. In addition, subclinical macular edema might have been involved, even though no edema was seen on fluorescein angiography or biomicroscopy. PRP may have disrupted foveal receptor orientation by creating traction along the retinal surface. Whether PRP of the midperipheral retina could disrupt foveal receptor orientation sufficiently to produce an effect of this magnitude or whether PRP affects retinal function proximal to the photoreceptors remains unclear.

We found no significant difference in contrast-sensitivity thresholds following PRP applied in one session as contrasted with those following two sessions. However, color-vision error scores were permanently affected. The phototestx test provided only subjective ancillary data and was not evaluated in detail.

In conclusion, we found a temporary loss of foveal contrast sensitivity following PRP, which was returned to and stabilized at the prelaser level 3 months after treatment. This temporary loss of contrast sensitivity in the initial period after PRP may have been due to the intraocular scattering effect of the laser beam, foveal receptor disorientation, and/or retinal edema. However, PRP can be carried out safely when indicated, since the deleterious effect on visual acuity and contrast sensitivity thresholds lasts no longer than 3 months.

REFERENCES