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Photostress recovery in chronic open angle glaucoma

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SUMMARY Photostress recovery time was measured in 30 eyes from 15 patients with chronic open angle glaucoma, and 30 eyes from 15 individuals of a similar age group with no ophthalmological disorder. The average recovery time in patients with glaucoma was 70·47 (SD 35·39) seconds. The average recovery time in the control population was 41·97 (SD 17·34) seconds. This difference was statistically significant ($p<0·001$). There was a small positive correlation between age and recovery time in the control population, whereas there was no correlation between age and recovery time in the glaucoma group. There was no correlation between visual acuity and recovery time for either group. There was also no correlation between intraocular pressure and recovery time for the glaucoma group. It was not possible to control for pupillary dilatation in this study. However, it has been previously demonstrated that pharmacological meiosis will not delay photostress recovery time in normal subjects. This is the first report of photostress recovery testing in patients with chronic open angle glaucoma. The results are discussed in terms of the pathophysiology of glaucoma and previous photostress studies in patients with macular disease.

Photostress recovery time (PSRT) has frequently been used as a quantitative measure of macular function. Initially described by Magder[1] as a clinically useful tool for following the progression of central serous retinopathy, photostress recovery has also been utilised in the study of the ocular toxicity of antimalarial medications,[2] ocular toxicity of birth control pills,[3] and various macular and optic nerve diseases.[4–7] Although a precise pathophysiological explanation for aberrant photostress responses has yet to be offered, it has been clearly demonstrated that optic nerve and macular disease can be differentiated on the basis of PSRT, where the retina with macular disease has an increased recovery time and that with optic nerve disease has a normal recovery time.[7]

A major criticism of photostress recovery testing has been the lack of a standardised technique in conducting the test. The test consists in measuring the visual acuity of a given eye, illuminating that eye with a high intensity light for a specific length of time—creating a central scotoma—and measuring the time required for the eye to return to baseline visual acuity once the illumination period is over. The time required to return to baseline acuity is recorded as the photostress recovery time. The photostress response is not entirely understood but has been explained as a transient state of visual insensitivity caused by bleaching of the visual pigments of the retina.[7] Return of sensitivity is dependent on resynthesis of the visual pigments, and resynthesis requires adequate perfusion of the photoreceptors and the pigment epithelium. The process is independent of optic nerve integrity.

In order to standardise the approach and simplify the implementation of photostress testing a hand held instrument was designed to measure photostress recovery time.[8] With this instrument, called a scotometer, the toxic effects of antimalarial medications on the retina, as manifested by an increased recovery time, were demonstrated.[2] This instrument has also been used to demonstrate the toxic effects on the retina of birth control pills.[3] Glaser *et al.* used an ordinary penlight for an illumination period of 10 seconds to compare recovery time in patients with macular disease and optic nerve disease.[7] They found a small increase in recovery time with increasing age in both groups and defined a normal recovery time as

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less than 50 seconds. Patients with macular disease (senile macular degeneration, submacular drusen without serous detachment, diabetic retinopathy, cystoid oedema) had an average recovery time greater than 150 seconds; eyes with optic nerve disease (optic neuritis, optic atrophy, ischaemic optic neuropathy, lymphomatous infiltrations) had recovery times of 50 seconds or less. The authors concluded that photostress recovery testing was 'a valuable adjunct in the clinical assessment of decreased vision and a sensitive discriminator between occult optic neuropathy and subtle maculopathy.'[7]

The following study presents the results of photostress recovery testing in patients with chronic open angle glaucoma and patients of a similar age group having no ophthalmological disorder.

Subjects and methods

We prospectively studied photostress recovery time in patients with chronic open angle glaucoma (COAG) being followed up at the Glaucoma Clinic of Montefiore Hospital, and patients from the General Eye Clinic with no known ophthalmological disorder. The ophthalmoscope was recharged before each testing period for at least eight hours. The photostress test was performed before any pharmacological preparation for routine ocular examination. Photostress testing was conducted as follows:

(1) Best corrected visual acuity was determined for each eye with Snellen test letters at a distance of 6 m (20 feet). All subsequent measurements were obtained with the same chart, under the same lighting conditions, and by the same investigator. All eyes were undilated. Because of the limited number of large test letters on the Snellen chart and the possibility of consequent inaccuracy of measurements, the data were considered invalid for eyes with visual acuity less than 6/30 (20/100) and were eliminated from the analysis.

(2) The right eye was tested first, with the left eye covered by an opaque shield. The patient was instructed to look directly into the halogen bulb attachment of a Welch Allyn halogen bulb ophthalmoscope, which was held 2 to 3 cm from the eye for an illumination period of 10 seconds.

(3) Immediately after the light was removed the patient was instructed to read the Snellen chart, starting at the first visible line and continuing to increasingly smaller lines as they became clear. The recovery time was recorded as the time (in seconds) required for the patient to read correctly three letters of the line just above that recorded as best visual acuity. Recovery time was recorded to a maximum of 150 seconds.

(4) The same procedure was then conducted for the left eye, with the right eye occluded.

Intraocular pressure was measured for the COAG group with a non-contact air tonometer. After being photostress tested each patient received a routine ophthalmological examination from an ophthalmologist who was not aware of the patient's photostress scores. Each patient's chart was later reviewed for data on visual field defects, systemic disease, medication history, and previous ophthalmological procedures and findings. Each control patient had a normal anterior and posterior eye.

Results

Only data from patients who were capable of having both eyes tested were included in the data analysis. The COAG group consisted of patients with a diagnosis of glaucoma in both eyes. The photostress recovery time for each eye of a given patient was averaged to provide a single photostress score for that patient.

Table 1 summarises the clinical data for patients in

Table 1 *Clinical data for patients with glaucoma*

Age (yr), sex	*Eye*	*Visual acuity*	*Intraocular pressure (mmHg)*	*PSRT* (s)*	*Recovery score† (s)*
73, F	OD	20/70	16	150	150
	OS	20/50	14	150	
78, F	OD	20/50	12	42	62
	OS	20/40	15	82	
44, F	OD	20/25	14	28	74
	OS	20/25	13	120	
78, F	OD	20/40	18	32	32·5
	OS	20/30	20	33	
72, M	OD	20/40	9	35	66
	OS	20/30	11	97	
77, F	OD	20/30	15	19	72
	OS	20/25	15	125	
77, M	OD	20/40	21	48	53
	OS	20/30	19	58	
71, M	OD	20/70	22	150	111
	OS	20/50	30	72	
74, M	OD	20/30	15	42	42
	OS	20/30	18	42	
51, M	OD	20/25	16	43	32
	OS	20/25	19	21	
76, F	OD	20/40	21	88	59
	OS	20/30	15	30	
68, F	OD	20/30	14	22	39
	OS	20/30	14	56	
63, M	OD	20/50	29	81	115·5
	OS	20/50	25	150	
56, F	OD	20/25	33	50	41
	OS	20/25	21	32	
66, M	OD	20/25	NA‡	73	108
	OS	20/25	NA	143	

*Photostress recovery time.
†Average PSRT.
‡Not available.

Table 2 *Clinical data for control patients*

Age (yr), sex	*Eye*	*Visual acuity*	*PSRT* (s)*	*Recovery score† (s)*
67, M	OD	20/70	20	34
	OS	20/50	48	
60, M	OD	20/30	45	34
	OS	20/30	23	
68, F	OD	20/30	35	33·5
	OS	20/30	32	
79, F	OD	20/30	79	96·5
	OS	20/30	114	
67, F	OD	20/25	31	34
	OS	20/25	37	
60, M	OD	20/25	20	33
	OS	20/30	46	
51, M	OD	20/25	22	21·5
	OS	20/20	21	
56, M	OD	20/25	45	41
	OS	20/25	37	
60, F	OD	20/25	47	36·5
	OS	20/25	26	
51, M	OD	20/25	57	40·5
	OS	20/20	24	
50, F	OD	20/30	53	40
	OS	20/30	27	
48, F	OD	20/40	25	30
	OS	20/25	35	
66, F	OD	20/30	25	51·5
	OS	20/30	78	
51, F	OD	20/25	59	54
	OS	20/25	49	
50, F	OD	20/30	55	49·5
	OS	20/25	44	

*Photostress recovery time.
†Average PSRT.

the COAG group. The average recovery score for patients in this group was 70·47 seconds, SD 35·39 seconds. The average age of patients in this group was 68·27 years, SD 10·52 years. The correlation between recovery score and age did not reach statistical significance in this group ($r=0·32$, $p>0·05$). The average intraocular pressure was 17·75 (SD 5·01) mmHg. The correlation between intraocular pressure and recovery score was not statistically significant. Likewise the correlation between visual acuity and recovery score was not statistically significant.

Table 2 summarises the clinical data for patients in the control group. The average photostress recovery score for patients in the control group was 41·97 seconds, SD 17·34 seconds. The difference in recovery time between the control group and the glaucoma group was significant ($p<0·001$). Fig. 1 illustrates the distribution of photostress recovery scores.

The average age of patients in the control group was 58·93 years, SD 9·04 years. The difference in age between the COAG group and control group was not statistically significant. There was a weak correlation

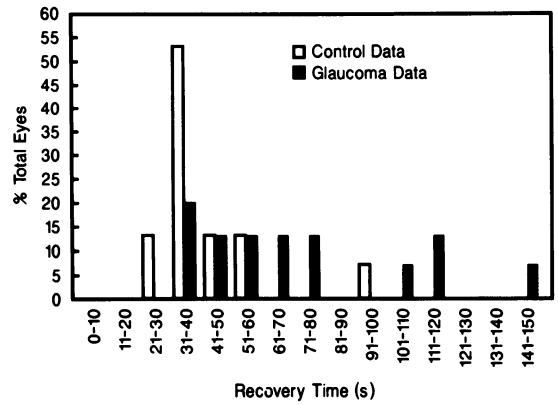

Fig. 1 *Distribution of photostress recovery scores from 15 patients with chronic open angle glaucoma and 15 patients with normal eyes.*

between recovery score and age in the control group ($r=0·41$, $p<0·05$). Fig. 2 illustrates photostress scores as a function of age. Repeat photostress testing on subsequent clinic visits for a subset of patients showed the test results were reproducible. Table 3 summarises the photostress results for glaucomatous and normal patients.

Discussion

Magder described photostress evaluation as being specific for diagnosing oedema of the macular area.[1] He proposed that in central serous retinopathy, where there is a separation between the pigment epithelium and neuroepithelium, normal chemical reactions between these layers cannot occur, resulting in a delayed responsivity to light in the photostress test. Severin *et al.*[9] explained the photostress response in terms of bleaching and resynthesis of the visual pigments. They proposed that any pathological state which interferes with the ability of the photoreceptors to resynthesise photopigment, either by interrupting enzymatic steps in the biochemical cycle or by altering anatomical relationships, will effect the performance reserve of the retina, as shown by the photostress recovery test.

Table 3 *Summary of photostress test results*

Group	*Patient no.*	*Mean age (yr)*	*Mean recovery score (s)*	*Correlation (r) of age vs recovery score*
Normal	15	58·93±9·04	41·97±17·34	0·41*
Glaucoma	15	68·27±10·52	70·47±35·39	0·32†

*Statistically significantly at $p<0·05$.
†Not statistically significant.
±=Standard deviation.

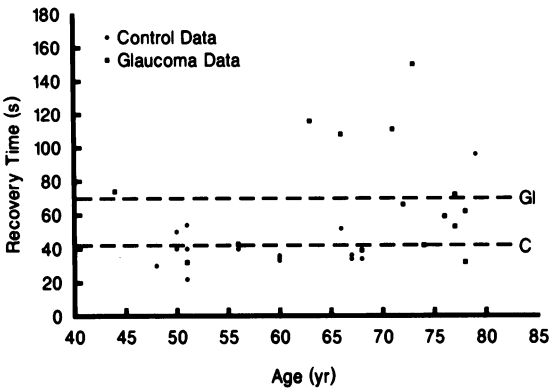

Fig. 2 *Photostress recovery score versus age in 15 patients with chronic open angle glaucoma and 15 patients with normal eyes.*

In cases where the disease state has already caused destruction of the photoreceptors the signs may be obvious, manifesting as a change in visual acuity, colour vision, visual field, or an abnormal retina. However, when the disease is incipient or its effects subtle, there may be no noticeable change in vision or deterioration on routine examination. In these cases aberrant photostress recovery times may be the first sign of pathological activity.

In the present study we found a statistically significant increase in photostress recovery time in patients with chronic open angle glaucoma as compared with normal patients of a similar age group with no ophthalmological disorder. There was no significant correlation between intraocular pressure and recovery time. The majority of the glaucoma patients had their pressure under control at the time of the study. However, when one examines the distribution of recovery times in Fig. 1, a broader range of scores for the glaucoma group is observed. This greater range of scores may indicate that different pathological stages were being tested. The increased scores of the glaucoma patients cannot be explained by the age of this population, as the difference in age between the glaucoma group and control group was not statistically significant.

It was not possible to correlate photostress response with medical therapy for glaucoma in this study. There were not enough patients on a single drug regimen to allow unbiased conclusions to be drawn. Several investigators have addressed the question of the effect of pupillary dilatation on photostress recovery.[6 10-13] These authors have consistently arrived at the conclusion that pupillary dilatation has little or no effect on recovery time. The explanation for this observation is that the iris of the meiotic pupil reduces the luminous intensity only in the extramacular region, whereas the photostress stimulus tests the physiological integrity of the macula itself.[11] Ulla *et al.* recently considered the effect of pharmacological alteration in pupillary size on macular recovery time.[12] They reported that pharmacological mydriasis had no effect, whereas pharmacological meiosis *reduced* the recovery period in a group of normal patients. Certainly this point deserves further investigation given the results of the present study.

All of the patients in the glaucoma group had some form of visual field defect. Only one previous study of photostress recovery has considered the issue of visual fields.[13] Severin *et al.*, using an elaborate apparatus for retinal dazzling, examined 14 cases of maculopathy of various aetiologies, and recorded each patient's visual field with his or her recovery time.[13] Their data are unfortunately inconclusive in terms of relating aberrant recovery times to field defects. However, they did observe markedly increased recovery time both in patients with a central scotoma (secondary to central serous retinopathy) and in a patient with a ring scotoma (secondary to chloroquine maculopathy). They did not test any patients with glaucoma.

It has been suggested that increased photostress recovery times may result from anatomical derangement in the area of the macula or decreased perfusion of the photoreceptors.[9] In previous studies of photostress recovery, increased recovery scores have been clearly related to pathological separation of cell layers of the retina. It seems reasonable to assert that any disease that causes damage to the photoreceptors, pigment epithelium, Bruch's membrane, choriocapillaris, or vascular supply to the macula may alter the photostress recovery time. Certainly further investigation is necessary to explain the pathophysiological basis for delayed photostress recovery in patients with glaucoma.

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