Xenon arc panretinal photocoagulation for central retinal vein occlusion: a randomised prospective study*

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SUMMARY Thirty-three patients with central retinal vein occlusion were evaluated for the effects of scatter xenon arc panretinal photocoagulation. Thirty-four eyes (2 eyes of 1 patient) were randomly divided into a treatment (15 eyes) and a nontreatment group (19 eyes). The average follow-up per eye has been 29 months. The visual prognosis was not significantly better in either group. There was no difference in the development of fundus neovascularisation between groups. None of the patients in the treated group developed rubeosis or neovascular glaucoma. Two patients in the nontreatment group initially had rubeosis and 3 developed it after entry. All 5 involved eyes developed neovascular glaucoma. A significantly greater amount of central retinal capillary drop-out was present in the untreated eyes that developed rubeosis than those untreated eyes which did not. The eyes in the treated group had a similar range of capillary drop-out area, but none of these eyes developed rubeosis or neovascular glaucoma. Panretinal photocoagulation prevented the development of rubeosis and neovascular glaucoma in susceptible eyes.

There has been little change in the prognosis for visual acuity and survival of the eye after central retinal vein occlusion since the original clinical description of this condition by Liebreich (1855). Poor visual acuity usually results from leakage or ischaemia of the macular venules. This process may eventually result in macular fibrosis and scar formation or retinal atrophy (Raitta, 1965; Hayreh, 1971).

The therapy of central retinal vein occlusion is controversial. Numerous clinical regimens have been in vogue, each with varying degrees of reported success. None has been satisfactorily proved to be advantageous. Therapeutic trials have included the use of the following: potassium iodide and pilocarpine (Braendstrup, 1950), irradiation (Gradle, 1937; Hessberg, 1944; Guyton and Reese, 1948), anti-coagulants (Holmin and Ploman, 1938; Klien and Olwin, 1956; Vannas and Orma, 1957), fibrinolytic

agents (Dolenek *et al.*, 1968; Kohner *et al.*, 1976), dextran (François and Verbraeken, 1974), corticosteroids (Van Loon, 1961; Mehlan *et al.*, 1974), and carbogen (95% O₂ and 5% CO₂) (Sedney, 1976).

Definite therapeutic effects have been achieved with the use of photocoagulation in diabetic eye disease (Diabetic Retinopathy Study Group, 1976). Promising results have also been reported with the use of photocoagulation in branch vein occlusion and central retinal vein occlusion (Krill *et al.*, 1971; Campbell and Wise, 1973; Gitter *et al.*, 1975). The most significant effect noted thus far in retrospective nonrandomised studies of panretinal photocoagulation for central retinal vein occlusion has been the prevention or elimination of rubeosis iridis and subsequent neovascular glaucoma (Krill *et al.*, 1971; François *et al.*, 1974; Freyler and Nichorlis, 1974; Theodossiadis *et al.*, 1974; Wetzig and Thatcher, 1974; Zweng *et al.*, 1974; Oosterhuis and Sedney, 1975; Callahan and Hilton, 1974; Fetkenhour *et al.*, 1975; Little *et al.*, 1976; Sedney, 1976).

In 1976 we reported the preliminary results of the first prospective controlled study to evaluate the efficacy of xenon arc panretinal photocoagulation in the treatment of central retinal vein occlusion

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(May *et al.*, 1976). The first 20 patients in the study were evaluated after an average follow-up of 18 months. There was no difference in the visual prognosis between groups. In the untreated group 4 patients developed rubeosis that progressed to neovascular glaucoma (NVG), but none of the patients in the treated group developed rubeosis or NVG. Photocoagulation had no significant effect on the development of disc or retinal neovascularisation. Similar results were reported by Laatikainen *et al.* (1977) in a more recent study.

This is the second report of the prospective randomised study of xenon arc panretinal photocoagulation for central retinal vein occlusion from the University of Illinois Eye and Ear Infirmary. The study was begun in January 1973 and continued through March 1978. A total of 33 patients were included in the study. The average follow-up time after occlusion was 28.5 months with a range of 5 to 64 months.

Patients and methods

Patients older than 40 years of age who had clinical central retinal vein occlusion and vision less than 6/12 (20/40) in the involved eye were referred to us from clinic and private patient populations of the University of Illinois Eye and Ear Infirmary. Patients with systemic or ocular disease such as diabetes mellitus, which could greatly influence the ocular prognosis, were excluded from the study. Other patients excluded were those whose involved eye had media with insufficient clarity to permit photocoagulation; those who were or had been treated with anticoagulants, corticosteroids, or vasodilators after the time of the central vein occlusion; and patients with an occlusion prior to evaluation for a duration of more than 1 year. Two patients entered in the study did have clinically evident central retinal vein occlusions for a longer time (18 and 24 months), but they had been followed up in our ophthalmology clinic.

All examination and treatment procedures were explained to those patients eligible for the study. The protocol that was adhered to had been approved by the University of Illinois Medical Center Ethics Committee, and the study was monitored by 3 assigned ophthalmologists who were not participating in the study.

A thorough medical history was recorded and each patient was referred for systemic evaluation. The following laboratory tests were performed: a complete blood cell count, urine analysis, 3-hour glucose tolerance test, plasma thromboplastin, partial thromboplastin time, haemoglobin electrophoresis, serum protein electrophoresis, electro-

cardiogram, chest roentgenogram, red blood cell sedimentation rate, and serum cholesterol and triglyceride levels.

During the initial ophthalmological evaluation, the following examinations were performed: visual acuity (Snellen chart, refracted) slit lamp examination of the anterior segment, gonioscopy and fundus biomicroscopy, applanation tonometry, visual fields (Goldmann), fundus fluorescein angiography, and stereoscopic colour photography of the fundus. Fundus fluorescein angiography was repeated in 3 months and then annually. All other procedures were repeated 1 month after the initial evaluation and then every 3 months thereafter. Iris angiography was performed on all patients during the last 18 months of the study if there was a clinical suspicion of rubeosis.

Each patient was randomly assigned to a treatment or nontreatment group. The treated group underwent photocoagulation as outpatients as soon as possible after entry.

Dilatation consisted of 1 drop of 1% tropicamide and 1 drop of 1% cyclopentolate hydrochloride on the eye to be treated. This was repeated again in 10 minutes. Topical 0.5% tetracaine (amethocaine) and a retrobulbar injection of 2 ml of 2% lidocaine (lignocaine) with epinephrine (1:100 000) and hyaluronidase were given prior to treatment. Panretinal photocoagulation was performed through a widely dilated pupil with the xenon arc photocoagulator (model 514 Optical Radiation) using a Goldman three-mirror lens and a Fankhauser slit-lamp delivery system. A 360° scatter panretinal photocoagulation was administered in 1 or 2 sessions. The burns were kept at least 1 disc diameter from the disc and macula, and no scattered burns were placed within the temporal arcades. Central burns (within 5 disc diameters of the disc) were 3°, and those further peripherally were 4.5°. The number of burns and the energy delivery in joules were recorded at each treatment session. The burns were placed at least 3° apart. Focal photocoagulation was applied directly to neovascularisation on the retina (NVE). Neovascularisation from the disc (NVD) was not treated directly. A polymixin-B-bacitracin-neomycin ointment and an eye patch were applied to the eye after treatment. The patient was examined the next day, then every week for 1 month, and then at 3-month intervals.

The area of initial retinal capillary obliteration was quantitated for each eye entered in the study. The measurements were obtained retrospectively from fundus fluorescein angiograms and photographs (central 30° of the fundus with the macula at the centre) taken at the time of initial evaluation. A midvenous phase angiogram was projected on a

screen with a colour slide of the same field shown beside it. This comparison was done to eliminate the confusion of those areas of retina obscured by haemorrhage with those having actual retinal capillary obliteration. The dimensions of the disc and the area of retinal capillary obliteration were measured on the projected fluorescein. Areas were calculated by standard geometric means in absolute terms (i.e., cm^2).

The absolute areas measured were relative to the projection magnification and as such have little significance when compared among patients. For this reason the area of retinal capillary obliteration was calculated in terms of disc areas for each eye. This was derived by dividing the area of capillary obliteration (cm^2) by the area of the disc (cm^2) of the same eye:

$$\frac{\text{area of retinal capillary obliteration (cm}^2\text{)}}{\text{area of disc (cm}^2\text{)}} = \frac{\text{area of retinal capillary obliteration}}{\text{(disc areas)}}$$

This information is presented in Table 1.

Results

Thirty-four eyes of 33 patients were entered into the study (both eyes of 1 patient). Nineteen eyes were randomly assigned to the nontreated group and 15 to the treated group. The age range was 50 to 81 years (64 average) in the nontreated group and 41 to 88 years (63 average) in the treated group.

There was a very high incidence of glaucoma among our patients. Thirteen patients (39%) had glaucoma, 10 of whom had not been previously diagnosed for the condition. Seventeen (52%) were systemically hypertensive. Of these, 3 were previously undiagnosed and 1 was a known hypertensive who had declined to take medication. These and other basic entrance data are presented in Table 1.

The average follow-up time after the central retinal vein occlusion was 28.6 months per eye (a range of 8 to 56 months) in the nontreated group and 28.5 months (a range of 5 to 64 months) in the treated group. Two patients in the treated group had an insufficient follow-up time of less than 6 months. The time period between the onset of symptoms and entry into the study was an average of 4.0 months for the nontreated group and 3.0 months for the treated group. Distribution data for the patients are summarised in Table 2. The follow-up times for each patient are listed in Table 3.

All patients had clinical evidence of macular oedema on entry into the study. The average duration of the oedema, as shown by fluorescein angio-

graphy, was 17.5 months in the nontreated group and 21.2 in the treated group.

Only 1 patient did not have cystoid macular oedema (CME) at some time during the course of the study. The average duration of the CME in those eyes in which it resolved was 13.0 months for the nontreated group (12 eyes) and 11.1 months for the treated group (9 eyes). In 2 eyes of the nontreated group and in 3 eyes of the treated group the CME persisted for the duration of the study. The duration of CME could not be adequately followed up in 3 eyes of each group: 3 eyes of the untreated group developed cloudy media all secondary to NVG; 1 eye of the treated group had insufficient follow-up; 1 eye developed a total nonrepairable retinal detachment; and the macula of 1 eye was inadvertently photocoagulated.

Five patients in each group developed neovascularisation from the disc (NVD). The average onset after vein occlusion was 12 months for eyes in the nontreated and 4.2 months for the treated group. The NVD regressed after 8 months' duration in one nontreated eye and after an average of 3.7 months in 3 eyes of the treated group. The NVD persisted for the duration of the study in 4 eyes of the nontreated and 2 eyes of the treated group. Shunt vessels developed on the disc in 2 eyes of the nontreated group and in 4 eyes of the treated group. There did not appear to be a correlation among the development and persistence of macular oedema, CME, or NVD.

One patient in each group developed neovascularisation on the retina (NVE). Patient 31 in the nontreated group was lost to follow-up after the NVE was found. Patient 29 in the treated group had NVE present on entry to the study and was focally treated. The NVE did not resolve until it was focally retreated again 3 months later. The neovascularisation and macular oedema data for the patients are summarised in Table 4.

Two patients in the nontreated group had rubeosis initially. One of the 2 also had clinical evidence of NVE. Three additional patients in this group developed rubeosis after entry into the study. Four of the patients had clinical evidence of neovascular glaucoma (NVG) when the rubeosis was first noted, the other patient developed NVG within 4 weeks. The average time when NVG was first noted was 13.2 weeks or 93 days after the onset of central vein occlusions. None of the patients in the treated group developed rubeosis or NVG.

The nontreated patients who developed NVG had retinal capillary drop-out areas in the range of 16.0 to 22.0 (19.6 average) disc areas. Other eyes in the nontreated group had capillary drop-out areas in the range of 0 to 9 (2.1 average) disc areas. The

Table 1 Entrance data

Patient	Age (yr)	Race	Sex	Involved eye	Primary open-angle glaucoma	Hypertension	Other medical problems*	Group	Area of capillary obliteration (disc areas)
1	72	B	M	OS	OU previously unknown	None		Nontreatment	18.8
2	66	B	M	OS	None	10 yr of therapy		Nontreatment	16.0
3	54	SA	M	OS	OU previously unknown	None		Treated	0
4	66	W	M	OS	None	5 yr of therapy		Treated	4.7
5	81	B	M	OS	OU previously unknown	Previously unknown	Positive FTA-ABS previously untreated	Treated	22.7
6	65	B	F	OD	OU previously unknown	5 yr of therapy	Sickle cell trait	Treated	0
7	59	W	M	OD	OU 9 mo of therapy	2 mo of therapy		Nontreatment	9.0
8	69	W	M	OS	None	None		Treated	5.1
9	61	B	M	OS	None	10 yr of therapy		Treated	4.2
10	73	B	F	OS	OU previously unknown	None		Nontreatment	21.4
11	48	B	F	OD	None	2 yr of therapy		Nontreatment	0
12	70	W	M	OS	None	Previously unknown		Nontreatment	0
13	67	W	M	OS	OU previously unknown	None		Treated	0
14	77	B	M	OS	None	None		Nontreatment	0
15	48	SA	F	OS	None	Previously unknown		Treated	15.2
16	88	B	F	OD	None	12 yr of therapy		Nontreatment	1.1
17	52	W	M	OS	None	None		Nontreatment	2.2
18†	64	SA	M	OS	None	None	Diabetes mellitus known for 7 mo	Treated	0
19	80	W	M	OD	OU previously unknown	None		Nontreatment	22.0
20	65	B	F	OD	None	8 yr of therapy		Nontreatment	0
21	73	W	M	OD	None	5 yr of therapy		Nontreatment	1.6
22	66	B	M	OS	None	10 yr of therapy		Nontreatment	0
23	53	W	M	OS	None	None		Treated	1.4
24	67	B	M	OS	OU 6 yr of therapy	None		Treated	12.8
25‡	61	SA	F	OU	OU previously unknown	None	Elevated serum cholesterol and triglycerides	OD nontreatment OS treated	4.6 0
26	76	B	F	OS	OU 5 yr of therapy	None		Nontreatment	0
27	41	B	M	OS	OU previously unknown	None		Nontreatment	19.8
28	76	B	F	OD	None	None		Treated	17.1
29	73	W	M	OS	OU previously unknown	2 mo of therapy		Treated	4.4
30	64	W	F	OS	None	5 yr of therapy		Nontreatment	2.8
31	77	B	F	OD	None	18 yr of therapy		Nontreatment	8.5
32	53	B	M	OS	None	Known for several years but previously untreated	MI & CHF Jan/77	Nontreatment	0
33	50	W	M	OD	None	None		Treated	0

*FTA-ABS indicates fluorescent treponemal antibody absorption test; MI, myocardial infarction; and CHF, congestive heart failure.

†The patient denied diabetes mellitus on entry into the study. After an abnormal glucose tolerance test, the patient admitted taking insulin for the past 7 months. There was no evidence of diabetic eye disease OD during the course of the study.

‡Both eyes were entered into the study.

Table 2 Summary of data on patients

	Nontreatment	Treated
No. of eyes*	19 11 OS 8 OD	15 12 OS 3 OD
Glaucoma†	7 patients	7 patients
Systemic hypertension	11 patients	6 patients
Average time elapsed before entry into study	4.0 mo	3.0 mo
Average follow-up postocclusion	28.6 mo	28.5 mo
Insufficient follow-up	0	2 patients

*Thirty-three patients were admitted to the study. Patient 25 had both eyes entered into the study.

†Thirteen patients admitted to the study had bilateral primary open-angle glaucoma, including patient 25 (OD was entered into the nontreatment group and OS into the treated group).

average capillary drop-out for the entire nontreated group was 6.7 disc areas. The range was 0 to 22.7 (5.8 average) disc areas in the treated group (see Table 1).

Analysis of the distribution of the areas of retinal capillary drop-out at the initial evaluation showed a totally random distribution of eyes to both groups. By *t* test analysis there was a significance of $P=0.485$. This means that on the basis of the distribution of areas of capillary drop-out all eyes had an equal chance of being assigned to either group.

Five eyes in the untreated group developed NVG; none in the treated group developed it. This suggests that the panretinal photocoagulation prevented the development of glaucoma in the treated group. This is supported by Fisher's exact test at the 0.031 significance level.

Fifteen (52%) of the eyes entered into the study (excluding eyes developing NVG) had initial intraocular pressures lower than in the noninvolved eye. The initial pressures averaged 5 mmHg (range of 3 to 12 mmHg) lower and did not return to equal in the 2 eyes until an average of 6.1 months after occlusion. Three involved eyes of 3 patients had pressures of 3 mmHg or greater than in the non-involved eye. For the remainder of the patients the difference between the intraocular pressures of both eyes was within 2 mmHg.

Each of the 15 treated eyes in the group received an average of 208 burns with an average energy delivery of 173 joules. The range was from 120 to 335 burns per eye with a delivered energy range of 70 to 340 joules. Patients receiving the less dense photocoagulation patterns (140 burns or less) did not appear to differ in their ultimate prognosis. A high energy level was required for those eyes with greater amounts of nuclear sclerotic lens changes. A treatment summary for each patient is outlined in Table 5.

Three patients had peripheral choroidal effusions

Table 3 Patient follow-up time

Patients	Involved eye	Onset of symptoms	Time elapsed before entering study	Postocclusion follow-up time
Nontreated				
1	OS	May/74	4 wk	8 mo
2	OS	Jan/74	4 wk	40 mo
7	OD	May/72	18 mo*	56 mo
10	OS	Jan/74	3 wk	40 mo
11	OD	May/73	24 mo*	54 mo
12	OS	Feb/76	3 mo	23 mo
14	OS	April/77	1 wk	8 mo
16	OD**	Feb/73	6 mo	17 mo
	OS	April/72	Not included in study	
17	OS	April/73	4 mo	51 mo
19	OD	March/73	3 mo	31 mo
20	OD	July/75	1 day	30 mo
21	OD	April/73	3 mo	56 mo
22	OS	Nov/75	5 days	22 mo
25	OD†	April/76	13 days	21 mo
26	OS	Feb/74	3 mo	21 mo
27	OS	April/76	2 days	14 mo
30	OS	Aug/76	2 mo	16 mo
31	OD	March/74	6 mo	20 mo
32	OS	Sept/76	1 wk	15 mo
	OD**	March/73	Not included in study	
Treated				
3	OS	May/73	6 wk	27 mo
4	OS	Nov/72	8 mo	64 mo
5	OS	Feb/76	1 mo	25 mo
6	OD	May/74	10 days	42 mo
8	OS	Oct/76	4 days	16 mo
	OD**	Sept/71	Not included in study	
9	OS	Oct/75	3 mo	28 mo
13	OS	April/75	3 wk	34 mo
15	OS	Nov/76	3 mo	12 mo
18	OS	Jan/77	2 wk	11 mo
23	OS	May/73	7 mo	8 mo‡
24	OS	June/73	4 mo	55 mo
25	OS††	May/76	10 days after entering study	20 mo
28	OD	June/77	7 wk	5 mo
29	OS	Jan/72	12 mo	59 mo
33	OD	June/74	2 wk	22 mo

*These patients had been followed up in the general eye clinic from within 3 months of their occlusions.

†OS entered in treated group.

‡Lost to follow-up one week after treatment.

††OD entered in nontreated group.

**The central retinal vein occlusions in these eyes had occurred too long previously to be included in the study; the other eye of each of these patients was included in the study.

Table 4 Neovascularisation and macular oedema*

Patient	Onset	Duration	Cystoid macular oedema		NVD		NVE		Other
			Onset	Duration	Onset	Duration	Onset	Duration	
Nontreated									
1	I	DS (8 mo)	I	DS (8 mo)	None		None		Rubeosis on entry (4 wk) NVG at 8 wk
2	I	1 yr	I	12 mo	None		None		Rubeosis at 24 wk with NVG
7	I	18 mo	I	18 mo	1 mo	DS (56 mo)	None		Minimal NVD persisted; shunt vessels on disc
10	I	UE	I	UE	UE		UE		Rubeosis at 12 wk with NVG
11	I	2 yr	I	12 mo	12 mo	DS (54 mo)	None		Minimal NVD persisted
12	I	23 mo	I	12 mo	None		None		
14	I	DS (8 mo)	I	DS (8 mo)	None		None		
17	I	17 mo	I	17 mo	6 mo	14 mo	None		
19	I	UE	12 wk	UE	UE		UE		Rubeosis at 12 wk with NVG
20	I	6 mo	I	2 mo	None		None		
21	I	DS (56 mo)	I	12 mo	None		None		
22	I	DS (22 mo)	2 mo	12 mo	None		None		
25	I	14 mo	I	4 mo	None		None		
26	I	14 mo	I	6 mo	21 mo	DS (21 mo)	None		Lost to follow-up after NVD noted
27	I	UE	I	UE	UE		UE		Rubeosis and NVE at 10 wk
30	I	DS (16 mo)	I	6 wk	None		None		
31	I	DS (20 mo)	I	12 mo	20 mo		20 mo (did not return)		Total superior vein occlu- sion of other eye (OS)
32	I	5 mo	None		None		None		Inferior temporal branch vein occlusion in the same eye 20 mo before; shunt vessels on disc at time of CRVO
Treated									
3	I	DS (27 mo)	I	6 mo	12 mo	DS (27 mo)	None		NVD persisted a 1/2 disc diameter
4	I	36 mo	I	19 mo	None		None		Macular drusen OU; shunt vessels on disc
5	I	DS (24 mo)	I	10 mo	None		None		Tortuous shunt vessels on disc; total superior vein occlusion of other eye (OD)
6	I	DS (42 mo)	10 wk	DS (42 mo)	5 mo	DS (42 mo)	None		Minimal NVD persisted; tortuous shunt vessels on disc
8	I	DS (16 mo)	I	DS (16 mo)	None		None		
9	I	DS (28 mo)	I	10 mo	None		None		Familial drusen OU; tortuous shunt vessels on disc
13	I	NA	2 mo	NA	NA		NA		Total retinal detachment following photocoagula- tion
15	I	DS (12 mo)	I	5 mo	1 mo 7 mo		None		Minimal shunt vessels remained on disc
18	I	DS (11 mo)	I	4 mo	1 mo 4 mo		None		
23	I	Insufficient follow-up	I	Insufficient follow-up	Insufficient follow-up		Insufficient follow-up		Macular hole present
24	I	30 mo	I	27 mo	None		None		Tortuous shunt vessels on disc
25†	I	10 mo	I	4 mo	2 mo 4 mo		None		
28	I	DS (5 mo)	I	DS (5 mo)	None		None		
29	I	14 mo (macula dry 2 mo post- treatment)	I	15 mo	None		I (treated again 3 mo later and resolved)		
33	I	NA	I	NA	None		None		Macula accidentally photocoagulated

*I indicates present on entry into study; DS, present for duration of time followed up in study; UE, unable to evaluate due to ocular media haze; NA, not applicable; NVD, neovascularisation on the disc; NVE, neovascularisation on the retina; and NVG, neovascular glaucoma.

†Both eyes were entered into study.

Table 5 *Xenon arc photocoagulation group*

Patient	Time treated postocclusion	No. of treatment sessions	Total burns	Energy delivered (joules)	Treatment problems and complications*
3	7 wk	1	139	105	Moderate NS
4	8 mo	1	335	160	Moderate NS
5	7 mo	1	140	220	Minimal 360° choroidal effusion; resolved within 2 wk
6	11 days	1	202	100	None
8	1 mo	2	308	340	Early NS; minimal 360° choroidal effusion resolved in 4 wk; macular haemorrhage 4 wk after treatment
9	8 mo	1	249	210	Early NS; iris burns
13	3 mo	3	231	295	Moderate NS; unrepairable retinal detachment 2 days after final treatment
15	4 mo	1	120	70	None
18	6 wk	2	240	176	A 60° schisis cavity was walled off with photocoagulation
23	7 mo	1	212	220	Intense retinal burns
24	4 mo	1	269	110	None
25	3 wk	1	138	140	Early NS; moderate 360° choroidal effusion resolved in 6 wk
28	7 wk	1	139	150	Moderate NS
29	4 wk	1	225	200	Moderate NS; iris burns
33	2 wk	1	173	95	Macula photocoagulated

*NS indicates nuclear sclerotic lens changes.

after photocoagulation, but these resolved without problems. Two eyes received iris burns during photocoagulation, and the macula of 1 eye was inadvertently photocoagulated. The eye of patient 8 developed a dense macular haemorrhage within 4 weeks after photocoagulation. The eye of patient 13 developed a nonrepairable tractional retinal detachment secondary to massive vitreous retraction within 2 days of the final treatment session. These eyes were treated heavily in 2 and 3 sessions, respectively. A summary of treatment data appears in Table 5.

Vision decreased in 8 eyes of the nontreated group, remained the same in 6 eyes, and improved in 5 eyes. The vision decreased in 7 of the eyes of the treated group, remained the same in 5, and improved in 3. There was insufficient follow-up of 2 patients in the treated group to evaluate visual change. The visual acuity changes for each patient are listed in Table 6.

Discussion

Focal and panretinal photocoagulation are beneficial in the treatment of some ocular vascular problems encountered with diabetes mellitus, sickle

cell disease, and branch vein occlusion. It is now believed that the destruction of hypoxic retinal tissue by photocoagulation lessens the neovascular stimulus present in the eye (Krill *et al.*, 1971; Cleasby *et al.*, 1974). By decreasing the amount of tissue that is perfused, the remaining tissue may receive a more adequate supply of oxygenated blood. This ablation of retina also may decrease the retinal vascular load and decrease macular leakage and oedema (Campbell and Wise, 1973; Theodossiadis *et al.*, 1974). Also, the destruction of the pigment epithelium may promote the drainage of fluid into the choroidal circulation (Peyman *et al.*, 1971).

The 2 most significant factors predisposing our patients to ocular vein occlusion were primary open-angle glaucoma and systemic hypertension. Most of the patients (77%) with primary open-angle glaucoma were unaware of their problem, as were 18% of the systemically hypertensive patients.

Our patients' eyes were at a very high risk for multiple occlusive problems. Three of the patients had had a previous central retinal vein occlusion in their opposite eye which was not eligible for the study because of the duration of the occlusion. One of the patients had a branch vein occlusion pre-

Table 6 *Change in visual acuity*

<i>Patient</i>	<i>Eye</i>	<i>Initial vision*</i>	<i>Final vision</i>	<i>Reason for final vision†</i>
<i>Nontreated</i>				
1	OS	LP	NLP	NVG
2	OS	6/21 (20/70)	NLP	NVG
7	OD	6/120 (20/400)	6/120 (20/400)	Pigment epithelial defect of macula
10	OS	1/100 (2/200)	NLP	NVG
11	OS	HM	HM	Macular scar
12	OS	6/60 (20/200)	1/40 (5/200)	Persistent macular oedema
14	OS	6/120 (20/400)	6/120 (20/400)	Persistent cystoid macular oedema
16	OD	1/100 (2/200)	1/100 (2/200)	Preretinal fibrosis
17	OS	6/120 (20/400)	1/40 (5/200)	Pigment epithelial defect of macula and preretinal fibrosis
19	OD	HM	LP	Mature cataract and NVG
20	OD	6/120 (20/400)	1/15 (20/50)	Disruption of macular pigment epithelium
21	OD	HM	HM	Macular scar
22	OS	1/40 (5/200)	1/40 (5/200)	Persistent macular oedema
25	OD	6/30 (20/100)	6/120 (20/400)	Macular scar
26	OS	1/66 (3/200)	6/120 (20/400)	Persistent macular oedema
27	OS	HM	NLP	NVG
30	OS	1/66 (3/200)	6/120 (20/400)	Persistent macular oedema
31	OD	LP	60/90 (20/300)	Persistent macular oedema
32	OS	6/15 (20/50)	6/9 (20/30)	Disruption of the macular pigment epithelium
<i>Treated</i>				
3	OS	6/150 (8/200)	NLP	Questionable final vision; secondary gain suspected
4	OS	1/66 (3/200)	HM	Macular atrophy
5	OS	1/100 (2/200)	1/100 (2/200)	Cataract and persistent macular oedema
6	OD	6/120 (20/400)	6/90 (20/300)	Persistent macular oedema
8	OS	6/15 (20/50)	1/66 (3/200)	Macular haemorrhage
9	OS	1/66 (3/200)	1/66 (3/200)	Persistent macular oedema
13	OS	6/21 (20/70)	LP	Retinal detachment
15	OS	1/40 (5/200)	6/120 (20/400)	Persistent macular oedema
17	OS	6/120 (20/400)	HM	Persistent macular oedema
23	OS	1/100 (2/200)	1/100 (2/200)	Macular hole
24	OS	1/40 (5/200)	1/40 (5/200)	Macular hole
25	OS	6/60 (20/200)	6/60 (20/200)	Disruption of the macular pigment epithelium
28	OD	1/200 (1/200)	HM	Persistent cystoid macular oedema
29		1/66 (3/200)	HM	Preretinal fibrosis
33		1/21 (9/200)	6/90 (20/300)	Macular photocoagulation scar

*NLP indicates no light perception; LP, light perception; and HM, hand motions.

†NVG indicates neovascular glaucoma.

viously in the involved eye. Another patient had bilateral occlusion within a month, and both eyes were included in the study. Two patients had total superior branch vein occlusion in their noninvolved eyes (see Table 4).

The persistence of oedema was the main cause of prolonged decreased vision. The average duration of macular oedema was longer in the treated group (13.0 months) than in the untreated group (11 months). The long duration of macular oedema and its sequelae were the main reasons for poor visual results in most of the eyes. This was especially true for the treated eyes. The duration of the oedema might have been reduced if we had administered photocoagulation within the temporal vascular arcades. All photocoagulation was outside the vascular arcades. All eyes except 1 developed CME prior to photocoagulation. This eye had a previous branch vein occlusion and had well-developed shunt vessels on the disc at the time of the central retinal vein occlusion.

On the average NVD was seen earlier in the treated than in the nontreated group. In 3 of the eyes it developed after photocoagulation. The NVD cleared faster in the treated eyes, but a small amount persisted in 2 treated eyes. No conclusion could be reached with respect to NVE because only 1 patient in each group developed it (see Table 4).

All 5 eyes in the nontreated group that developed rubeosis and NVG had large areas of retinal capillary obliteration (16.0 disc areas or more), as demonstrated by fundus fluorescein angiography. The remaining nontreated eyes had significantly smaller areas of retinal capillary obliteration (9.0 disc areas or less). Four of the treated patients had large areas of retinal capillary obliteration that were comparable to those of the eyes developing rubeosis and NVG. None of the eyes in the treated group, however, developed rubeosis or NVG.

The development of rubeosis may be directly related to the degree of retinal capillary obliteration. This may serve as a useful prognostic indicator as to which eyes have a high probability of developing rubeosis after central retinal vein occlusion. Eyes with large areas of retinal capillary obliteration can be treated early with panretinal photocoagulation, and the unfortunate sequelae of rubeosis and NVG may be prevented.

This is a small series of eyes, and the areas of capillary drop-out reported here provide only a relative indication of the degree of retinal hypoxia required to produce rubeosis and NVG. Additional studies on many more eyes will be required for any absolute determination to be made as to what amount of retinal capillary obliteration renders an eye susceptible to rubeosis and NVG. At present

from the results of this study it would seem that eyes with 10 or more disc areas of retinal capillary obliteration would be at greater risk and should be considered for panretinal photocoagulation.

The progression of rubeosis to NVG was very rapid in the affected eyes. Neovascular glaucoma was clinically present an average of 93 days after the onset of central retinal vein occlusion, which was consistent with the classic presentation of the so-called 100-day glaucoma.

More than half of the involved eyes in our series that did not develop NVG had lower intraocular pressures than in the noninvolved eye for an average of greater than 6 months. This has been noted by others (Moore, 1922; Vannas and Raitta, 1970; Hayreh, *et al.*, 1978) as well, but no explanation is available at present. No relationship was found to the degree of retinal capillary drop-out or to any other known factors in the eyes of our series. If the noninvolved eye of a patient in our series had primary open-angle glaucoma, both eyes were treated for open-angle glaucoma. Eventually both eyes of each patient equalised their pressures to within 2 mmHg or less, including those being treated for glaucoma.

Panretinal photocoagulation is not without complications. Two patients of our series had a decrease or loss of useful vision as a result of treatment. These included 1 eye that developed a retinal detachment and an accidental photocoagulation of the macula of another eye. A macular haemorrhage that developed 4 weeks after treatment also may have been precipitated by photocoagulation. Iris burns were created inadvertently in 2 eyes and transient choroidal effusions resulted in 3 eyes. Caution must be exercised in the delivery of photocoagulation.

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