

Photocoagulation Treatment of Proliferative Diabetic Retinopathy

Clinical Application of Diabetic Retinopathy Study (DRS) Findings, DRS Report Number 8

THE DIABETIC RETINOPATHY STUDY RESEARCH GROUP

Abstract: Additional follow-up confirms previous reports¹⁻⁴ from the Diabetic Retinopathy Study (DRS) that photocoagulation, as used in the study, reduces the risk of severe visual loss by 50% or more. Decreases of visual acuity of one or more lines and constriction of peripheral visual field due to treatment were also observed in some eyes. These harmful effects were more frequent and more severe following the DRS xenon technique.^{3,4} The two-year risk of severe visual loss without treatment outweighs the risk of harmful treatment effects for two groups of eyes: (1) eyes with new vessels and preretinal or vitreous hemorrhage; and (2) eyes with new vessels on or within one disc diameter of the optic disc (NVD) equaling or exceeding 1/4 to 1/3 disc area in extent, (Fig 1), even in the absence of preretinal or vitreous hemorrhage. For eyes with these characteristics, prompt treatment is usually advisable. For eyes with less severe retinopathy, DRS findings do not provide a clear choice between prompt treatment or deferral unless progression to these more severe stages occurs. [Key words: clinical trial, diabetic retinopathy, photocoagulation.] Ophthalmology 88:583-600, 1981

The Diabetic Retinopathy Study (DRS) is a randomized controlled clinical trial sponsored by the National Eye Institute to evaluate photocoagulation treatment for proliferative diabetic retinopathy. Seventeen hundred fifty-eight patients were enrolled in the study between 1972 and 1975. On the basis of data demonstrating a strong beneficial treatment effect, the protocol was changed in 1976 to allow photocoagula-

tion treatment of eyes initially assigned to the untreated control group in which the risk of severe visual loss without treatment clearly outweighed the risk of harmful effects from treatment. Patient follow-up was completed on June 30, 1979.

MATERIALS AND METHODS

Eligible patients had proliferative diabetic retinopathy in at least one eye or severe nonproliferative retinopathy in both eyes, and visual acuity of 20/100 or better in each eye. One eye of each patient was randomly assigned to immediate photocoagulation and the other to follow-up without treatment regardless of the course followed by either eye. The eye chosen for photocoagulation was randomly assigned to either of

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Presented at the Eighty-Fifth Annual Meeting of the American Academy of Ophthalmology, Chicago, November 2-7, 1980.

The DRS is supported by contracts with the National Eye Institute, National Institutes of Health, US Department of Health and Human Services.



Fig 1. Standard Photograph No. 10A, Modified Airlie House Classification of Diabetic Retinopathy. New vessels cover about 1/4 the area of this disc, which is a little larger than average. New vessels equaling these might cover 1/3 the area of a smaller disc. New vessels equaling or exceeding those in this photograph are sufficient to place an eye in the "high-risk" category (without regard to the size of the disc).

two treatment techniques, one using the argon laser and the other the xenon arc photocoagulator. Patients were followed at four-month intervals according to a protocol which provided for measurement of best corrected visual acuity by examiners who did not know the identity of the treated eye and who attempted to reduce patient bias by urging the patient to read as far down the chart as possible with each eye, guessing at letters until more than one in a line was missed. All patients gave written informed consent and understood that the information being collected would be analyzed at frequent intervals and used, if possible, for their benefit.

Key features of the argon and xenon photocoagulation techniques used in the DRS are summarized in Table 1. Both techniques included "scatter" (panretinal) photocoagulation extending to or beyond

the vortex vein ampulae and often completed in a single sitting. The argon treatment technique specified 800 to 1600 500-micron scatter burns of 0.1 seconds duration and focal treatment of new vessels whether on or within one disc diameter of the optic disc (NVD) or outside this area ("elsewhere," NVE). This focal treatment was applied both for new vessels on the surface of the retina and for elevated new vessels. Focal treatment was also applied to microaneurysms or other lesions thought to be causing macular edema. Follow-up treatment was applied as needed at four-month intervals. The xenon technique was similar, but scatter burns were generally of longer duration and stronger and focal treatment was not applied to NVD or elevated NVE. Figs 2 and 3, which are montages of 24-hour post-treatment photographs, illustrate the seven standard photographic fields used in the DRS and the two treatment techniques employed. Further details regarding methods are described elsewhere.⁵

RESULTS

As in previous DRS reports, "severe visual loss" is defined as visual acuity of less than 5/200 at two or more consecutively completed follow-up visits (visits scheduled at four-month intervals). The difference between the rates of occurrence of severe visual loss in eyes assigned to the control and treated groups provides the major measure of treatment effect. Cumulative incidence rates of this event, calculated by a life-table method,⁶ are presented in Table 2. In this table, eyes are classified by the treatment group to which they were randomly assigned at entry. No exceptions have been made, even for control group eyes that were treated after the 1976 protocol change. All follow-up observations collected prior to termination of patient follow-up in June 1979 are included in this analysis. In Table 2, Z is the difference between the rates for control and treated eyes measured in standard errors.^{1,2}

The rates in the "both groups combined" section of Table 2 are plotted in Fig 4, together with the comparable rates from an analysis which excludes observations made after the 1976 protocol change. The two curves for control group eyes are very similar over the first 20 months of follow-up and those for treated eyes are similar over at least the first 28 months. The difference between the two control group curves is probably due, at least in part, to the beneficial effect of treatment experienced by some of these eyes after the 1976 protocol change. A rough estimate of the extent to which the curve which includes post-change-of-protocol observations may be influenced by treatment may be obtained from Table 3, which presents the total number of patients completing specified follow-up visits, the percentage of these visits completed after the 1976 protocol change, and the cumulative percentages of control group eyes that had been treated prior to each visit. In each of the analyses presented in Figure 4, the incidence of severe visual loss in eyes

Table 1. DRS Photocoagulation Techniques

	Argon	Xenon
Scatter		
No. of Burns	800-1600 (500 μ m) or 500-1000 (1000 μ m)	400-800 (3°) or 200-400 (4.5°)
Exposure time	0.1 second	not specified
Focal		
Surface NVE	+	+
Elevated NVE	+	—
NVD	+	—
Macular Edema	+	+
Follow-up	+	+

Fig 2. Twenty-four-hour post-treatment photographs of an eye treated with the DRS argon technique. Note extensive 500 μ m scatter burns, confluent focal treatment of two small patches of NVE along the inferior temporal artery inferotemporal to the macula, and focal treatment of NVD adjacent to the disc.

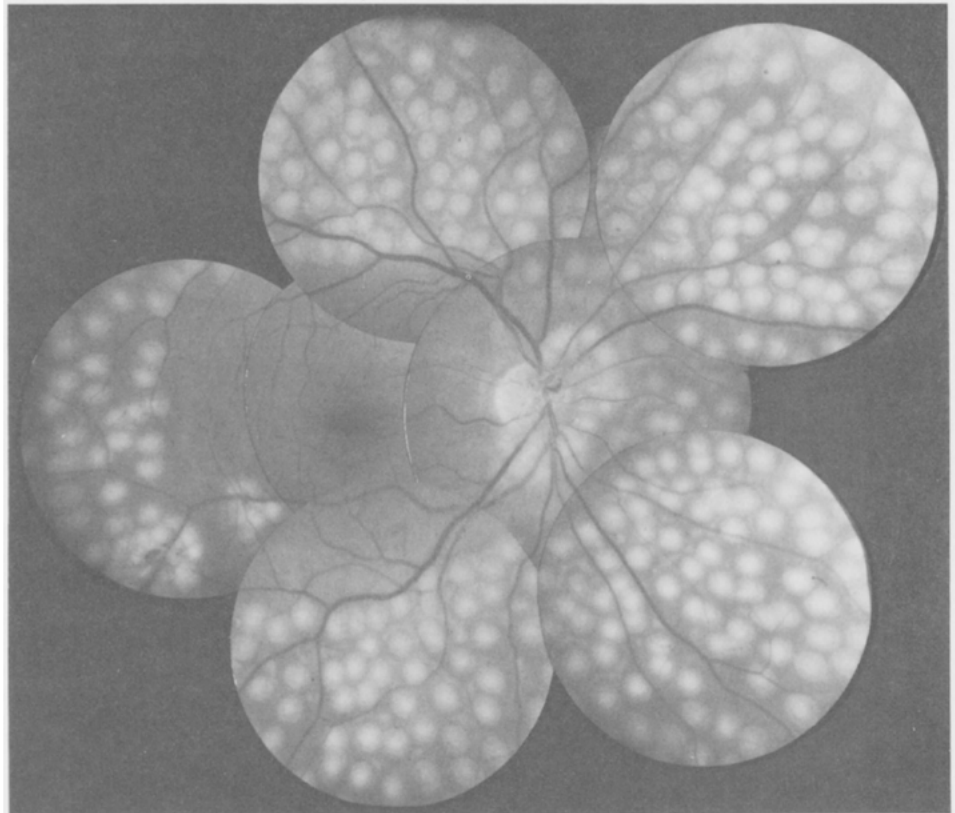


Fig 3. Twenty-four-hour post-treatment photographs of an eye treated with the DRS xenon technique. Scatter burns were made with the 4.5° field stop and are less evenly spaced than the argon burns in Fig 2. Confluent focal treatment has been applied to four patches of surface NVE. The NVE superotemporal to the disc are congested and a small preretinal hemorrhage has occurred since treatment. Focal treatment has been applied temporal to the macula to microaneurysms thought to be the cause of mild macular edema.

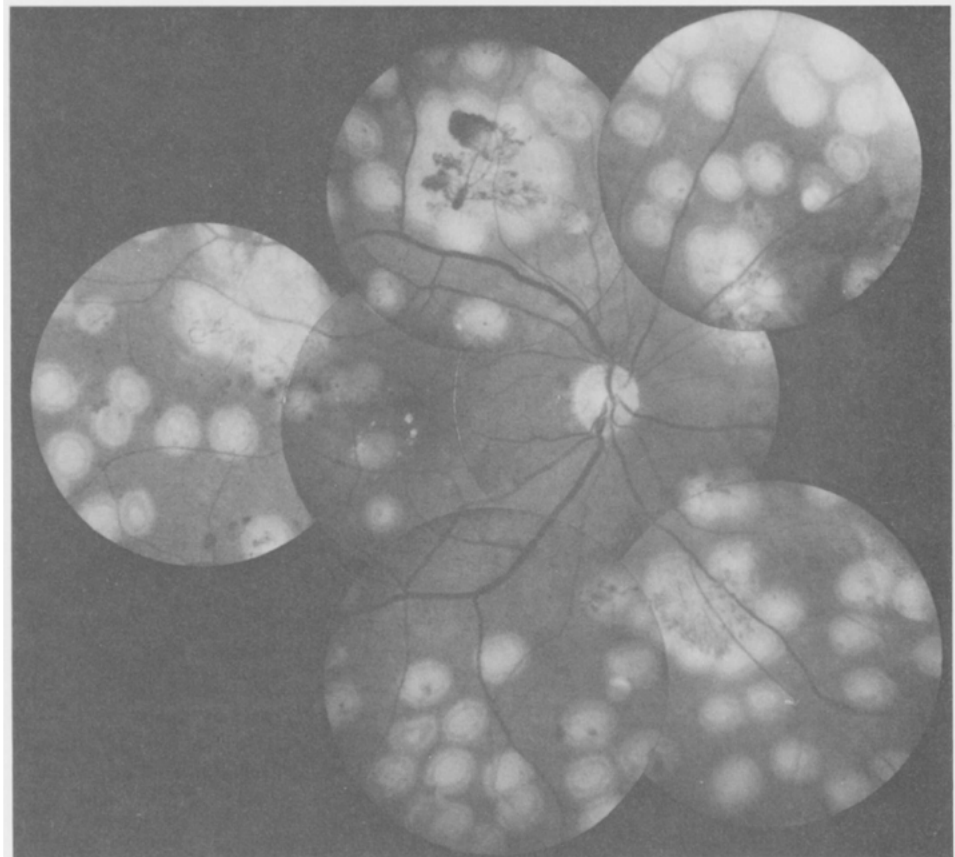


Table 2. Cumulative Event Rates of Severe Visual Loss, Z Values of Control-treated Differences, Number of Eyes at Risk in Each Four-month Interval and Standard Errors by Months of Follow-up and Treatment Group

Follow-up Months	Argon Group			Xenon Group			Both Groups Combined		
	Control	Treated	Z	Control	Treated	Z	Control	Treated	Z
A. Cumulative Event Rates Per 100 Eyes at Risk									
8	1.0	0.4	1.5	1.5	0.9	1.1	1.2	0.7	1.8
12	3.4	1.8	2.0	3.9	2.9	1.2	3.6	2.3	2.2
16	6.6	3.3	3.1	6.9	3.7	2.9	6.8	3.5	4.3
20	10.3	5.1	4.0	10.9	5.1	4.4	10.6	5.1	5.9
24	13.6	6.6	4.7	14.3	5.8	5.7	14.0	6.2	7.4
28	16.5	7.9	5.2	17.0	6.6	6.5	16.7	7.2	8.3
32	19.2	9.0	5.8	19.1	7.4	6.9	19.1	8.2	9.0
36	21.1	10.2	5.9	22.4	7.9	8.1	21.7	9.0	9.9
40	23.6	11.2	6.4	24.7	9.1	8.3	24.1	10.1	10.4
44	25.4	11.5	7.0	26.3	9.9	8.4	25.9	10.7	10.9
48	27.4	12.6	7.2	28.2	10.7	8.7	27.8	11.6	11.2
52	29.2	14.4	6.8	30.2	11.4	9.0	29.7	12.9	11.2
56	31.1	14.6	7.4	32.3	12.4	9.0	31.7	13.5	11.6
60	32.1	15.2	7.3	33.8	12.7	9.3	33.0	13.9	11.8
64	32.5	15.2	7.4	35.9	13.4	9.2	34.2	14.3	11.8
68	34.2	16.3	6.8	35.9	13.4	9.2	35.1	14.8	11.3
72	34.2	17.5	5.9	39.3	15.9	6.6	36.7	16.6	9.0
B. Number of Eyes at Risk in Each Four-month Interval									
4-8	834	835		847	847		1681	1682	
8-12	812	818		812	816		1624	1634	
12-16	767	786		781	797		1547	1584	
16-20	734	763		741	777		1475	1539	
20-24	692	738		689	754		1381	1492	
24-28	656	711		655	734		1311	1445	
28-32	614	680		628	715		1243	1394	
32-36	589	658		598	700		1187	1358	
36-40	557	633		571	684		1127	1317	
40-44	531	617		539	651		1071	1269	
44-48	477	560		483	597		960	1157	
48-52	407	480		406	516		813	997	
52-56	329	391		335	429		664	820	
56-60	259	310		260	341		519	650	
60-64	190	224		186	244		376	469	
64-68	118	142		117	154		235	297	
68-72	65	74		55	67		120	141	
C. Standard Errors									
8	0.3	0.2		0.4	0.3		0.3	0.2	
12	0.6	0.5		0.7	0.6		0.5	0.4	
16	0.9	0.6		0.9	0.7		0.6	0.5	
20	1.1	0.8		1.1	0.8		0.8	0.5	
24	1.2	0.9		1.2	0.8		0.9	0.6	
28	1.3	1.0		1.3	0.9		0.9	0.6	
32	1.4	1.0		1.4	0.9		1.0	0.7	
36	1.5	1.1		1.5	0.9		1.0	0.7	
40	1.5	1.1		1.5	1.0		1.1	0.8	
44	1.6	1.1		1.6	1.1		1.1	0.8	
48	1.6	1.2		1.6	1.1		1.1	0.8	
52	1.7	1.3		1.7	1.2		1.2	0.9	
56	1.8	1.3		1.8	1.2		1.2	0.9	
60	1.8	1.4		1.8	1.2		1.3	0.9	
64	1.8	1.4		2.0	1.3		1.3	1.0	
68	2.0	1.6		2.0	1.3		1.4	1.0	
72	2.0	1.9		2.7	2.2		1.7	1.4	

Fig 4. Cumulative rates of severe visual loss, including and excluding observations made after the 1976 protocol change, argon and xenon groups combined.

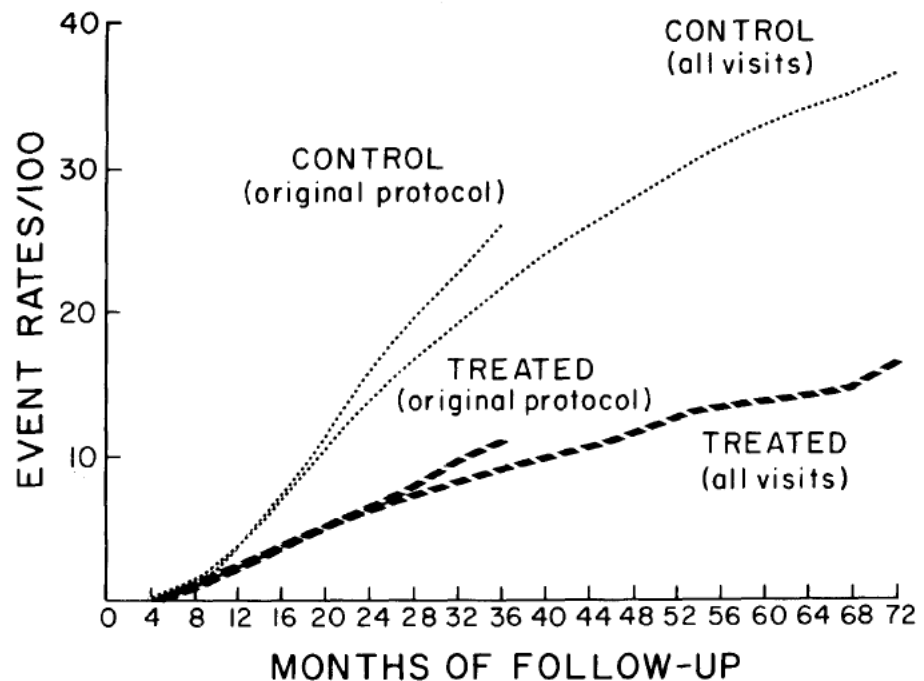


Table 3. Number of Patients Who Had Completed Specified Visits as of July 1, 1979, Percentages of Visits Completed After 1976 Protocol Change, and Percentage of Control Group Eyes Which Had Received Photocoagulation Prior to the Visit

Visit	Number of Patients Who Completed Visit	Percentage of Visits After 1976 Protocol Change	Percentage of Control Eyes Previously Treated
Pre-treatment	1742*	0.0	0.0
FV01 (6 weeks)	1673	0.0	0.0
FV04 (1 year)	1570	8.3	2.4
FV07 (2 years)	1468	44.3	11.9
FV10 (3 years)	1342	89.1	24.2
FV13 (4 years)	1176	100.0	34.4
FV16 (5 years)	673	100.0	39.6

* Sixteen patients assigned to the combined argon-xenon treatment group, which was discontinued shortly after recruitment began, have been excluded from all analyses in this report.

assigned to immediate treatment is reduced by 50% or more in comparison to control group eyes, and this reduction persists throughout the follow-up period.

Cumulative incidence rates of severe visual loss are plotted separately for the argon and xenon treatment groups in Fig 5, using the information in Table 2. The treatment effect, ie, the difference in rates between treated and control eyes, appears somewhat greater in the xenon than in the argon group, but the difference in treatment effect between argon and xenon groups is small, its statistical significance borderline, and its clinical importance outweighed by the greater harmful treatment effects observed following the DRS xenon technique.²⁻⁴

Analyses of the occurrence of severe visual loss in

eyes classified⁷ according to severity and location of new vessels and presence of vitreous and/or preretinal hemorrhage (VH/PRH) in baseline fundus photographs allowed documentation in previous DRS reports¹⁻³ of certain "high-risk characteristics." High-risk characteristics are presence of NVD equaling or exceeding those in Fig 1 (which are about 1/4 to 1/3 disc area in extent), with or without VH/PRH, or the presence of VH/PRH and less extensive new vessels, either NVD less than those in Fig 1 or NVE one-half disc area or more in extent (in any photographic field). It was for eyes with these characteristics that the 1976 protocol change was implemented. Fig 6 illustrates the greater risk of severe visual loss observed in these eyes than in the remaining eyes in the study, which had less severe

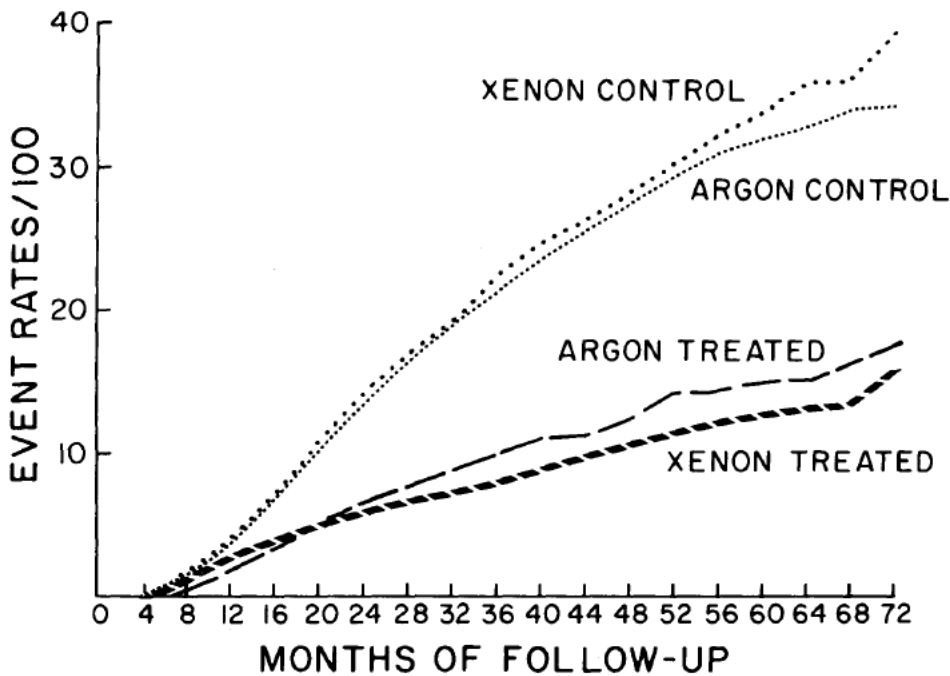


Fig 5. Cumulative rates of severe visual loss for argon and xenon groups separately.

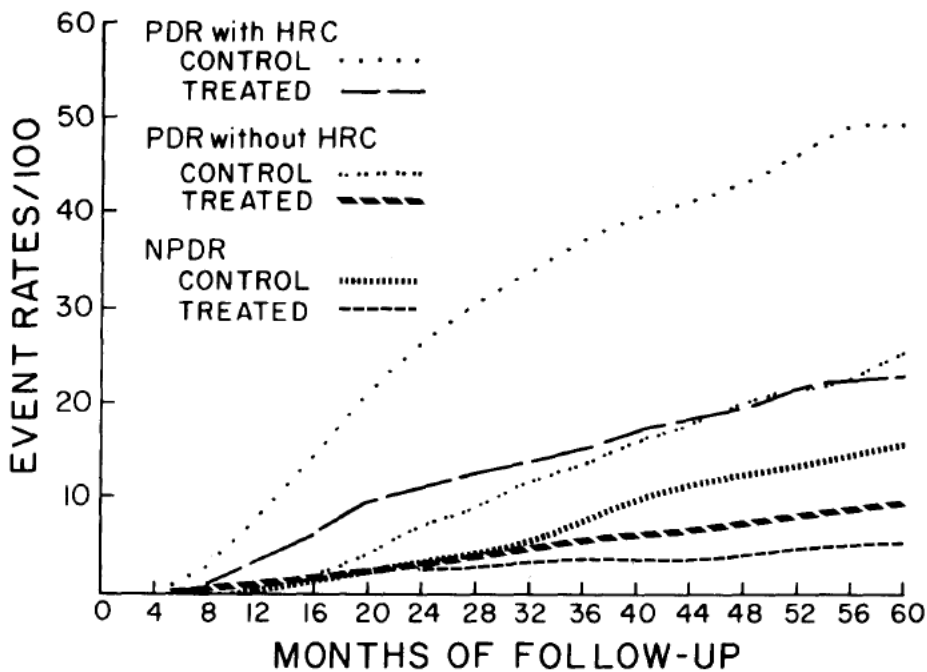


Fig 6. Cumulative rates of severe visual loss for eyes classified by presence of proliferative retinopathy (PDR) and high-risk characteristics (HRC) in baseline fundus photographs, argon and xenon groups combined. NPDR signifies nonproliferative diabetic retinopathy.

retinopathy. After 24 months of follow-up, the rate for control eyes with high-risk characteristics is about 26% and is reduced to about 11% in treated eyes. After 24 months of follow-up, a similar 50% treatment effect can be seen in eyes with proliferative retinopathy without high-risk characteristics, but both control and treated rates are lower, about 7 and 3%, respectively. Only after 36 months of follow-up does the incidence of severe visual loss in untreated eyes with nonproliferative retinopathy reach the 7% level. At this point, a similar 50% treatment effect is apparent. Some eyes with nonproliferative retinopathy are those of patients who were eligible for the study because severe nonproliferative retinopathy was present in both eyes and some are the fellow eyes of patients eligible because proliferative retinopathy was present in the other eye. In many of the eyes without high-risk characteristics at baseline which subsequently developed severe visual loss, the development of high-risk characteristics presumably preceded the occurrence of severe visual loss.

Harmful effects of treatment were greater in the xenon group, as indicated in Table 4, which summarizes information from previous DRS reports.²⁻⁴ Twenty-five percent of xenon-treated eyes suffered a modest loss of visual field and an additional 25% a more severe loss. In the argon group, 5% of eyes showed some constriction of visual field to the large test object used (Goldmann IIVe4). We estimate that a persistent visual acuity decrease of one line may be attributed to treatment in 19% of xenon-treated eyes and a persistent decrease of two or more lines in an additional 11%. Comparable estimates for the argon group are 11% and 3%, respectively.

Partly on the basis of results similar to these, photocoagulation techniques were modified when treatment was carried out in eyes initially assigned to the untreated control groups after the 1976 protocol change. Argon treatment was preferred but the most technically difficult features of the original protocol, focal treatment of NVD and elevated NVE, were not re-

quired and were rarely used. In the hope of reducing the risk of visual acuity decreases, many DRS investigators divided scatter treatment into two or more episodes, days or weeks apart.

CLINICAL APPLICATION OF DRS RESULTS

Clinical application of DRS results to eyes with high-risk characteristics is straightforward. The risk of severe visual loss without treatment substantially outweighs the risks of photocoagulation, particularly those observed following the DRS argon technique, and prompt treatment is usually advisable. Figs 7 through 11 illustrate the course followed by five DRS patients who had high-risk characteristics in both eyes. The patient presented in Fig 11 illustrates the remarkable spontaneous improvement sometimes observed in patients with proliferative diabetic retinopathy and underscores the need for randomized, controlled trials in evaluating treatment.

For eyes with severe nonproliferative retinopathy or with proliferative retinopathy without high-risk characteristics, prompt photocoagulation is attractive, since with it, the 12% two-year risk of severe visual loss observed in the high-risk group even *with* photocoagulation might be avoided. On the other hand, deferral of photocoagulation unless high-risk characteristics develop defers the risks of photocoagulation. Eyes followed and treated as soon as high-risk characteristics develop might be expected to do better than did the DRS high-risk group, which included eyes which already had more severe retinopathy at the time of treatment. DRS findings do not provide a clear choice between prompt photocoagulation and deferral.

Careful, periodic follow-up is essential for patients with diabetic retinopathy. When nonproliferative retinopathy changes are present, follow-up visits should be scheduled at frequent intervals.

For eyes with nonproliferative retinopathy or proliferative retinopathy without high-risk characteristics, important questions remain unanswered. Is early photocoagulation preferable to deferral? Is the extensive scatter treatment used in the DRS necessary, or might less extensive treatment retard the progression of retinopathy and be attended by fewer harmful side effects? Apart from its value for the proliferative features of diabetic retinopathy, what is the role of photocoagulation in diabetic maculopathy? Might low dose aspirin slow the progression of retinopathy? These questions are being addressed by another randomized controlled clinical trial, the Early Treatment Diabetic Retinopathy Study (ETDRS).

Table 4. Estimated Percentages of Eyes With Harmful Effects Attributable to DRS Treatment

	Argon	Xenon
Constriction of visual field (Goldmann IIVe4) to an average of		
$\leq 45^\circ$, $> 30^\circ$ per meridian	5	25
$\leq 30^\circ$ per meridian	0	25
Decrease in visual acuity		
1 Line	11	19
≥ 2 Lines	3	11

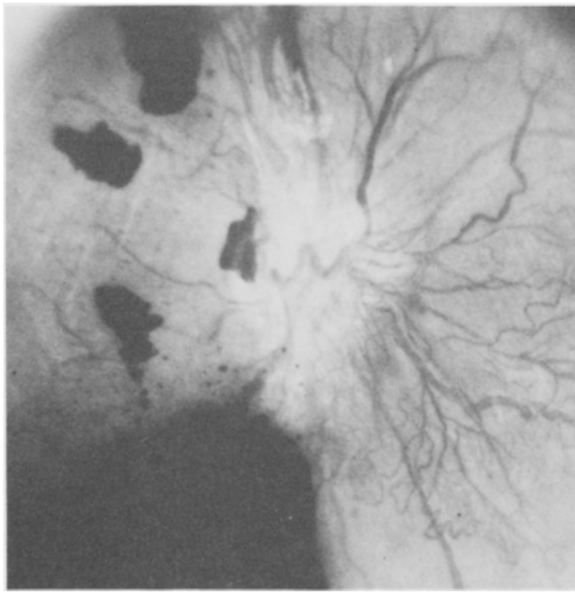


Fig 7A

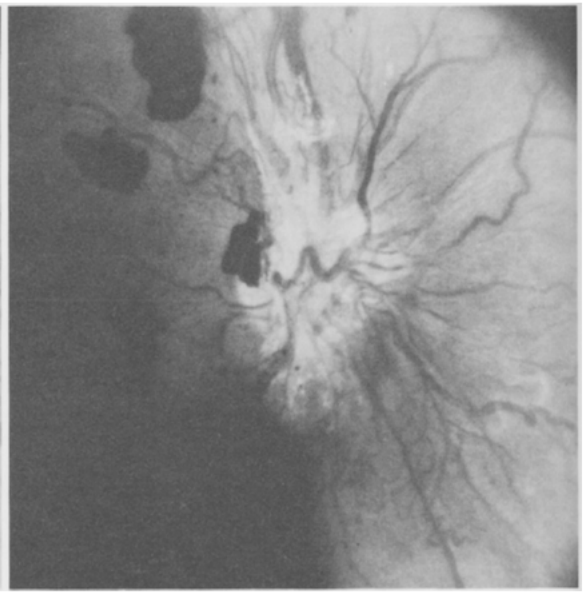
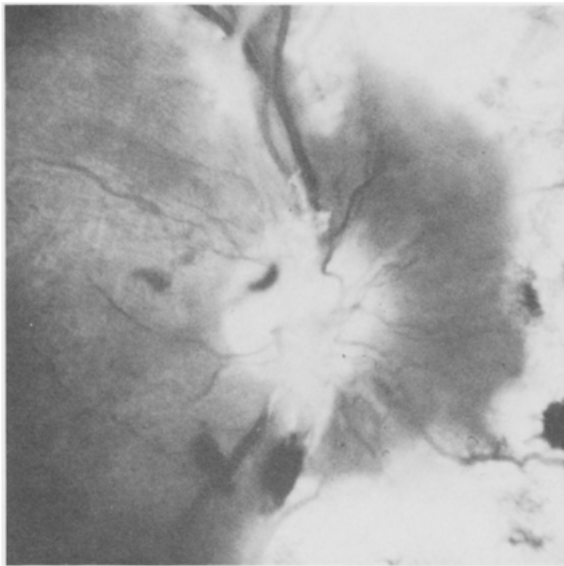


Fig 7B

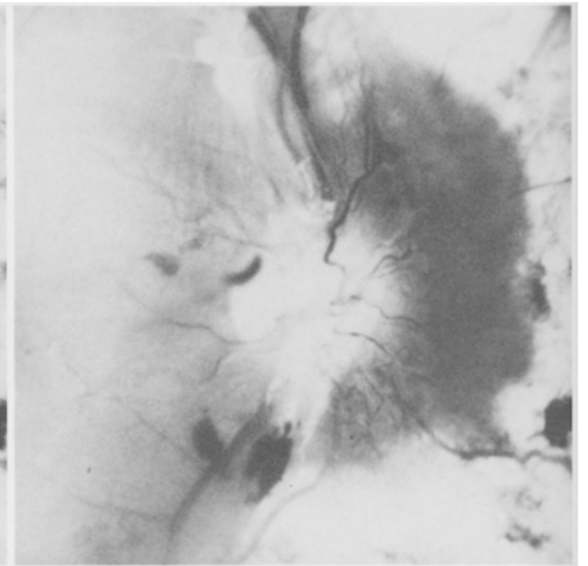
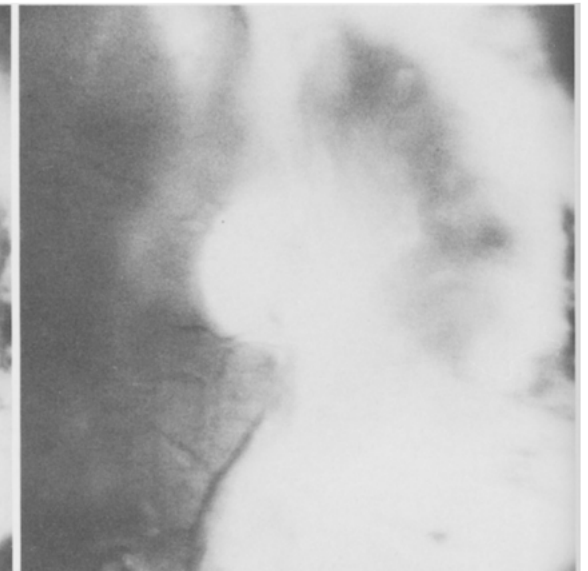


Fig 7C



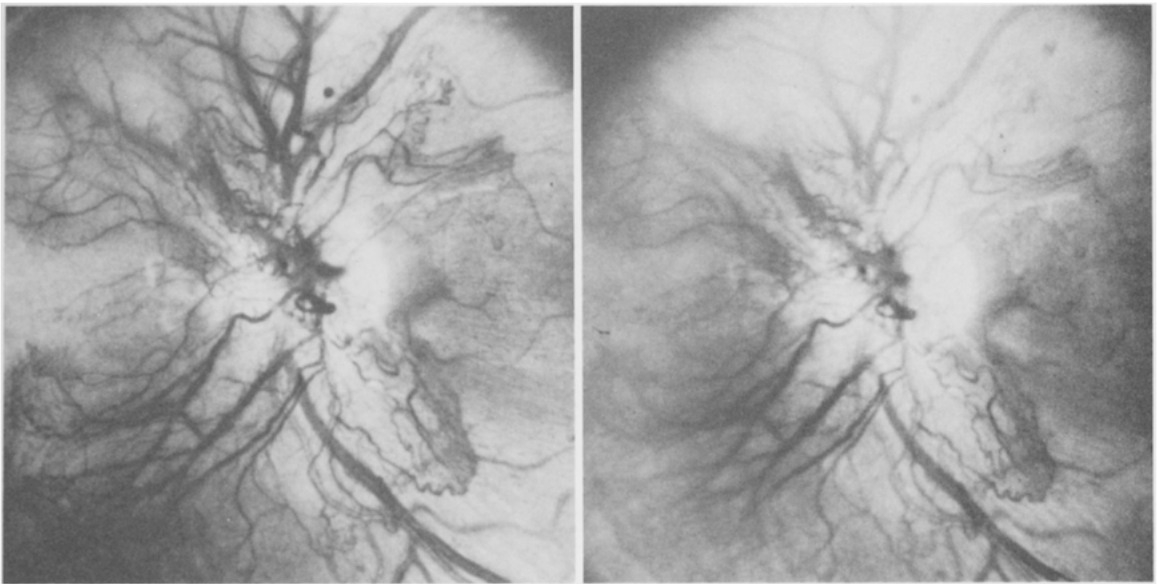


Fig 7D

Figs 7A–D. Twenty-three-year-old white man, diabetes diagnosed at age 4. Decreased vision first noted in left eye two years ago, with little change since. This and the remaining figures present stereo pairs. Some observers may require up to 10 diopters prism base out and, if presbyopic, sufficient plus power for a viewing distance of 20 to 25 centimeters in order to obtain a stereoscopic view. **7A.** Right eye at entry. Visual acuity 20/30. Very severe NVD are present, with preretinal and vitreous hemorrhage. There is little stereoscopic effect in this photo. **7B.** Right eye four months following argon laser photocoagulation. Considerable regression of NVD has occurred and they have been pulled forward by vitreous contraction. **7C.** Right eye four years following initial photocoagulation. Follow-up photocoagulation was carried out on two occasions. NVD have regressed completely. Visual acuity 20/30. **7D.** Left eye at entry. Visual acuity 20/40. Very severe NVD. Visual acuity four months following entry into the study was less than 5/200 in the left eye, and fundus photography was precluded by vitreous hemorrhage. Vitreous hemorrhage failed to clear and two years later visual acuity was no light perception. Neovascularization of the iris, posterior synechia, and cataract were present.



Fig 8A

Fig 8A. Fifty-three-year-old white woman, diabetes diagnosed at age 27. Severe retinopathy noted at routine eye examination. Visual acuity 20/30 (RE), 20/15 (LE). **8A.** Right eye, initial visit. Severe NVD accompanied by fibrous proliferations extend forward, presumably along the detached posterior vitreous surface. There are tension lines in the macula. No preretinal or vitreous hemorrhage is present. (Figs 8B–F continued on following page.)

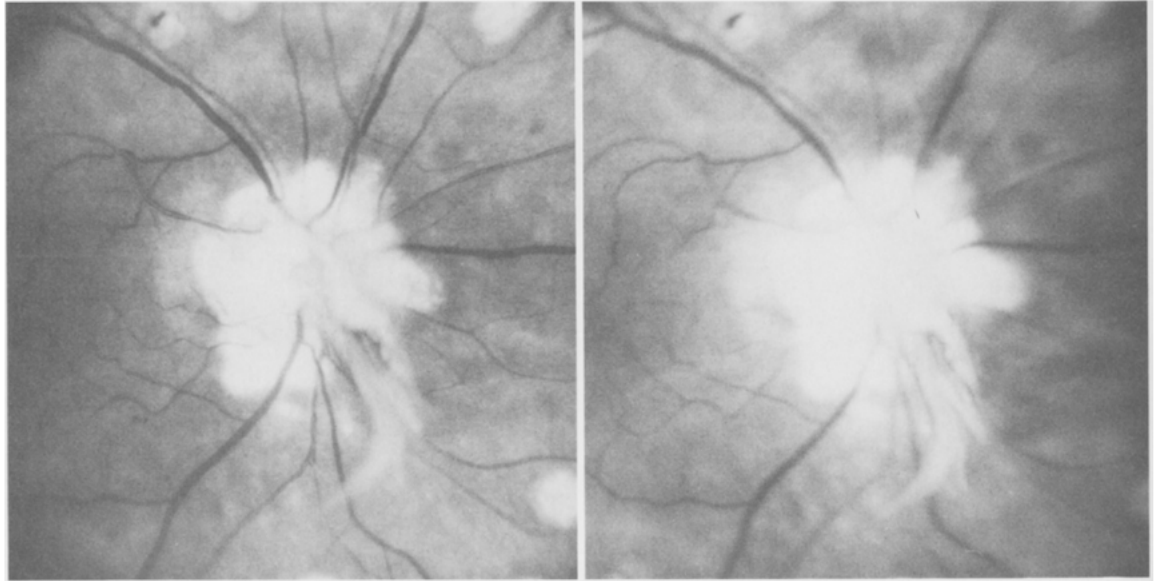


Fig 8B

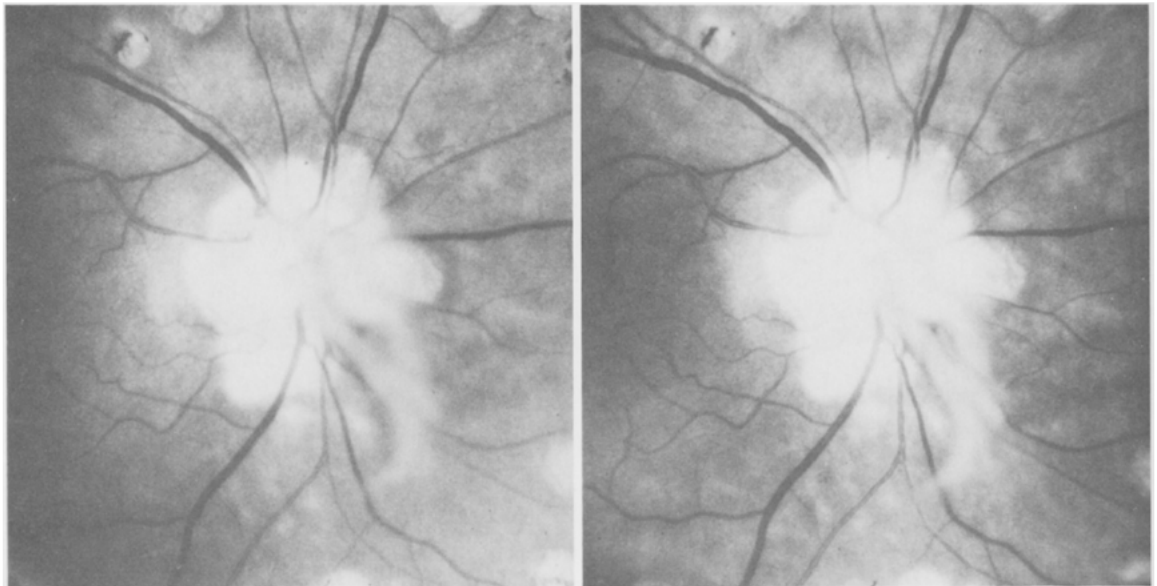


Fig 8C

Figs 8B–F. Right eye two years after argon laser photocoagulation. New vessels have regressed almost completely and tension lines have disappeared. Visual acuity 20/15. **8C.** Right eye four years post-treatment. New vessels have not increased and visual acuity remains 20/15. **8D.** Left eye, initial visit. NVD are perhaps slightly less extensive than those in the right eye, without fibrous proliferations and with only slight elevation of the temporal edge of the patch. A small preretinal hemorrhage was present in the lower temporal quadrant. **8E.** Left eye two years after entry, no treatment. New vessels, fibrous proliferations and posterior vitreous detachment have increased. Fine new vessels and fibrous proliferations are present on the surface of the retina superotemporal to the disc and there are faint tension lines in the macula. **8F.** Left eye four years after entry and one and one-half years after argon laser photocoagulation applied following the 1976 protocol change. Scatter treatment only was applied. Traction retinal detachment involving the macula occurred following photocoagulation, with decrease in visual acuity to 20/70, but resolved spontaneously with recovery of visual acuity to 20/30. New vessels have regressed almost completely. Fine tension lines in the macula remain.

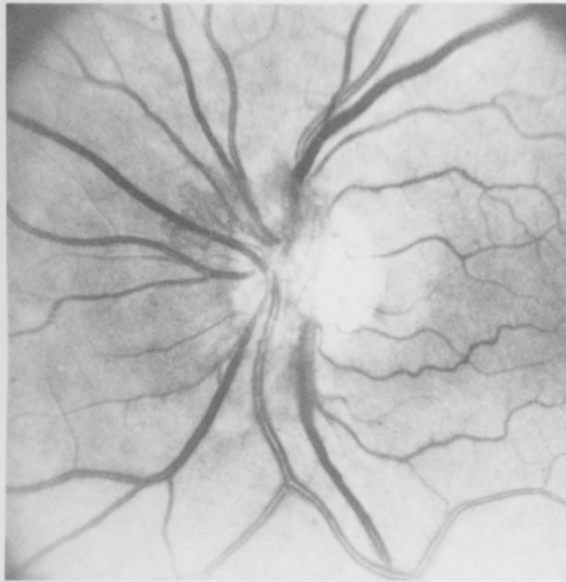


Fig 8D

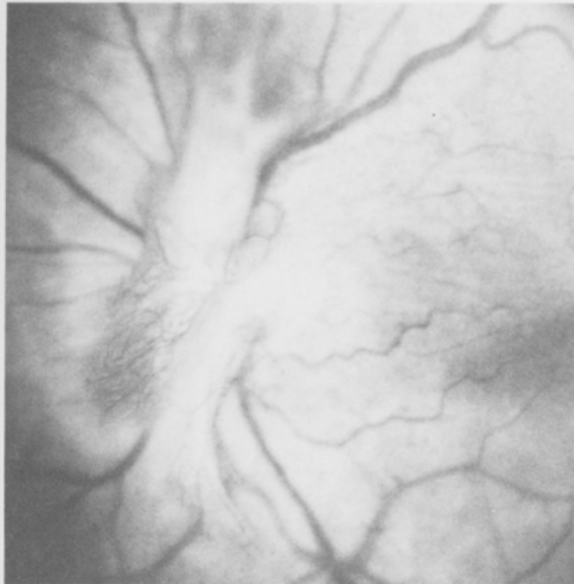


Fig 8E

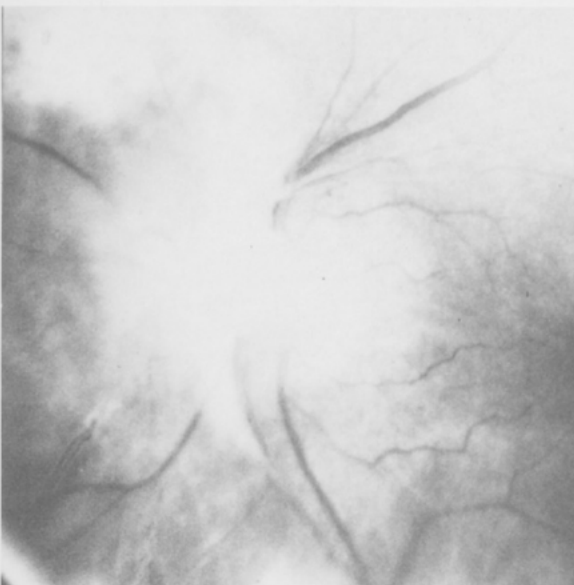


Fig 8F

Fig 9A

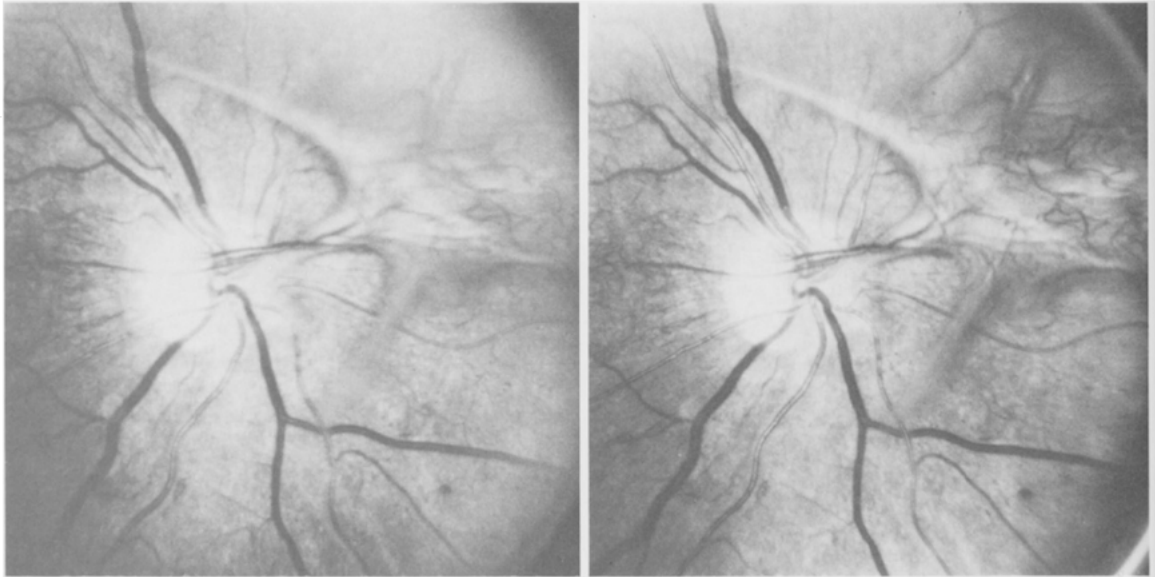
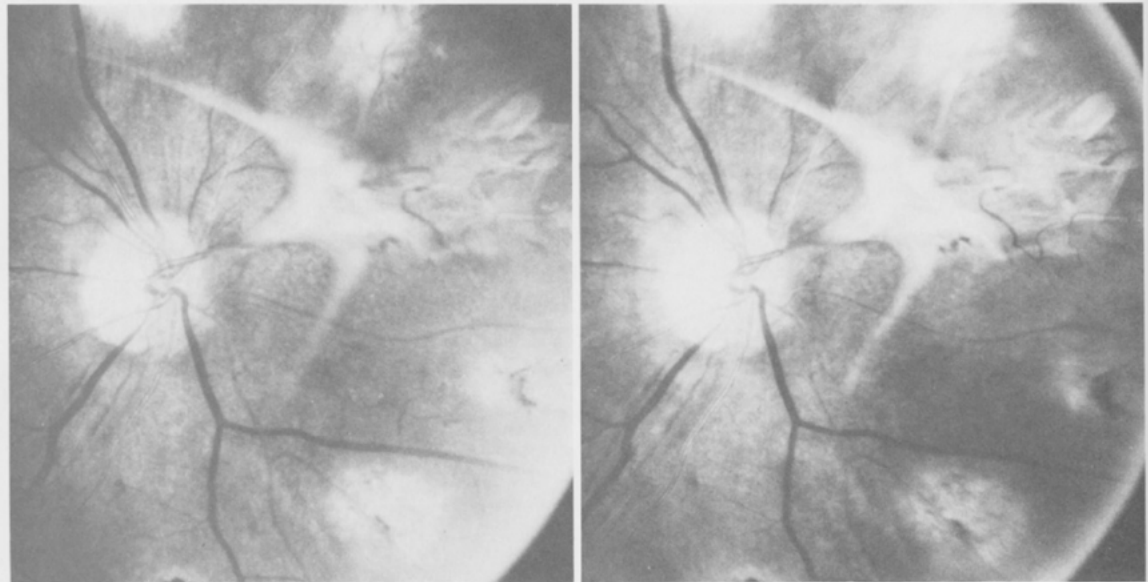


Fig 9B



Figs 9A-E. Thirty-six-year-old white woman, diabetes diagnosed at age 8. Visual acuity 20/20 (RE); 20/20 (LE). **9A.** Right eye at entry. A large patch of fibrovascular proliferations arises from the disc and extends nasally. The temporal edge of the patch is elevated. Intraretinal microvascular abnormalities (IRMA) are present temporal to the disc. A symptomatic vitreous hemorrhage had been noted in this eye two years previously and had cleared without recurrence. **9B.** Right eye three years following xenon arc photocoagulation. New vessels have regressed partially, IRMA more completely. There is narrowing and sheathing of some retinal arterioles. Fresh vitreous hemorrhage is visible on and anterior to the fibrous proliferations. This occurred periodically, apparently arising from elevated NVE in the upper temporal quadrant which had not regressed. Visual acuity 20/30. Visual field constricted to 20 degrees in the upper and lower nasal quadrants. **9C.** Right eye five years following photocoagulation. Disc new vessels remain almost completely regressed. Visual acuity remains 20/30. **9D.** Left eye at entry. NVD extend straight forward from the disc, suggesting posterior vitreous detachment. Vitreous hemorrhage is present. **9E.** Left eye two years after entry. New vessels, fibrous proliferations and IRMA have increased somewhat. Visual acuity has improved to 20/15. This was the patient's first visit after the 1976 protocol change. The patient was reluctant to have photocoagulation in this eye because she attributed the decrease in visual acuity and visual field in the right eye to treatment. During the next 18 months, several small vitreous hemorrhages occurred and argon laser photocoagulation was performed. (Fig 9F continued on following page.)



Fig 9C



Fig 9D

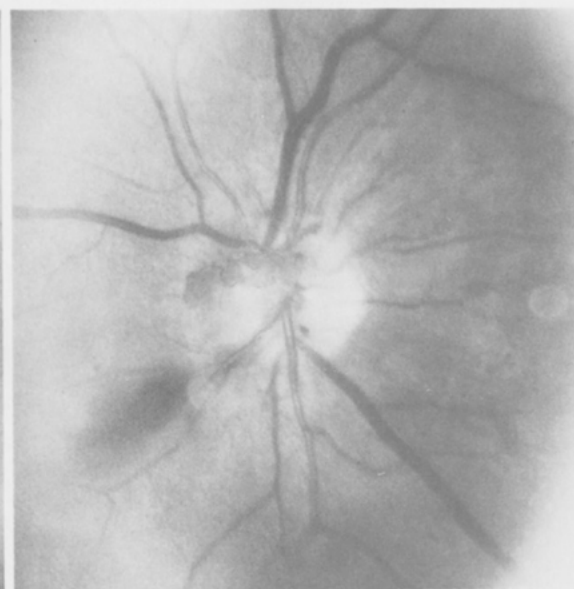
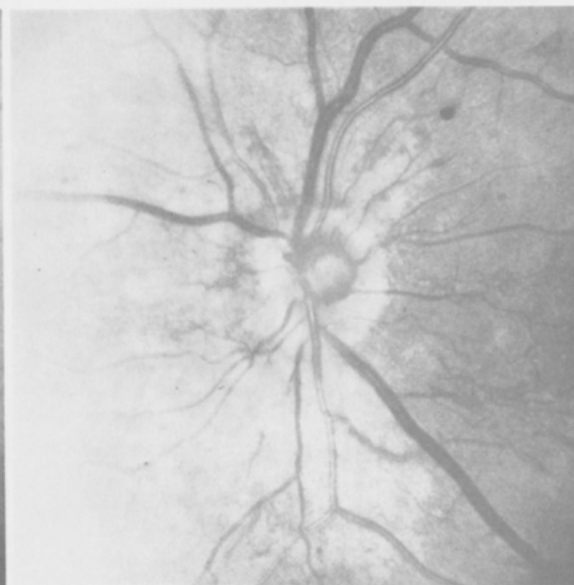


Fig 9E



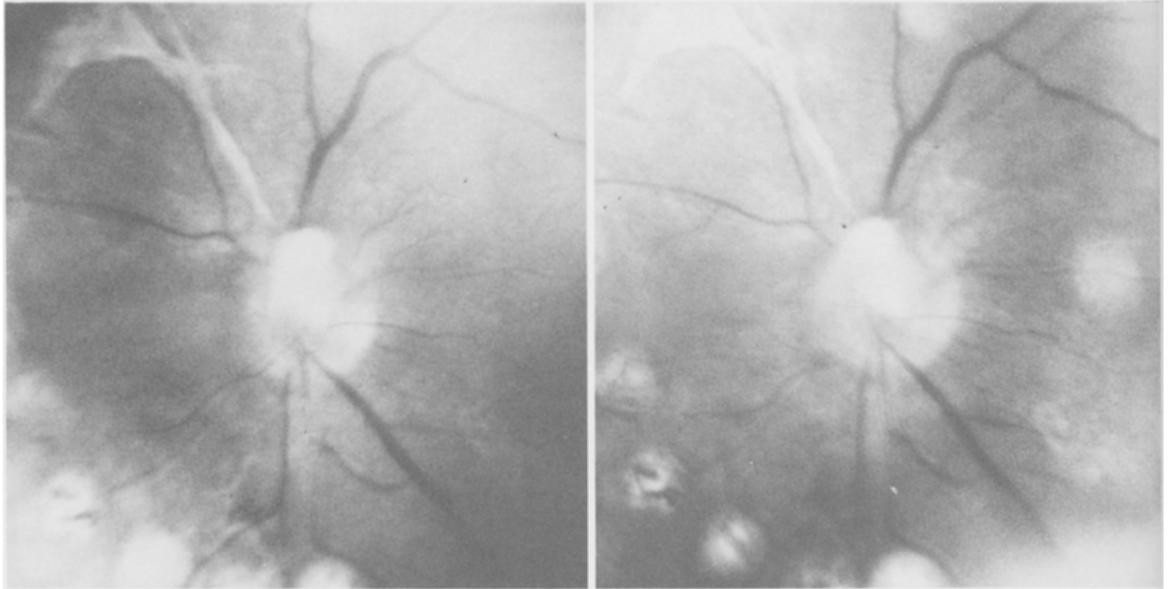


Fig 9F

Fig 9F. Left eye five years after entry and two years after argon laser photocoagulation. NVD have regressed completely. Visual acuity 20/30. Occasional vitreous hemorrhages occur from elevated NVE which have not regressed.

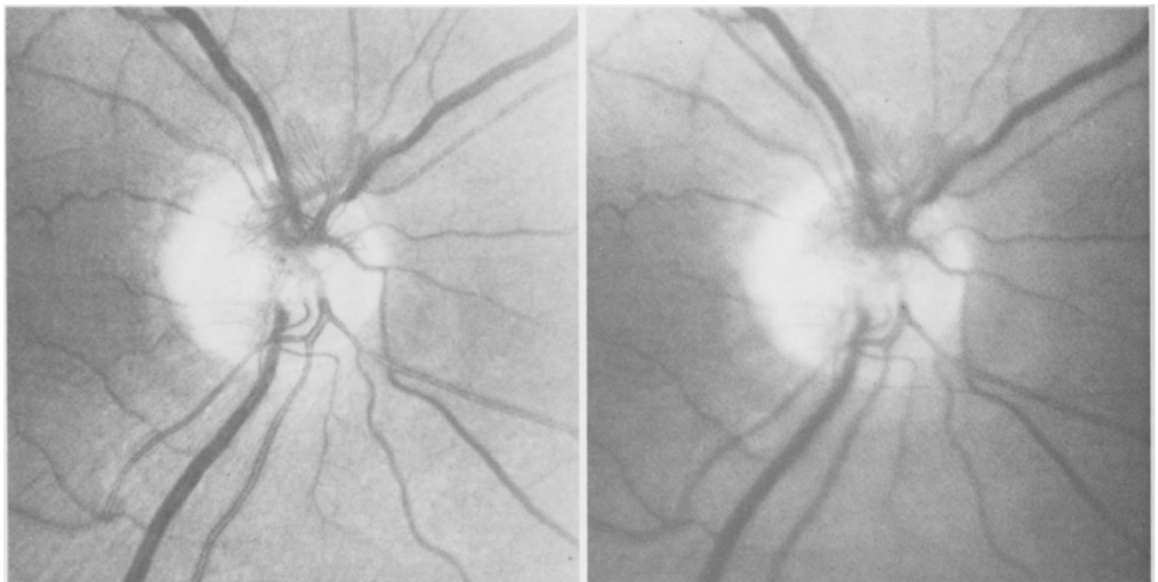


Fig 10A

Figs 10A–D. Fifty-two-year-old white man, diabetes diagnosed at age 20. Visual acuity 20/20 (RE); 20/20 (LE). **10A.** Right eye at entry. NVD of moderate extent are present. There were no NVE nor preretinal or vitreous hemorrhage. **10B.** Right eye, three years following xenon arc photocoagulation. Complete regression of NVD has occurred. Retinal arterioles are narrow. Visual acuity remains 20/20. Visual field is constricted to about 30 degrees in most meridians, 55 degrees temporally. **10C.** Left eye at entry. A tiny strand of NVD can be seen near the disc margin from 3 to 4 o'clock. There is a little preretinal hemorrhage lying over the inferior temporal artery. A small amount of vitreous hemorrhage was present in the inferior nasal quadrant. **10D.** Left eye, three years after entry. New vessels have grown superiorly along the superior temporal vein. Vitreous detachment has occurred and the new vessels have been pulled forward. Vitreous hemorrhages have occurred several times during the past three years. Visual acuity was 20/50 at the three-year visit, but fell to less than 5/200 eight months later. Photocoagulation could not be carried out following the 1976 protocol change because of vitreous hemorrhage.

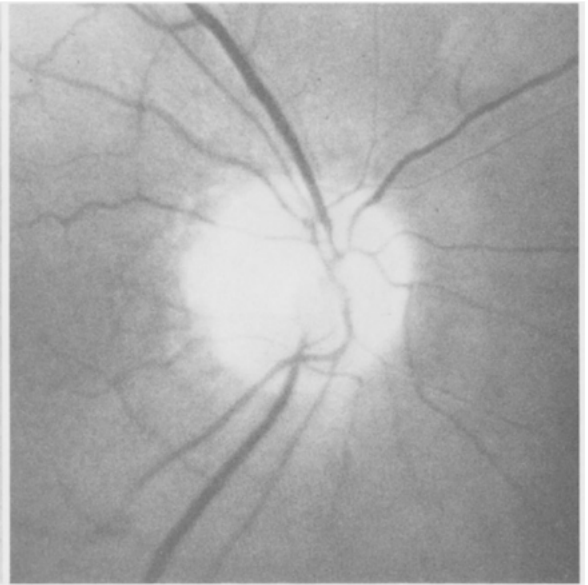


Fig 10B



Fig 10C

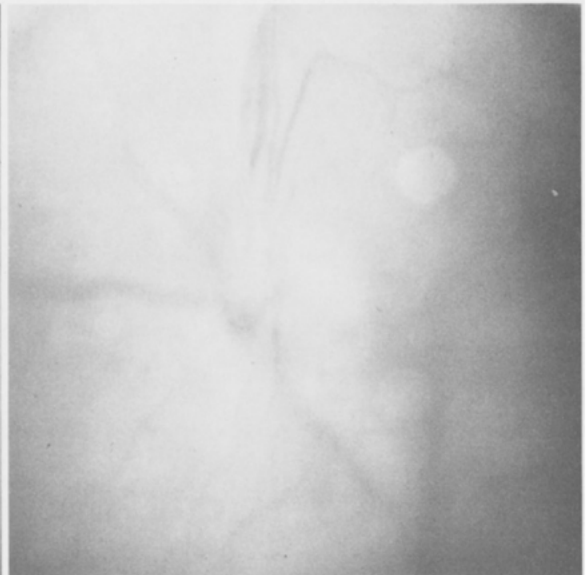


Fig 10D

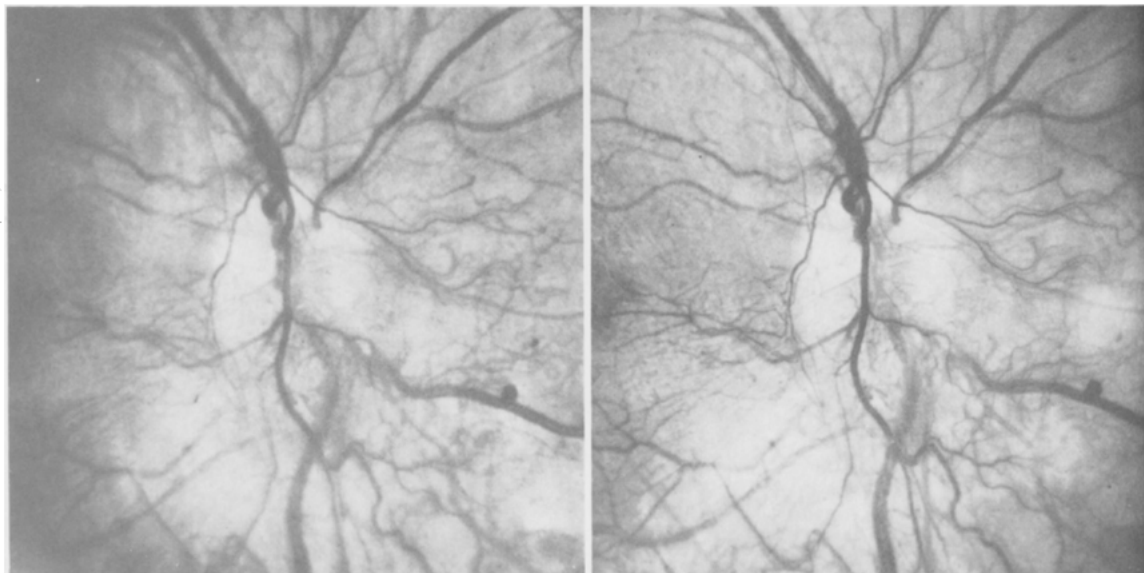


Fig 11A

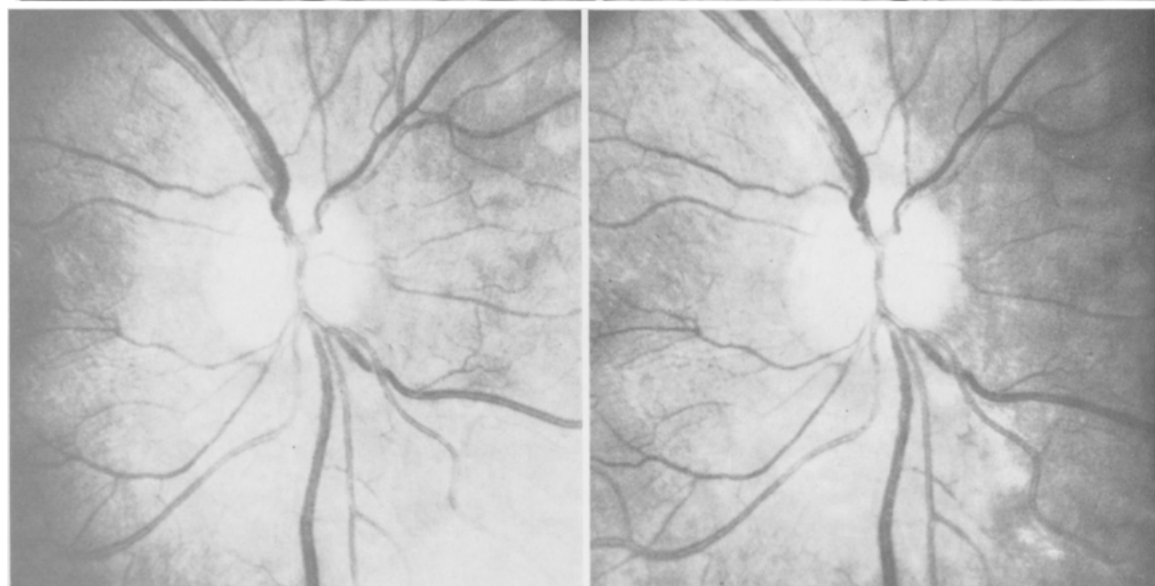


Fig 11B

Figs 11A–E. Twenty-year-old white man, diabetes diagnosed at age 14. Floaters and decreased vision noted several weeks ago in the left eye, and a few days later in the right eye. **11A.** Right eye at entry, visual acuity 20/15. There are extensive new vessels on and near the disc. The disc is swollen. Two flecks of preretinal hemorrhage are present nasally. **11B.** Right eye four months following entry. Remarkable spontaneous regression of new vessels has occurred. Visual acuity 20/15. **11C.** Right eye four years following entry. New vessels have not recurred. **11D.** Left eye at entry. Visual acuity 20/30. New vessels, disc swelling, and preretinal hemorrhage similar to right eye. More vitreous hemorrhage was present inferiorly. **11E.** Left eye four months following argon laser scatter photocoagulation. Focal photocoagulation was not required by DRS protocol for NVD as severe as those present at entry in this eye, and such treatment was not carried out. Almost complete regression of new vessels has occurred. Visual acuity 20/20. (Fig 11F continued on following page.)



Fig 11C

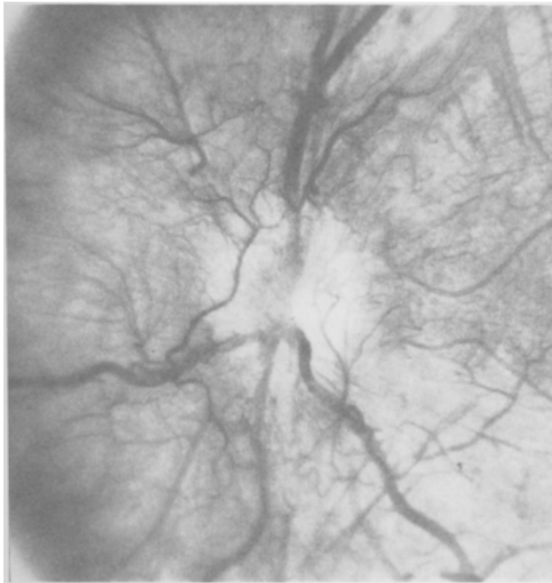


Fig 11D

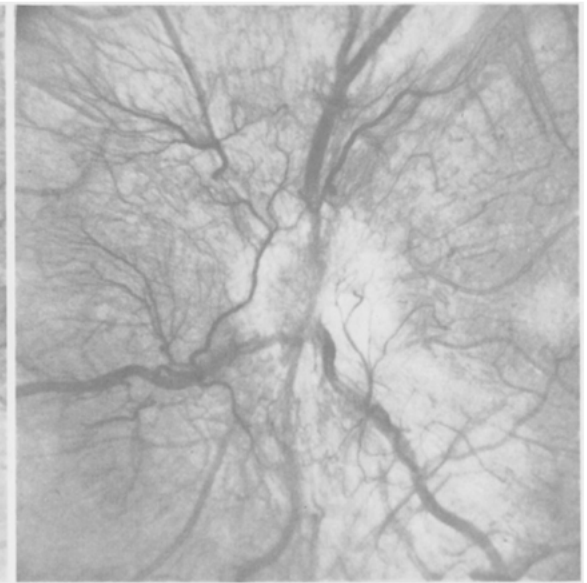
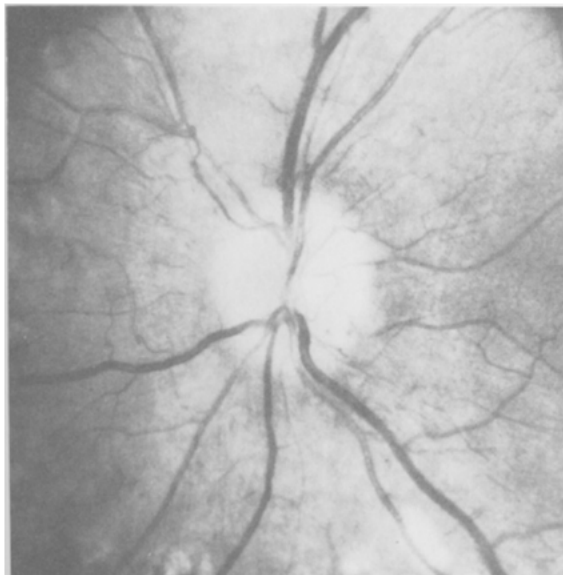


Fig 11E



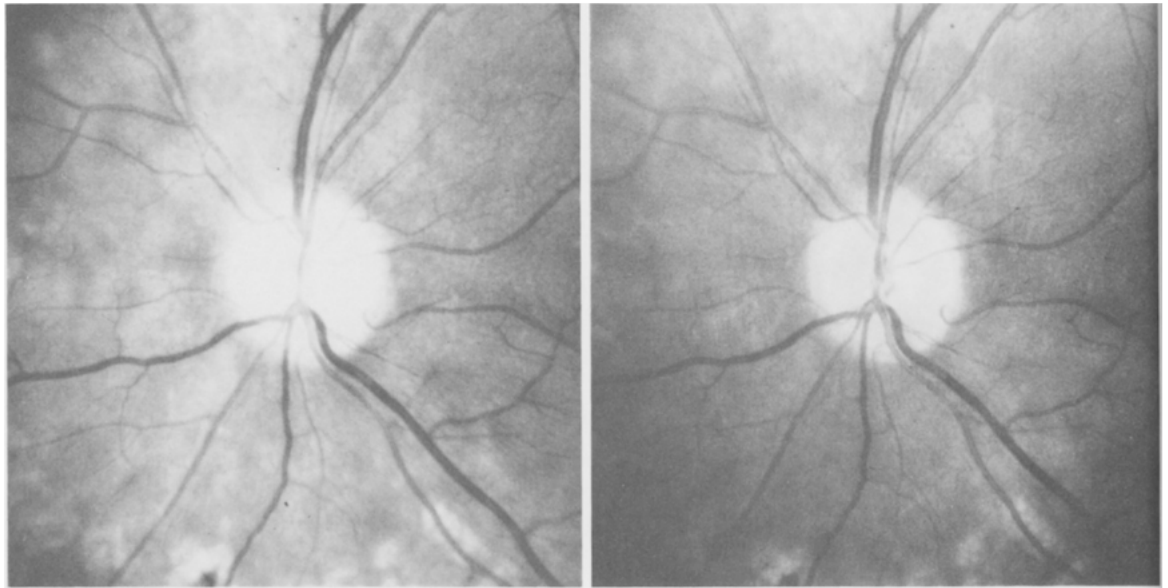


Fig 11F

Fig 11F. Left eye three years following photocoagulation. There is complete regression of new vessels.

REFERENCES

1. The Diabetic Retinopathy Study Research Group. Preliminary Report on Effects of Photocoagulation Therapy. *Am J Ophthalmol* 1976; 81:383-96.
2. The Diabetic Retinopathy Study Research Group. Photocoagulation Treatment of Proliferative Diabetic Retinopathy: The Second Report of Diabetic Retinopathy Study Findings. *Ophthalmology* 1978; 85:82-105.
3. The Diabetic Retinopathy Study Research Group. Photocoagulation Treatment of Proliferative Diabetic Retinopathy: A Short Report of Long Range Results. DRS Report #4. In: Proceedings, 10th Congress International Diabetes Federation. Amsterdam: Excerpta Medica, in press.
4. The Diabetic Retinopathy Study Research Group. Photocoagulation Treatment of Proliferative Diabetic Retinopathy: Relationship of Adverse Treatment Effects to Retinopathy Severity. DRS Report #5. *Dev. Ophthalmol*, in press.
5. The Diabetic Retinopathy Study Research Group. Baseline Monograph. DRS Report #6. *Invest Ophthalmol*, in press.
6. Littell AS. Estimation of the T-year survival rate from follow-up studies over a limited period of time. *Hum Biol* 1952; 24:87-116.
7. The Diabetic Retinopathy Study Research Group. A Modification of the Airlie House Classification of Diabetic Retinopathy. DRS Report #7 (to be published).