
SOME OF THE CHANGES IN DIABETIC RETINOPATHY ARE SIMILAR TO THOSE FOUND IN THE RETINA IN CERTAIN OTHER NEOVASCULAR DISEASES WHICH ARE BENEFITED BY LIGHT-COAGULATION. THESE INCLUDE LEBER'S ANEURYSMS, EALES'S DISEASE, AND ANGIOMATOSIS. SINCE IT HAS BEEN ESTABLISHED THAT LIGHT-COAGULATION CAN DESTROY CERTAIN VESSELS WITHIN THE EYE AND THAT FIRM CHORIO-RETINAL ADHESIONS CAN BE FORMED, IT WAS FELT THAT THIS TREATMENT MIGHT BE OF SOME VALUE IN REDUCING THE INCIDENCE OF RECURRENT INTRA-OCULAR HAEMORRHAGE AND DETACHMENT OF THE RETINA ASSOCIATED WITH THIS DISEASE.

**Material and Method**

This series consists of 28 patients (42 eyes) involving a total 117 treatments. There were seventeen women and eleven men, their ages ranging from 26 to 78 years. They all had advanced diabetic retinopathy as manifested by retinitis proliferans and a history of recurrent haemorrhage into the vitreous, and were referred by physicians, who continued their medical management while the light-coagulation therapy was being evaluated. The eyes were observed for from 12 to 43 months after treatment.

A pertinent medical history was taken, including the onset of diabetes, the onset of ocular symptoms, and the degree of control. Careful and complete physical examination of the eye was carried out to find the best correctable visual acuity and the visual fields when feasible, with slit-lamp examination, ophthalmoscopy, tonometry, and serial retinal photographs.

The light-coagulation was performed under local anaesthesia using 2 per cent. retrobulbar Xylocaine, the pupil being maximally dilated before each treatment. The amount of light delivered to the involved area was varied in each case according to what was felt to be the minimal reaction necessary to produce a mild retinal burn. This was altered somewhat depending upon the degree of abnormality of the lesion being treated. The light was directed to the geographic site of the retina showing the maximum diabetic involvement. An attempt was made to delimit the retinitis proliferans and to obliterate the neovascular tissues. Care was taken to avoid the macular and paramacular regions. Proliferation on the nerve head was not treated directly. Aneurysms and vessels suspected of recent haemorrhage were coagulated. In general, the course of the large vascular trunks where the proliferation or neovascularization was most prominent was followed, treatment being...
directed to either side of the main vessels. It was thought at first that it would be more objective, in bilateral cases, if only the more involved eye was treated. However, after one year, it was found that in three of these patients the treated eye was the only remaining seeing eye. In view of this, and because the involvement of each eye varies so much, this course was abandoned, and both eyes were treated when this appeared to be indicated. Each eye received an average of three treatments with intervals of 2 to 3 months between treatments. Three patients received only one treatment and one patient received eight. After the first treatment most patients were hospitalized for 4 to 7 days with rest in bed and binocular dressings.

At the follow-up examination the best correctable visual acuity and visual fields were determined, and ophthalmoscopic examinations made. Serial photographs were again taken in all cases.

Subjective symptoms, including awareness of peripheral field loss, scotomata, and photopsia, and evidence of recurrent bleeding into the vitreous, were also noted.

It was found that the intensity of light-coagulation used produced a well-circumscribed scotomatous defect rather than a sector defect in the visual field. This was demonstrated both by perimetry and by the after-image phenomenon. Most of the patients were not aware of any loss in the field of vision after treatment.

Complications.—These were infrequent, the chief being retinal haemorrhage, which occurred in one case immediately after treatment. Fortunately the bleeding was mild and cleared without permanent visual impairment. In a second case there was a delayed haemorrhage into the retina after 3 days, resulting in macular involvement with deterioration of central vision. The only other complication was direct involvement of the macular region by light-coagulation resulting in central visual loss in one patient.

Results

It is very difficult to evaluate the effectiveness of any treatment in a disease such as diabetic retinopathy which is known to undergo spontaneous remissions and exacerbations. It is fair to assume, however, that retinitis proliferans and neovascularization do not, as a rule, undergo spontaneous regression and that the overwhelming majority of patients with retinitis proliferans become blind in the course of 3 to 5 years.

The results were evaluated by dividing the course of diabetic retinopathy into three main categories:

1. Eyes in which the progress of the disease was "apparently arrested".
2. Eyes in which it was felt that treatment delayed the progression of the disease.
3. Eyes which showed no benefit from the treatment.

1. Six patients fulfilled the criteria for inclusion in the first category: stabilization of vision, obliteration or retardation of the progress of retinitis proliferans, absence of haemorrhage into the vitreous, disappearance of exudates, and diminished venous congestion and retinal oedema.

All were observed for a minimum of 31 months after treatment.

2. Seventeen patients formed a more nebulous group, in which the factor of spontaneous remissions had to be more strongly considered. They demonstrated
a slowing in the progression of the disease according to the criteria established in the first category.

(3) The third group of five patients showed no benefit, but demonstrated a rapid progressive loss of retinal integrity leading to blindness.

In Groups (2) and (3) ocular complications of diabetes other than retinopathy (including cataract formation, ruberosis iridis, and secondary glaucoma) played a role in the diminished visual acuity.

This series is too small and the length of follow-up too short to arrive at any statistical conclusions, but certain clinical observations merit further elaboration.

In some cases moderately elevated retinitis proliferans can be arrested, but the vessels projecting into the vitreous continue to be active and appear to be unaffected by the light, some of them remaining an apparent source of haemorrhage into the vitreous.

Flat retinitis proliferans can be completely obliterated. It is known that certain phases of diabetic retinopathy, such as the development of aneurysms, exudates, venous congestion, and retinal oedema are spontaneously reversible, but the clinical impression of the benefit of light-coagulation in these patients was determined by the fact that the condition was progressive for many months before treatment, and that soon after the treatment there was a remission.

This gives rise to many interesting theoretical speculations regarding the pathogenesis of diabetic retinopathy. It may lend some credence to the theory that the primary cause of the disease is retinal anoxia. Similar changes of retinal neovascularization and proliferation are found in retro-lental fibroplasia and also after central venous thrombosis. It would appear that the light-coagulation removes the stimulus for the progressive phase of diabetic retinopathy by the destruction of certain retinal elements. There may be a certain "local factor" in the development of this disease.

In conclusion, it has been observed that the course of diabetic retinopathy is somewhat similar to that of Leber's aneurysms, Eale's disease, and angiomatosis. The effect of light-coagulation on diabetic retinopathy is similar in many aspects to its effect on these other diseases. The course demonstrated by some of the patients in this series indicates that this treatment is of value in preserving vision in some cases and in delaying the course of the disease in others. In view of the minimal risk involved with proper technique, it is felt that this method deserves further study.

BIBLIOGRAPHY

TREATMENT OF DIABETIC RETINOPATHY BY LIGHT-COAGULATION: A PRELIMINARY STUDY
Paul C. Wetzig and James T. Worlton

Br J Ophthalmol 1963 47: 539-541
doi: 10.1136/bjo.47.9.539

Updated information and services can be found at:
http://bjo.bmj.com/content/47/9/539.citation

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/