Communications

Treatment of diabetic maculopathy

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The diabetic macula may be affected by the various elements of retinopathy—as observed in other locations in the retina—and the patterns here are similarly variable. The direct threat to the macular integrity starts with macular oedema which is reversible in the early stages, but invariably leads to failure of visual acuity in time. This oedema may be caused by the presence of diseased, fluorescein-leaking capillaries or new vessels in the immediate vicinity, or by a spreading front of retinal oedema reaching the macula from a more remote focus of retinal hypoxia. Hard exudates are deposited at the macula in this way, and they eventually affect the macular function by their bulk and by the destruction of retinal structure. They regress promptly as a rule when the hypoxic centre round which they form is converted into a scar by light coagulation, and these centres are commonly located at some distance from the macula itself. Haemorrhages and microaneurysms at the macula are the result of angiopathy and themselves present no danger. Soft exudates do not occur at the macula.

Clinical material

Among 110 diabetics treated by us during the past 2 years with light coagulation as a planned, prospective trial, forty were treated specifically for maculopathy (four bilaterally), making the number of eyes treated 44. There were 24 males and 16 females. The age distribution was 30–40 years two; 40–50 six; 50–60 eighteen; and over 60 fourteen.

Methods of treatment

The main cause of failing macular function in the early stages is macular oedema. Our approach to the treatment of this oedema was based on a hypothesis (Rubinstein and Myska, 1971) that elimination of diseased and new bloodvessels surrounding a hypoxic focus in the vicinity of the macula would restore the local as well as the macular metabolism to normal with regression of oedema as well as of secondary hard deposits. It was accepted that an anatomically damaged macula could not recover its function but, as this function fails gradually, it could perhaps be arrested at any particular level by elimination of the focus of disturbed metabolism. We also kept in mind reported suggestions that elimination of parts of the retinal tissue would lessen the circulatory requirement of the retina as a whole, thus allowing better perfusion of the vital area of the macula.

Table I sets out the five possible light coagulation approaches to the macula proposed by various workers at one time or another and Fig. 1 depicts the five sites of application; of these the last, peripheral pattern bombing, has not yet been practised here, as it seems the least likely to succeed. Table I also shows the number of applications of each of these methods in the series; the relatively small number of paramacular applications (raphe) is explained by our efforts to put the perimacular approach to the test first.

Table I *Methods of treatment and numbers of eyes*

<i>Method</i>			<i>No. of eyes</i>
(1)	I Direct	(a) Centres of circinate rings	15
(2)		(b) Leaking bloodvessels (including neovascularization)	2
(3)	II Indirect	(a) Perimacular	18
(4)		(b) Paramacular (raphe)	9
(5)		(c) Peripheral pattern bombing	0
Total			44 (40 patients)

FIG. 1 *A scheme of five photocoagulation approaches to diabetic maculopathy. Numbering as in Table I*

The choice of approach was determined by the pattern of retinopathy. Whenever possible the direct methods of application were practised, and these were found relatively most effective. Where macular and central changes were gross or so near to the fovea that the direct approach would be likely to damage foveal function, the perimacular and paramacular approach was practised. All cases had fluorescein angiography studies before the right procedure was decided upon; this we considered to be an essential guide, particularly in relation to the direct approaches. The procedure was performed after a retrobulbar injection of 3-4 ml. xylocaine (without adrenalin). As a rule the worse eye was treated leaving the other eye as control. Several patients, however, had only one useful eye, the other being already blind through diabetic retinopathy or having severely diminished central vision (*e.g.* to 6/60 or less) through macular change or lens opacities. Throughout, the Zeiss (Meyer-Schwickerath) light coagulator was used, with aperture 3, as aperture 1.5 proved to give too limited a field of vision for safety around the macula. The applications were intended to be as light as possible with just a suggestion of blanching of the retina or sometimes even without its being visible at the time of application; 2 seconds' application with Black 1 or 2 control was commonly used even when applied to oedematous areas.

Complications

In no case were we confronted with postoperative haemorrhage, retinal gliosis, or late macular pigmentation. In fact, no treated eye was found to see less well during the period of observation (6 months to 2 years). When applications were very close to the macula, there was, very rarely, a diminution of vision by one line for the first few days because of reactive oedema. The influence of the procedure on the visual fields seemed to be different when assessed subjectively and when assessed objectively by field plotting. In only three cases had we complaints of scotoma, which persisted in the worst case for 6 weeks (Fig. 2).

FIG. 2 Direct approach close to the macula. Patient's drawing of scotomata giving a precise reciprocity with the pattern of photocoagulated foci

Objectively, however, central field changes could be detected in every case (by Goldmann perimetry) and the scotomata were a mixture of absolute and relative density, and of patterns reciprocal to the light coagulation scarring (Fig. 3). Some eyes treated at the beginning of this series, when our applications were stronger, did show wedge scotomata indicating conduction fibre damage (Fig. 4). To assess these field changes it is necessary to compare the postoperative and preoperative fields: areas of hypoxia cause isolated scotomata in the visual fields and unless the preoperative state of the fields is known the assessment of postoperative fields is meaningless.

Results

None of the 44 eyes in this series showed worsening of visual acuity in the treated eye; twelve eyes improved by two lines or more, and eleven eyes improved by one line. In many patients with maculopathy the visual acuity fluctuates at successive tests and improvement by one line was accepted only if it was repeatedly obtained for a period of time. There were four cases which improved by 4 and 3 lines, and 21 eyes retained the same visual acuity (Table II).

Of the untreated—preoperatively better—eyes, eleven showed worsening of visual acuity during the period of observation, twelve remained the same, and eight improved by one line (Table II).

Table II *Visual results in treated and untreated eyes*

<i>Method of treatment</i>	<i>Visual acuity</i>						
	<i>Treated eyes (44)</i>				<i>Untreated eyes* (31)</i>		
	<i>Improved</i>				<i>Improved</i>	<i>Same</i>	<i>Worse</i>
	<i>2 lines or more</i>	<i>1 line</i>	<i>Same</i>	<i>Worse</i>			
(1) Centres of circinate rings	4	2	9	0	4	8	1
(2) Leaking bloodvessels	0	1	1	0	0	0	0
(3) Perimacular	3	7	8	0	4	2	7
(4) Paramacular	5	1	3	0	0	2	3
Total	12	11	21	0	8	12	11

* In five cases the untreated eye had a visual acuity of less than 6/60 and these cases are not included

Regarding the effectiveness of each particular approach pattern, Table II should be compared with Table III. The preoperative visual acuity in cases suitable for direct approach is seen here to be relatively better, pointing to less severe macular damage, and the postoperative visual acuity is also better in this group than in the others.

Table III *Preoperative visual acuity of 44 treated eyes*

<i>Method of treatment</i>	6/5	6/6	6/9	6/12	6/18	6/24	6/36	6/60	<i>Total</i>
(1) Circinate rings	1	4	2		5	1	1	1	15
(2) Leaking vessels			1			1			2
(3) Perimacular		1	3	1	5	1	2	5	18
(4) Paramacular			3	3	1		1	1	9
Total eyes	1	5	9	4	11	3	4	7	44

The perimacular approach was used in cases of heavily affected maculae with a consequently poorer response; paramacular application in similar cases seemed to be more effective, perhaps because it meant in effect a partially direct approach by coming much closer to the affected macula.

Conclusions

Light coagulation of diabetic maculopathy seems to be effective in improving local metabolism at the macula in a percentage of cases high enough to encourage continuation of these trials. Light coagulation of diabetic retinopathy is being increasingly used, especially in Great Britain, Germany, and the United States, but reports on this work are few. Regarding maculopathy the only report available is that of Spalter (1971), which describes the results of the treatment of hard exudates involving the macula in twenty patients, of whom 50 per cent. improved by 1 to 4 lines, 30 per cent. showed no change, and 20 per cent. showed further deterioration. Spalter advocates heavy light coagulation to the centres of circinate rings. A few other reports (Meyer-Schwickerath and Schott, 1968; Beetham, Aiello, Balodimos, and Koncz, 1970; Irvine and Norton, 1971) obviously include maculopathy problems but these are not specified and no statistics are given. In competent hands and with preoperative assessment by fluorescein angiography, light coagulation is a safe procedure, and its results can be judged by recording visual acuity only, making assessment relatively simple. The treatment is best used prophylactically at the stage when the macular changes are early and visual acuity still good, but macular oedema or early hard exudates threaten the vision. In cases in which advanced damage to the macula exists—apart from oedema itself—it is futile to expect improvement of vision, but it seems that in some cases at least visual deterioration can be arrested.

References

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