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Central visual field in diabetes

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Little detailed work has been reported on the central visual field in diabetes. Scott (1957) stated that "the causation of scotomata by endogenic toxins is extremely rare"; Dubois-Poulsen (1952) reported a study of diabetic patients using rod scotometry (Livingston, 1943) in which central field scotomata were found in eyes with no visible retinopathy. The original reference to diabetic patients has not been traced (Livingston, personal communication). King, Dobree, Kok, Foulds, and Dangerfield (1963), during trial of a corn oil diet, found central field scotomata, using the Bjerrum screen, in diabetic patients with exudative retinopathy, which persisted after the exudates had cleared. Lakowski and Aspinal (1966) mentioned three types of defects in diabetic visual fields, found by static methods using the Goldmann perimeter. These defects were a general loss of sensitivity, peripheral field losses, and central field losses; it is not clear whether retinopathy was present or not.

The present paper reports the investigation of central visual fields of diabetic patients with and without retinopathy, using a new central field scotometer (Roth, 1968).

Methods

Patients were selected as follows:

Non-diabetic controls

(1) no history or past suspicion of diabetes;
(2) no history or ocular signs of any disease likely to cause visual field defects;
(3) visual acuity in the eye(s) to be tested better than 6/9 (6/12 pt.) corrected.

Diabetics

(1) known diabetics attending the Diabetic Clinic at the Radcliffe Infirmary, Oxford, or referred for investigation from the Hammersmith Hospital, London;
(2) as for the Control Group, except that diabetic patients with visible retinopathy were included;
(3) patients with visual acuity better than 6/9 (6/12 pt.) corrected if there was no retinopathy, and better than 6/12 (6/18 pt.) corrected if retinopathy was present.

All patients with obvious opacities of the media were excluded.

Visual acuity was tested with a 6 metre Snellen chart, distance correction being worn when required. Diabetic eyes were examined with dilated pupils (Cyclopentolate 1 per cent.), using polychromatic light, at least 3 days before the visual field tests. The presence or absence of visible retinopathy was then noted and fundus photographs were taken (Kowa RC and Kodachrome 2 film). The ocular media were examined with an ophthalmoscope.

The apparatus used for visual field testing was designed for this project (Roth, 1968) to investigate the central 20° field at a test distance of 1 metre.

Patients were given from 10 to 20 minutes to adapt to the dim lighting conditions of the test (about 1 foot candle reflected from the screen). Distance correction, if necessary, was worn for the test;

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care was taken that spectacle frames and bifocal segments did not obstruct the patient's view of the screen (Chamlin, 1947).

Test objects were moved centrifugally from fixation and from areas of vision towards blind areas. The patient signalled the disappearance of the test object by pressing a bell-push which sounded a buzzer.

When a scotoma was found its limits were explored by approaching the test object to it from at least four different directions, starting each exploratory run from the fixation area. The same three test objects were used whenever possible:

(1) 3·5 mm. diam., white, at maximum contrast.
(2) 1·0 mm. diam., white, at maximum contrast.
(3) 1·0 mm. diam., white, at half maximum contrast.

Patients with severe retinopathy who could see none of these test objects were investigated with larger targets of uncontrolled high contrast projected by an ophthalmoscope.

Object (1) was employed in at least 14 meridia, object (2) in at least 24 meridia, and object (3) in at least 12, when seen. Nine of eighteen control and diabetic patients over the age of 60 were unable to see this test object at all.

A scotoma was recorded if the defect found showed at least four distinct, separate points on its circumference; it could not be traced back to the physiological blind spot as an extension from it, *i.e.* was not part of the angioscotoma; it could be approached from any direction and shown to be a defect distinct from the isoptre within the boundaries of which it was found, therefore irregularities in an isoptre were not considered as scotomata. Patients were included in the group to be referred to later as "with scotomata" if at least one scotoma was found which met the specifications above.

The time needed to perform the test on one eye varied from 15 to 20 minutes if no scotoma was found, and up to 45 minutes if scotomata were present.

If the patient complained of symptoms of fatigue during the test (photopsia or watering eyes), he was allowed to rest until the symptoms had subsided.

Certain errors and inconsistencies are well known in the results of testing visual fields with mobile test objects. These should be borne in mind when evaluating the results of this experiment. Errors may arise from the apparatus, the patient, and the examiner.

The apparatus was designed to incorporate as many standardizable features as possible, so that all patients would be subjected to the same conditions of test (Thomasson, 1934). The following features are standardized: background illumination, by the photometric extinction of a standard light source; contrast between test objects and screen, achieved by the same method; test distance fixed at 1 metre by a chin rest and headband, the limbus of the eye under test is aligned with a fixed point 1 metre from the screen; test environment—the entire visual field of the eye under test is contained by the apparatus; alignment with the fixation point, the fixation device cannot be seen correctly unless the eye is in line with the intersection of the horizontal and vertical meridia of the screen. All patients therefore were subjected to the same test conditions within the apparatus.

The patient may be the source of two main errors, arising from unsteady fixation (Evans, 1936) or delay in reporting the disappearance of the test object (Gradle and Meyer, 1929). These errors may be reduced by ensuring that the patient understands the nature of his task (Darley, 1950). A careful and detailed explanation of the test was given to each patient and a short practice attempt was made when necessary. Fixation was monitored by the repeated investigation of the inner edge of the blind spot on the horizontal meridian.

Some delay in reporting the disappearance of the test object was inevitable; this varied from patient to patient. It was therefore necessary for the examiner to adapt the rate of movement of the test object to the speed with which the patient could report its loss; the correct rate of progress could be determined from the practice run. A rate of test object progress of about 1° per second suited most patients.

A subsidiary experiment was introduced about half way through the study in order to determine the effects of delay in reporting the disappearance of the test object.

2″ squares were cut from graph paper calibrated in tenths of an inch, so that each large square contained 400 small squares. During a routine investigation one of these pieces of graph paper was stuck on to the operator's side of the screen between the 10° and 15° calibrations. The paper was opaque enough to prevent light from the test object projector passing through it to the screen and thus acted as an artificial scotoma of known dimensions.

The 1 mm. test object was brought to the artificial scotoma along extensions of its diagonals, so crossing the paper at the corners. As the test object crossed a corner it passed from the patient's view. It progressed in the same direction and at the same rate until the patient sounded the buzzer; the paper was marked at the point at which the test object was lying when the buzzer sounded. Each corner of the artificial scotoma was approached so that at the end of the test there were four points marked on it. The paper was detached from the screen and the four points were connected to make a four-sided figure (Fig. 1). The following measurements were then made. The number of small squares within the figure was counted and compared with the total number of squares on the paper; this gave a rough comparison between the area of the scotoma found with the area of the scotoma actually present. The distance the test object had travelled across the paper before its loss was reported was measured in millimetres; the mean of the four measurements was recorded.

Table I shows that most patients were capable of reporting between 3/8 and 5/8 of the area of an artificial scotoma of this size, and that most patients reported losing sight of the test object before it had travelled more than 11 mm. beyond the point at which it actually vanished. The artificial scotoma was in an area where 11 mm. represents approximately 0·5°; therefore most patients could detect scotomata of about 0·5° in diameter, and all could detect scotomata of 1° in diameter. This suggests that the test was satisfactorily performed by all patients.

Table I *Results of reliability test*

Number of patients tested	31
Number of eyes tested	59
Actual area of artificial scotoma (squares)	400
Largest scotoma reported	375
Smallest scotoma reported	110
Mean scotoma reported	213
Standard deviation	±54
Minimum distance object travelled before loss reported (mean of four points)	3·0 mm.
Maximum distance (mean)	16·5 mm.
Overall mean performance	7·8 mm.
Standard deviation	±3·0

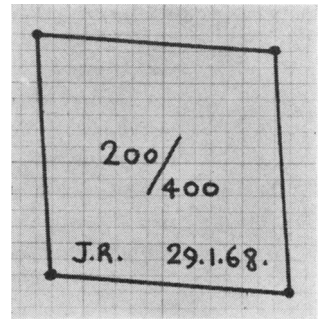


FIG. 1 *Artificial scotoma*

Results

There were no isolated scotomata found in the control group. All scotomata found could be linked and shown to be parts of the angioscotomata.

The scotomata found in patients with little or no retinopathy were relative, and could be detected only with small or dim test objects (Fig. 2). The visual fields of both groups of patients were similar. The scotomata were rarely more than 3° in maximum diameter. There were rarely more than five scotomata in each eye. Under these conditions it was possible to explore the entire 20° field.

When retinopathy was severe (see Fig. 3) the scotomata were usually absolute to test objects of 1 cm. diameter displayed at high intensity. The scotomata were usually so numerous as to make it impracticable to explore more than a small area of the visual field. Under these circumstances it was necessary to confine the examination to the central 5°. Patients with severe retinopathy could not see the smaller, dimmer standardized test objects at all.

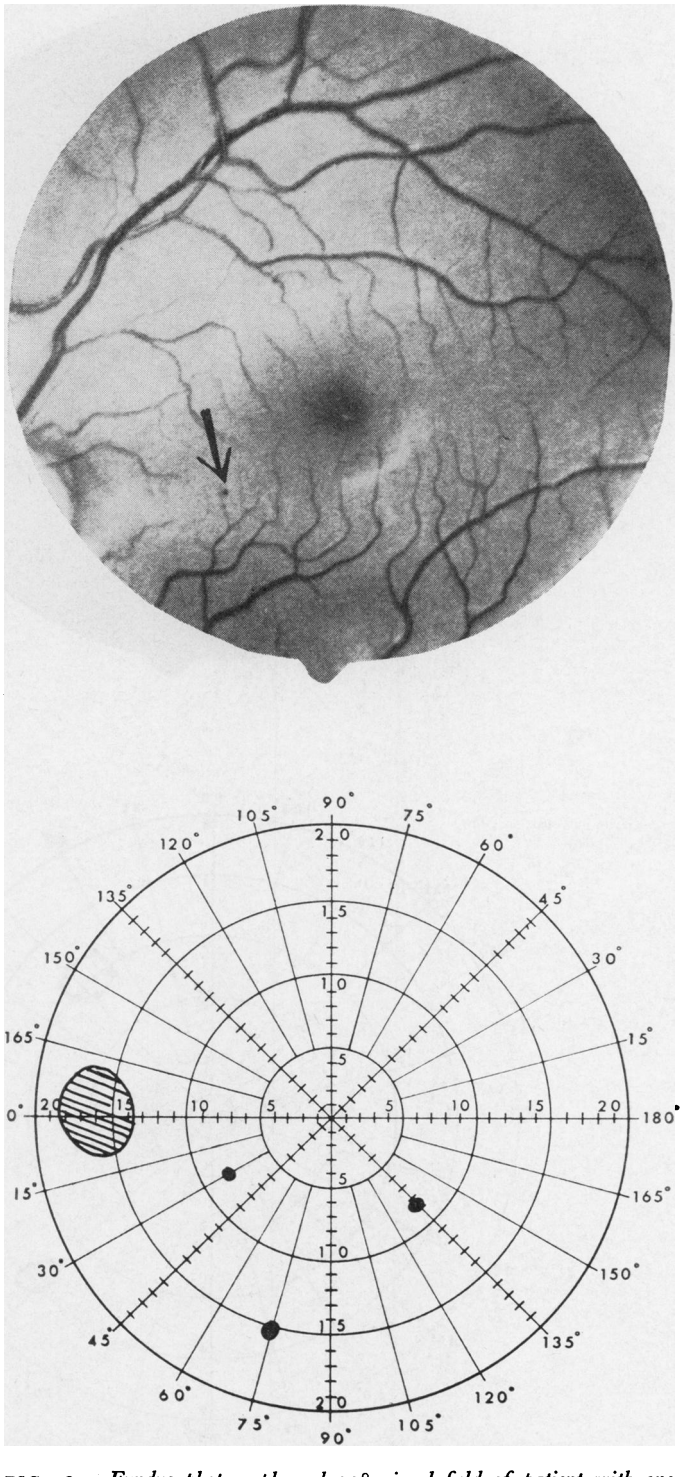
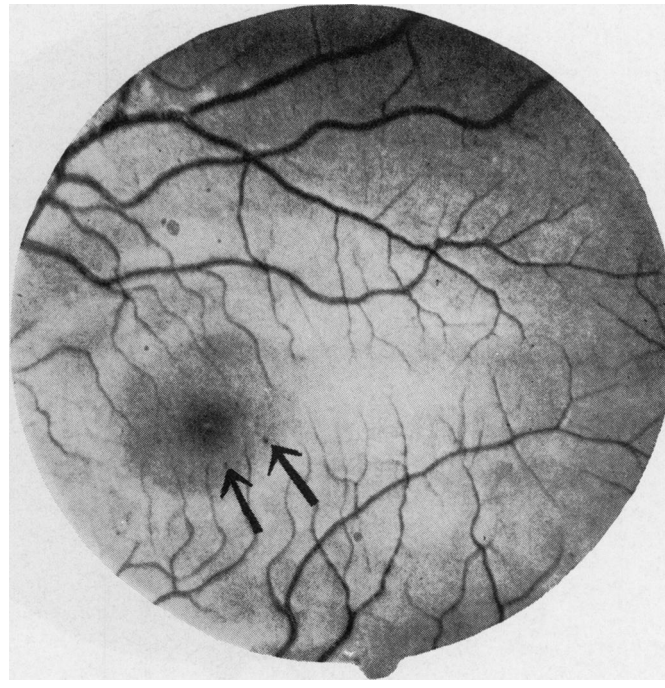


FIG. 2a *Fundus photograph and 20° visual field of patient with one microaneurysm (isoptres omitted)*



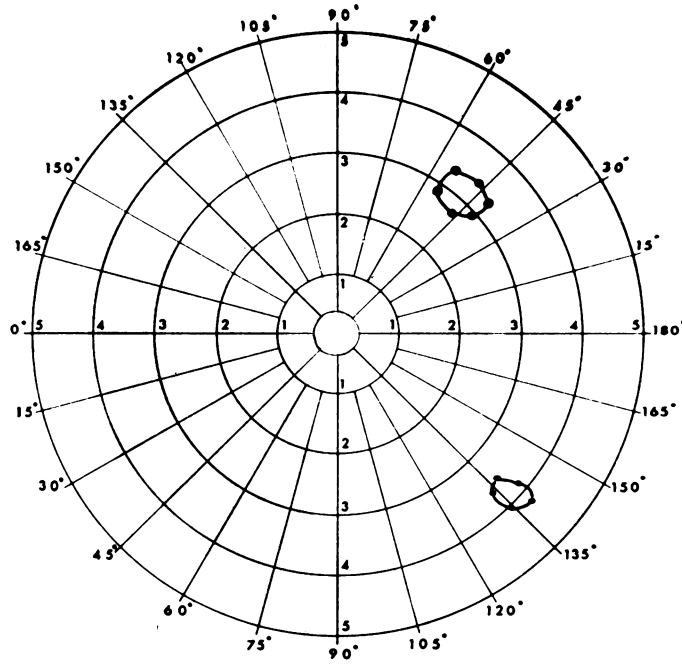


FIG. 2b *Fundus photograph and* 5° *visual field of same patient as Fig.* 2a, 4 *weeks later (isoptres omitted)*

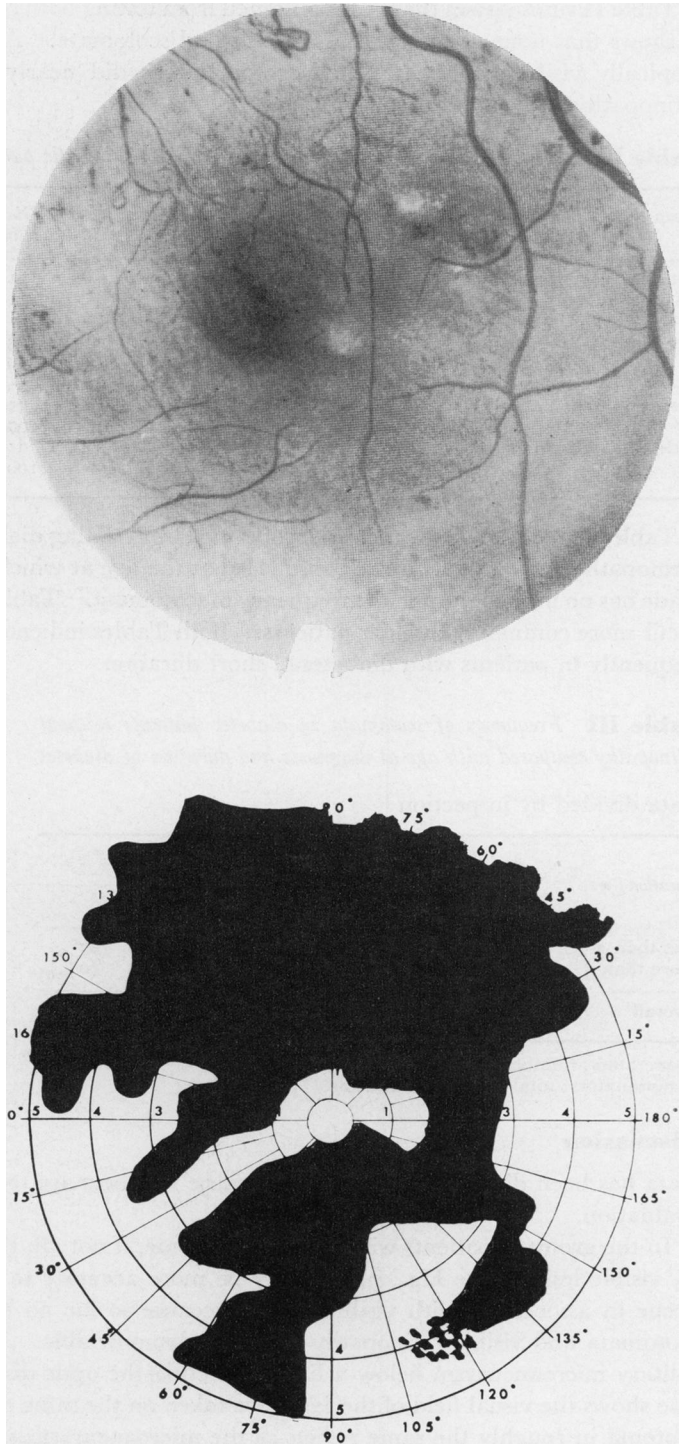


FIG. 3 *Fundus photograph and 5° visual field of patient with severe diabetic retinopathy*

Table II summarizes the results obtained from testing both control and diabetic patients. It shows that none of the control patients had scotomata. All patients with ophthalmoscopically visible retinopathy had scotomata, as did nearly half those without visible retinopathy.

Table II *Summary of results obtained from control and diabetic patients*

Group		*Non-diabetic*	*Diabetic with retinopathy*	*Diabetic without retinopathy*
Total no. of patients		60	21	66
No. of eyes tested		110	31	132
Age (yrs) Youngest		18	23	13
Oldest		74	69	73
Eyes with scotomata	No.	0	31	52
	Per cent.	0	100	39·4
Patients with scotomata	No.	0	21	32
	Per cent.	0	100	48·5

Tables III and IV show the results obtained from testing diabetic patients without visible retinopathy. It appears from Table III that the age at which the diagnosis of diabetes is made has no influence upon the frequency of scotomata. Table IV suggests that scotomata occur more commonly in older patients. Both Tables indicate that scotomata occur more frequently in patients with diabetes of short duration.

Table III *Frequency of scotomata in diabetic patients without retinopathy compared with age at diagnosis and duration of diabetes*

Data divided by inspection

Duration (yrs)	*Age at diagnosis (yrs)*					
	0–19		20–59		*Over* 60	
Less than 5	5/10	(50%)	12/20	(60%)	4/6	(67%)
More than 5	7/15	(47%)	4/13	(31%)	0/2	(0%)
Overall	12/25	(48%)	16/33	(48%)	4/8	(50%)

Numerators: number of patients with scotomata
Denominators: total number of patients

Discussion

Data has been divided by inspection because numbers are too small for proper statistical evaluation.

In the group of patients with retinopathy some, if not all, the scotomata may be caused by visible lesions (see Fig. 2). It may be more accurate to say that scotomata seem to occur in association with visible lesions because so far no precise relationship between scotomata and visible retinopathy has been demonstrable. Fig. 2*a* shows an eye with a solitary microaneurysm below a line connecting the optic disc with the macula. Fig. 2*a* also shows the visual field of the same eye taken on the same day. The field map shows a scotoma in roughly the same region as the microaneurysm and two other scotomata not related to visible lesions. Fig. 2*b* shows the macular region and the field of the same eye 4 months later. The solitary microaneurysm has disappeared; there is now a cluster of

Table IV *Frequency of scotomata in diabetic patients with retinopathy compared with age at test and duration of diabetes*

Data divided by inspection

Duration (yrs)	*Age at test (yrs)*					
	0–19		20–59		*Over* 60	
Less than 5	4/9	(44%)	12/20	(60%)	5/7	(71%)
More than 5	3/10	(30%)	6/13	(46%)	2/7	(28%)
Overall	7/19	(37%)	18/33	(55%)	7/14	(50%)

Numerators: number of patients with scotomata
Denominators: total number of patients

microaneurysms close to the macula. The visual field map (central 5° field only) shows two scotomata close to the fixation point. No other scotomata were found in the central 20° field. These comparisons suggest that scotomata and visible lesions may be related; the same approximate relationship was found in most other eyes with visible retinopathy.

It has not yet been possible to demonstrate a precise relationship between visible lesions and scotomata, probably because of distortions imposed on the final picture by the lens systems of the camera and enlarger. This problem is under investigation.

It is more difficult to account for visual field defects found in diabetic eyes without retinopathy. Oosterhuis and Lammens (1965), using fluorescence retinal photography, found areas of capillary closure in the retinal circulation of diabetics without retinopathy. Kohner (1968), using fluorescence retinal photography, also found areas of capillary closure in patients with long-standing diabetes but no visible retinopathy. The relationship between capillary closure and scotomata is being investigated by Kohner and Roth (in preparation).

Defective circulation in the retina may lead to defective function because of local anoxia. Livingston (quoted by Dubois-Poulsen, 1952) thought the defects he discovered in the central visual fields of diabetic eyes were similar to those found in the lighter degrees of anoxia.

Kohner and Roth (in preparation) are trying to determine whether the postulated relationship between retinal capillary circulation and scotomata does in fact exist. Preliminary results are encouraging; demonstration of an approximate geographical relation between the two phenomena has sometimes been achieved. To establish a precise relationship it is necessary first to solve the photographic problem mentioned previously.

The distribution of the data in Tables III and IV must be accounted for. This may be possible if the assumption is made that scotomata represent a form of preretinopathy. Henkes and Houtsmuller (1965) defined preretinopathy as a state in which there are alterations in the retinal function in diabetic eyes when the fundus appearance is normal; they found altered electrical characteristics in the diabetic eye without retinopathy. Electroretinography is an "all or none" phenomenon. It may reflect altered metabolism in the diabetic retina as a whole. Scotometry is more selective and can detect alterations in function of small areas of retina.

The frequency of diabetic retinopathy increases with age (Caird, 1967; Howells, 1953; Kornerup, 1957) and with the duration of the disease (Post and Stickle, 1950; Caird and Garrett, 1962), but the age at which the diagnosis of diabetes is made is not important (Walker, 1950).

If scotomata indicate preretinopathy, it would be understandable that the age at diagnosis of diabetes appears not to influence the incidence of scotomata, and this may also account for the fact that scotomata are seen more commonly in older patients, when the age at which the test was performed is considered (Table IV).

Patients shown in Tables III and IV were selected because they had no retinopathy. It is likely, therefore, that an increasingly greater proportion of available diabetic patients has been excluded in the upper age groups. Maximum exclusion will have occurred in the oldest age group with the longest duration of diabetes.

From Tables III and IV it appears that patients with diabetes of short duration may have a higher frequency of scotomata than patients with diabetes of longer duration. Patients diagnosed under the age of 20 years show an insignificant difference in the frequency of scotomata between the two "duration" groups. This suggests that patients whose diabetes has been diagnosed at an early age may progress in three ways; they may stay as they are, with or without scotomata; they may "improve" and lose their scotomata, or they may "deteriorate" and develop visible retinopathy. Both age and the duration of the disease increase with time and an increasing proportion of patients develops retinopathy. Thus, in middle age, larger numbers of patients are excluded in the selection for this test. More patients are likely to develop retinopathy, therefore more have preretinopathy. Patients having had diabetes for more than 5 years without developing retinopathy are presumably those in whom it is comparatively unlikely to develop. In old age this trend is exaggerated; a still higher proportion of patients have preretinopathy, and the longer-duration patients who have remained without retinopathy are unlikely to develop it.

It thus seems that available data may indicate that scotomata are a form of preretinopathy.

To confirm this theory these patients should be followed up over a period of time to see whether in fact those with scotomata develop retinopathy more frequently than those with no visual field defect.

The follow-up study is just beginning. Sixteen patients without retinopathy have been seen at 3-monthly intervals for 9 months. During this period two patients have developed visible retinopathy (a few microaneurysms in both cases). Both had scotomata which were found at their first attendance; 3 months before the retinopathy appeared both showed a sharp increase in the total number of scotomata found.

The follow-up study will be continued and enlarged. It is hoped that at least fifty patients will be observed for at least one year. A group of patients without scotomata will be observed simultaneously in order to determine whether retinopathy can appear in the absence of scotomata.

Conclusions

Scotomata occur in many diabetic eyes without visible retinopathy; it is necessary therefore to find the physical change in the retina responsible for its altered function. The suggestion that scotomata represent a state of preretinopathy has been put forward and requires substantiation. If this can be done, then scotometry could be a useful screening test for retinopathy. Scotometry could also be used to determine the efficacy of a particular treatment in a clinical trial.

Tests of visual acuity are sometimes used as a method of assessing the effects on retinal function of treatment of diabetic retinopathy (*e.g.* Cullen, Ireland, and Oliver, 1964), but these only indicate the degree of macular involvement. Scotometry can be used to

determine the state of function of a larger area of the retina. The main disadvantage of the technique described here is the amount of time it takes.

A larger number of patients must be investigated, as described, to amass sufficient data for statistical analysis. This project is in hand. The aim is to provide enough information for conclusions to be reached about the incidence of scotomata in patients with varying degrees of diabetic control.

Summary

A new apparatus was used to investigate the central visual fields of diabetic patients with and without retinopathy; a group of non-diabetic controls was also investigated. All diabetic eyes with retinopathy showed central visual field defects, some of which could be related to visible lesions; approximately half the diabetic patients without retinopathy also had visual field defects; no visual field defects were found in the control group. The methods used to investigate the visual fields have been considered and the conclusion reached that all patients studied performed the test satisfactorily. The suggestion has been made that scotomata in diabetic eyes without retinopathy represent preretinopathy and that scotomata may also be related to defects in the retinal capillary circulation. If the relationship between scotomata, visible lesions, and areas of capillary circulation defect can be established, then scotometry may provide a useful screening test of possible retinopathy.

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