COMMUNICATIONS

EFFECTS OF TRANSIENT INDUCED ELEVATION OF THE INTRA-OCULAR PRESSURE ON THE VISUAL FIELD*

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THE cavernous atrophy of the optic nervehead and the visual field loss characteristic of the progressive glaucomatous process are thought to be due to an upset of balance between the intra-ocular pressure (I.O.P.) and the arteriocalillary pressure in the network supplying the optic nervehead. Which of these factors is primarily or predominantly responsible for this imbalance and for the pathogenesis of the impaired visual function is still under dispute.

Clinically the degree of loss of visual field does not vary proportionally with the level of the I.O.P. in each case. Furthermore, the factors known to influence the glaucomatous process, such as the age of the patient or the state of the systemic and retinal circulation, do not explain why some eyes continue to lose visual field with a low tension and almost normal hydrodynamic conditions, while others can withstand a high pressure with little or no damage. This fact became evident soon after the application of tonometry, and the "low tension" glaucoma described as a separate clinical entity is to-day accepted as no more than an extreme example of a high sensitivity of the optic nerve to the I.O.P., so that even a statistically "normal" tension produces damage to the optic nerve. The most valid concept relating these facts is that the primary event in chronic simple glaucoma is a functional vascular dyscrasia of the capillary bed of the eye as a whole, leading to structural changes in the vessel walls and to tissue sclerosis. This process affects in different degree the structures responsible for the production and outflow of the aqueous humour as well as the optic nerve. Therefore if the complication of a raised I.O.P. is added to the sclerosis of the optic nervehead, excavation of the optic disc will run on apace (Duke-Elder, 1957, 1962). Studies focused especially on the circulation of the optic nervehead led to the following more or less widely accepted conclusions:

(1) Anatomically, François and Neetens (1954) have shown that the lesion producing the glaucomatous field defect lies in the vascular system of the anterior part of the optic nerve and that its extent is related to the individual variations in the anatomical disposition of the nutrient vessels.

(2) Gafner and Goldmann (1955) assumed that the rate of blood flow in the lamina cribrosa was especially sensitive to I.O.P. changes and that the effect of diminution of the pressure in the capillaries supplying this region was the same as the effect of an increase in the I.O.P.

(3) From the clinical point of view, ophthalmodynamometry, although used extensively, cannot be considered as a useful method for the investigation of the circulation of the optic

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nervehead region (Bronner, 1959; Lobstein, 1959; Weigelin and Lobstein, 1963); on the other hand, if the capillary circulation of the optic nervehead is considered as part of the systemic circulation, then it is reasonable to assume that the systemic blood pressure must play a major role in maintaining the gradient I.O.P.—capillary pressure within safe limits, and this has been shown to be true in individual cases (Harrington, 1959). Moreover, Broadfoot, Gloster, and Greaves (1961), by photo-electric analysis of light returning from the fundus of the eye, found that during compression of the globe the blood volume in the choroidal vessels was reduced. This experimental evidence can be applied quite reasonably to the vascular network of the optic nervehead, being in accordance with the generally-accepted views on the mechanical influence of raised I.O.P. upon the intra-ocular structures.

With these concepts in mind, the question now arises whether functional tests done during an artificial increase in the I.O.P. could permit an evaluation of the "susceptibility" of a given eye to the I.O.P.—a parameter of the utmost clinical importance in glaucoma.

The authors who have engaged in this study so far have approached the problem in different ways, although all have used provocative tests designed to show alterations in visual function during an artificially increased I.O.P. Thus Samojloff (1923, 1924, 1925) compared Bjerrum screen campimetry findings in glaucomatous eyes on and off pilocarpine drops and during the water-drinking test and in healthy eyes before and after a subconjunctival injection of hypertonic saline that produced a rise of I.O.P. His findings were that, in healthy eyes during the increased I.O.P., an enlargement of the blind spot or even a Seidel type of scotoma was produced, while in the glaucomatous eyes the existing scotomata were increased. On the basis of these findings he assumed that hypertension of the eye is the principal cause of the appearance or the increase of the glaucomatous arcuate scotomata.

Bailliant (1930), using his ophthalmodynamometer, exerted a compression of the eyeball in healthy subjects, raising the I.O.P. to the level of the diastolic pressure of the central retinal artery. He noticed that there was a slight nasal constriction of the visual field immediately after the application of compression, and that a considerable diminution of the visual acuity was recorded soon after.

Gafner and Goldmann (1955), in cases of early chronic simple glaucoma without disturbance of the visual field, found a diminution of the sensitivity in the Bjerrum area by methods such as skiascotometry and quantitative perimetry. In a series of normal eyes, compression of the eyeball through the upper lid by the ophthalmodynamometer of Bailliant produced the same reaction; the level of the I.O.P. at the moment when the first demonstrable disturbance happened was correlated with the pressure in the ophthalmic artery.

Harrington (1961), in an effort to establish a ratio or gradient which might be of use in measuring the individual sensitivity to the I.O.P., developed the following technique:

The patient fixed the centre of a Bjerrum screen and indicated the time of disappearance of a white target placed in the Bjerrum area, the I.O.P. being increased at a fairly even rate by application of an ophthalmodynamometer. The force in grammes exerted at the time of disappearance of the target was a measure of the sensitivity in this particular area of the visual field. Also, the force required to produce disappearance of the visual stimulus was correlated with the I.O.P. and with the pressure of the central retinal artery. The results

indicated individual variations in sensitivity to pressure as far as the visual fields were concerned.

Drance (1962a, b), after mydriasis and instillation of local anaesthetic to the eye, raised the I.O.P. by a modification of the Kukan suction dynamometer attached to the lateral side of the eyeball. The level of I.O.P. was recorded when the patient indicated the disappearance of a white target placed in the upper or lower Bjerrum area; then, in a further step, the diastolic pressure of the central retinal artery was determined under the same level of I.O.P. and the brachial blood pressure was recorded. This study was carried out on healthy, glaucomatous, and suspected glaucomatous eyes. The author found that the more advanced the disease the more sensitive was the eye to the artificially-raised I.O.P.; the same reaction was produced in cases of incipient glaucoma as well as in the healthy eyes, the only difference being the level of I.O.P. necessary to evoke the disappearance of a given target.

Lynn (1962) increased the I.O.P. of normal eyes by introducing air under compression in a gas-mask firmly attached round the orbit; using the Goldmann perimeter he noticed a slight constriction of the peripheral isopters and an enlargement of the blind spot.

The purpose of the present paper is to contribute further to the study of variations in the sensitivity of the pericentral visual field produced during an artificial increase in the I.O.P. in a series of healthy and diseased eyes and to discuss some individual cases.

In this experimental study the I.O.P. was increased by compression of the eyeball through the lower eyelid. Perimetric examination was done by two methods:

- (1) Static or light-sense perimetry, in which a number of illuminated points were presented in the visual field (Weekers and Lavergne, 1958);
- (2) Kinetic perimetry, in which the usual method of determination of isopters for given moving targets was used.

The investigation was carried out in two parts: (a) the findings in glaucomatous eyes were compared with those in suspected glaucomatous and healthy eyes, and (b) the study was extended to patients suffering from other diseases of the fundus and of the optic nerve.

Apparatus and Methods

The investigation was carried out on 46 subjects aged from 19 to 75 years. Certain criteria were laid down for the selection of the subjects: the corrected visual acuity was not less than 6/12, there was no defect of fixation, the media were clear, and no gross anomalies of refraction were present. In chronic simple glaucoma cases no other eye disease was present and the tensions were controlled by medical therapy at the time of examination (17–22 mm. Hg applanation); only one surgically treated case was included in the series.

Only four patients had to be rejected from the study on the grounds of insufficient co-operation.

The patients were selected mainly from the out-patient departments or the special clinics (glaucoma and uveitis) of both branches of Moorfields Eye Hospital; their general health was good and none of them showed any clinical evidence of vascular hypertension.

The requirements of the apparatus were that it should be capable of producing a transient acute rise in I.O.P. whilst causing the least possible discomfort to the patient and the

minimal displacement or distortion of the eyeball. A small stainless-steel cylindrical plunger of 4 mm. diameter, carried on the end of an arm, could be made to press against the globe, through the lower lid, by adjustment of a coil spring; the arm was the extension of the coil spring the other end of which was anchored to the body of a 25 Kohm potentiometer incorporated in a suitable Wheatstone bridge circuit. The rotation of the potentiometer body changed the force applied by the spring arm, the current flowing through the micro-ammeter across the bridge giving a measure of the force exerted on the eye. The surface of the cylinder pressing on the lower lid had the same curvature as the globe and was covered by a thin rubber film. Its position was adjusted so that the pressure was exerted 5 mm. behind the limbus. The coil spring and its regulating mechanism were held by two movable metal arms attached by means of two ball-joints to the vertical bar of the chin-rest of the 360 Haag-Streit slit lamp. The patient was seated as for applanation tonometry (Figs 1 and 2). Using this apparatus and after some experience in manipulation, it became possible to increase and maintain a fairly stable level of I.O.P. of 32–36 mm. Hg, measured by applanation, for a period of 3 to 4 minutes.

FIG. 1.—The apparatus with subject in position.

FIG. 2.—Close-up showing plunger applied to eye through lower lid.

In order to avoid artefacts due to backward and upward displacement of the eye and to the distortion of the cornea, the field under examination was limited to an area of 25° round the fixation point and the elevation in I.O.P. was restricted to a maximum of 32–36 mm. Hg. Under these circumstances the discomfort to the patient was trivial, so that he was able to concentrate adequately on the subjective test, whilst the displacement of the globe was minimal and the astigmatism produced amounted to no more than 0.5 D; furthermore, at this level of pressure, there was no visual blackout nor difficulty in maintaining steady fixation.

An apparatus recently designed by Buchanan and Gloster (1964) at the Institute of Ophthalmology was used for the examination of the visual field. It consists of a plane black surface, 1 metre square, over which 74 holes, each of 0.8 mm. in diameter, are distributed round a dim red central light that serves as a fixation point. These holes are disposed regularly in five concentric circles corresponding to eccentricities of 5, 10, 15, 20, and 25°, the eye examined being at a distance of one metre. Each of the holes can be lit internally, one at a time and in a random order of presentation. If the patient fails to perceive the flash, it can be repeated, and in the case of a second failure, the operator can register this automatically. The brightness and duration of presentation of the white light flash can be adjusted at will over a wide range, these two parameters remaining stable for all the stimuli. In this investigation the brightness and the duration of the stimulus were kept constant so that all eyes tested had a common basis for comparison. (The brightness of the test stimulus was equal to that of a white screen illuminated with a 40-watt pearl lamp to give an illumination of 32.5 foot-candles and viewed so as to make its field size equal to that of the test field. The duration of presentation was 0.75 sec.)

The test for each eye lasted about half an hour and consisted of two parts:

(1) The patient was left for 10 minutes in the perimeter room in order to become adapted to an ambient level of illumination of 1.5 lux, and thereafter all the perimetric examinations were accomplished under the same conditions; according to Dubois-Poulsen (1952, 1960) this increases the sensitivity of the perimetric study by making use of the well-recognized hemeralopia of glaucoma. The first examination to be done was kinetic perimetry on a black Bjerrum screen, performed with the 7.5 and 3/2,000 mm. white test objects, other sizes being used if necessary.

(2) The patient was given 5 minutes' rest and a brief explanation of the second part of the test. Light sense (static) perimetry was then carried out, the patient being seated one metre from the screen. The first eye was tested, 2 minutes' rest was allowed, and then the compression was applied with the apparatus described. In many cases, after the application of the plunger, there was slight blurring of vision affecting especially the fixation area, but lasting for a few seconds only. With the eye under compression, light-sense perimetry was repeated. The average time for examining the visual field under compression was never more than 3-4 min. For this short period and for the pressure applied, it was thought that the tonographic effect in each eye tested, and especially in the non-glaucomatous eyes, was not important enough to alter significantly the maintenance of the I.O.P. in the range adopted. Immediately after the test, the compression was discontinued by withdrawing the patient's head from the chin-rest, novesine and fluorescein drops were instilled in the eye just tested, and the fellow eye was uncovered. The patient then replaced his head in exactly the same position so that the same compression was applied to the eye as during static perimetry. In this position the I.O.P. was measured by the Goldmann applanation tonometer. It was not found possible to measure the pressure during compression of the eye in all subjects. However, in the fifteen eyes in which this was possible, the pressure obtained was between 32 and 36 mm. Hg.

The same procedures were then followed for the second eye.

Results

The present study was carried out on 46 subjects (88 eyes) and is based on an assessment of the general condition of the eye, including examination on the Bjerrum screen, and on a comparison between static perimetry carried out before and during compression on the eye.

The eyes examined were grouped as follows (see Table):

- (1) Healthy, 25.
- (2) Glaucoma, 35 (low-tension glaucoma 4, traumatic glaucoma 1, glaucoma secondary to uveitis 1, chronic simple glaucoma 29).
- (3) Suspected glaucoma, 21.
- (4) Inactive chorio-retinal or optic nerve disease, 7.

TABLE
SUMMARY OF RESULTS OBTAINED IN 88 EYES

Group	Initial Diagnosis		No. of Eyes Tested	Pathological Reaction in Bjerrum Area	Normal Reaction
(1)	Healthy		25	—	25
(2)	Glaucoma	With visual field defect	29	29	—
		Without visual field defect	6	4	2
(3)	Suspected glaucoma	Suspicious tension or outflow	16	9	7
		Suspicious tension or outflow <i>and</i> optic disc changes	5	5	—
(4)	Chorio-retinal lesion		1	1	—
	Old retrobulbar neuritis		1	1	—
	Congenital optic nerve anomaly		1	—	1
	Degenerative macular lesions in young subjects		4	—	4
Total			88	49	39

Assuming that the number of stimuli not perceived by the patient in static perimetry is a measure of the depression of the light sense in corresponding areas of the visual field, the results may be described as follows:

Group 1.—In healthy eyes there was a slight decrease in sensitivity during the compression, predominantly at the upper part of the 25° circle. Their effect was accentuated with age, being more obvious in subjects over the age of 55. There was never any reduction of sensitivity in the Bjerrum area of healthy eyes (10°, 15°, and 20° circle) during compression.

Groups 2 and 3.—In glaucomatous eyes compression produced an obvious fall in sensitivity in the Bjerrum area as a whole and especially in its upper half, the lower half being affected rarely. If cases are grouped according to the changes in the visual field existing under normal conditions of examination, the following facts emerge. In advanced cases, compression tends to expand the existing Bjerrum scotomata leaving intact central and peripheral islands of vision. In very advanced cases, with only central and lower temporal islands of vision left, compression had practically no effect. In most of the suspected glaucoma cases in which the I.O.P. or the facility of outflow were found suspicious but no visual field defects were present, the test was found to be positive; that is, some stimuli in the Bjerrum area were not perceived

during compression. Only 21 suspected cases were examined, this number being too small for any statistical analysis; but it is felt that the outcome of the test is closely related to the condition of the optic disc rather than to the tension and coefficient of outflow. In this series there were five cases with suspicious discs (*i.e.* cupping but little pallor) in addition to doubtful tensions and outflows, and all five gave a characteristically glaucomatous reaction. These results are summarized in the Table, and some illustrative cases will be described more fully.

Case 32, a man aged 61 years, had a healthy right eye with visual acuity of 6/6 and I.O.P. of 17–18 mm. Hg (applanation). During compression there was a reduction of sensitivity in the upper periphery, a response commonly found in older subjects (Fig. 3).

* FIG. 3.—Case 32, right eye.

FIG. 4.—Case 6, right eye.

* Explanation of symbols in Figs 3–12: ● defect before compression
 △ defect during compression

Case 6, a man aged 64 years, had controlled early chronic simple glaucoma under miotic control in the right eye. The visual acuity was 6/6 with pathological cupping of the optic disc without marked atrophy. On the Bjerrum screen an enlargement of the blind spot was detected. With static perimetry two points were not perceived in the upper part of the 25° circle. During compression an upper Bjerrum type of scotoma developed (Fig. 4).

The left eye had advanced chronic simple glaucoma.

Case 22, a man aged 39 years, had very advanced chronic simple glaucoma in the right eye (Fig. 5, overleaf); early glaucoma in the left eye was detected two years ago. In the right eye the visual acuity was 6/6; miotic treatment failed to control the I.O.P. but a fistulizing operation kept the tension well within safe limits (16 mm. Hg). During compression the existing scotomata enlarged (Fig. 6, overleaf). This was the only surgically treated eye tested.

In the left eye with incipient glaucoma without cupping of the disc or visual field loss, no changes were found during compression.

Case 15, a man aged 50 years, had visual acuity 6/5 in each eye. This patient was under observation in the glaucoma clinic because the tensions and outflows had been found to be pathological on three occasions (24–27 mm. Hg applanation). The discs showed wide physiological cupping but there was also a suspicion of pathological change, although no visual field defects were found. During compression the reaction was characteristically glaucomatous (Fig. 7, overleaf).

FIG. 5.—Case 22, right eye.—10/2000 . . . 5/2000.

FIG. 6.—Case 22, right eye.

FIG. 7.—Case 15, right eye.—7·5/2000 . . . 3/2000.

Case 44, a woman aged 44 years, had visual acuity 6/6 in each eye. She was under observation in the glaucoma clinic because borderline tensions and coefficient of outflow were found on two occasions (right and left eyes, 24 and 26 mm. Hg applanation). No evidence of cupped discs or visual field defect was present. During compression no pathological reaction was found.

Group 4.—This comprised a small number of eye affections not complicated by increased I.O.P. but producing scotomata in the area of visual field examined.

Case 2, a man aged 19 years, had visual acuity 6/12 in each eye. In the right eye there was an old quiet chorio-retinal scar, probably of toxoplasmic origin, below and temporal to the optic disc, with pallor of the corresponding segment of the optic disc. There was an incomplete upper Bjerrum scotoma with rather steep margins, which during compression tended to become complete (Figs 8 and 9).

FIG. 8.—Case 2, right eye.—15/2000 . . . 2/2000.

FIG. 9.—Case 2, right eye.

Case 12, a woman aged 54 years, had been observed for several years in the glaucoma clinic because of pathological cupping and pallor in the temporal section of the disc in the left eye. The visual acuity was 6/6; paracentrally there were two scotomata of unusual shape, which had shown no progress over a period of 5 years. Elaborate tests to reveal glaucoma were always negative and in view of these facts a diagnosis of congenital optic nerve anomaly had been made. The right eye was always found to be healthy. During the compression a decreased sensitivity round the existing scotomata was produced but there was no evidence of an abnormal reaction in the Bjerrum area (Fig. 10).

FIG. 10.—Case 12, left eye.

FIG. 11.—Case 39, right eye. . . . 3/2000.

Case 39, a woman aged 43 years, had had a typical attack of retrobulbar neuritis in the right eye one year ago with a central scotoma extending towards the upper nasal periphery. The disease settled down quickly, leaving a visual acuity of 6/6, slight pallor of the disc temporally, and a slight constriction of the peripheral isopters nasally and above. On examination by static perimetry nothing definitely abnormal was found but under compression a marked diminution in sensitivity in the upper Bjerrum area was recorded (Fig. 11).

Discussion

The possible sources of error in the present method of examining the visual field during ocular compression are associated firstly with the upward and backward displacement of the globe and the consequent slight ptosis of the upper lid, secondly, in some young subjects, with a slight lacrimation, and lastly with the induced corneal astigmatism. These factors may not only disturb the optics of the eye but may also render unreliable the applanation tonometer readings; in the present study they were obviated by restricting the extent of visual field examined and the amount of the elevation of I.O.P. Obviously the perimetric method used, though offering a quick way of testing, falls short of the ideal approach, which would be a comparison of light sense thresholds of a selected area of the retina. The realization of this ideal would necessitate a drastic reduction in the number of points examined, as the time available for the examination during ocular compression cannot be extended beyond 4 or 5 minutes if a relatively constant level of raised I.O.P. is required. The method adopted is therefore a compromise which has the advantage of permitting the rapid exploration of a relatively large area of visual field.

The use of an interrupted light stimulus avoids the phenomenon of local retinal adaptation which may account for the disappearance of a small static test-object placed eccentrically, as was used by Harrington (1961) and Drance (1962b).

In summarizing the results, it is convenient to divide the changes in the light sense into those which occur in the periphery, those in the Bjerrum area, and those round existing scotomata of non-glaucomatous origin.

In normal eyes, there was often evidence of a lowered sensitivity in the upper periphery during the compression of the eye. This manifested itself in two ways: either the stimulus was not seen at all or it was perceived only at a second presentation. Moreover, this peripheral depression was more marked in the older subjects, occurring in almost all over the age of 55. This type of reaction was encountered in patients of the three remaining groups, being superimposed upon the changes occurring in other parts of the visual field. It is felt, therefore, that this type of reaction, although sometimes more marked in glaucoma, was nevertheless not specific for that condition.

In the Bjerrum area none of the normal eyes showed any evidence of depression of sensitivity when the I.O.P. was increased.

In glaucomatous eyes, where campimetry showed a scotoma in the Bjerrum area, the effect of ocular compression was to cause this to extend, a phenomenon so characteristic that it seems reasonable to regard the appearance of a Bjerrum scotoma during compression, or the extension of an existing one, as a "glaucomatous reaction".

In cases of advanced glaucoma, where only central and temporal islands of vision were left, compression had no effect.

In suspected glaucoma, where only the tension and facility of outflow raised doubts the glaucomatous reaction was found in nearly 50 per cent. of cases, but in five eyes in which there was also a suspicious appearance of the disc this reaction was always found.

A "glaucomatous" type of reaction was also obtained in Cases 2 and 39, both of which had evidence of damage to nerve fibre bundles at the optic disc, in one case

as a consequence of past uveitis, in the other as a consequence of retrobulbar neuritis. The "glaucomatous" reaction was not obtained in Case 12, which showed a congenital defect of the disc, although the pericentral scotomata did extend.

From these facts certain conclusions can be drawn. As regards the responses to compression in the Bjerrum area, there is a clear differentiation between the healthy and the diseased eyes. From the observations on Cases 2 and 39, it is obvious that the "glaucomatous reaction" is not confined to cases of glaucoma but is obtainable in eyes in which optic nerve fibre bundles have been damaged from some other cause. It is not possible, of course, to decide from the present data whether or not the reactions described are specific for field defects originating from fibre-bundle lesions, but it is quite clear that if such lesions exist the functional defects are accentuated by compression. From the diagnostic point of view the most useful manifestation of this effect is the production of scotomata in the Bjerrum area in cases of suspected glaucoma, where fibre-bundle damage is so early as not to produce perimetric defects by the usual methods of examination or to give a pathological appearance of the disc on ophthalmoscopy.

By means of this test a clinically useful comparison can also be accomplished between the two eyes of the same subject, giving some help in deciding on the further treatment, as the following example illustrates:

Case 3, a man aged 42, was struck in the left eye by a cricket ball 6 months ago. When he visited the casualty department the day after the accident, the eye was red, slightly painful, and showing mild iritis and marked hypotony. The reaction slowly settled down completely during the ensuing month, but a little later the I.O.P. began to rise. Consequently, the eye was treated medically as secondary glaucoma, the tension being stabilized at a level of 19–21 mm. Hg (applanation), but rising occasionally by 3–4 mm. Hg. The visual acuity was now 6/9 and the disc slightly cupped and pale, and the visual field showed a lower Bjerrum scotoma for 2/2000 white test object. In static perimetry there was a reduction in sensitivity in the upper and lower Bjerrum area, affected dramatically during compression (Fig. 12).

FIG. 12.—Case 33 (*see text*), left eye.

The right eye was healthy, the I.O.P. being 10 mm. Hg. The patient was treated medically during the succeeding 4 months, the tension in the left eye being regarded as controlled since it was within the accepted conventional limits of normality. If we assume that the I.O.P. in the left eye before the accident was at the same level as in the right eye, we would regard the control as inadequate, even though the I.O.P. falls within the statistical range of normality (Gloster, 1962). The considerable functional loss under compression of the eye is further evidence that in this case the susceptibility to pressure is high, so that more drastic measures are required to bring the I.O.P. within safe limits.

The work of Leydhecker (1959) on cases of chronic simple glaucoma indicates that, between the onset of a pathological increase in the I.O.P. and the early manifestations of visual field changes, there is a lapse of nearly 20 years, so that what we call the full picture of established chronic simple glaucoma is a late phase of this long-term insult to the optic nerve. It also seems that, anatomically, the optic nerve fibre bundle supplying the Bjerrum area is the earliest to be affected. From the clinical point of view, the early pathological variations of the I.O.P. fall, for the great majority of glaucomatous patients, just below or at the upper limit of the statistically-accepted normal range of values, but nevertheless insidious damage to the optic nerve proceeds, reducing the sensitivity in the Bjerrum area and giving rise to the earliest perimetric findings. Thus, in the stage of glaucoma evolution at which "suspected glaucoma" cases may be selected for further investigation, the facility of outflow, intra-ocular pressure, and response to the provocative tests are usually found to be in the doubtful range, though these hydrodynamic disturbances are not sufficiently advanced to give rise to optic disc and visual field changes demonstrable by the ordinary clinical methods. The procedure described may be regarded as a provocative test which reveals the existence of diseased optic nerve fibres, its effectiveness being based on the fact that damaged fibres are more sensitive than healthy ones to an acute artificial increase in the I.O.P. This study seems to contribute therefore to the exploration of this clinically important stage of the glaucomatous process, being designed to reveal the existence of damage to the ganglion cell neurons supplying the Bjerrum area of the visual field by depressing the blood supply in the tissue of the optic nervehead.

The reliability of this test as a means of estimating the susceptibility of the individual eye to further functional loss under abnormally high I.O.P. and the extent of its contribution to an individual prognosis cannot as yet be assessed. Only long-term clinical experience will decide whether the theoretical expectation will yield a true enrichment of the diagnostic armamentarium and whether this test should be advocated as a routine method of examination in certain categories of ocular disease.

Summary

Observations were made on 46 subjects of the effects upon the visual field of artificially increasing the I.O.P. The mode of approach is described and the results obtained in four groups of patients are discussed:

(A) In cases of established glaucoma, the most significant finding was that scotomata in the Bjerrum area were enlarged during compression of the eye.

(B) In cases of glaucoma where scotomata were not found by ordinary examination, compression of the globe almost always produced visual field defects. Reactions of this type were found in none of the normal eyes.

(C) In cases of suspected glaucoma, a proportion of the eyes reacted like those with established glaucoma; five out of 21 cases had suspicious discs in addition to tonometric and tonographic evidence and all of these gave reactions characteristic of glaucomatous eyes; nine of the remaining cases, in which the discs were normal in appearance, reacted in the same manner.

(D) This type of reaction is not diagnostic of glaucoma, however, as it was noted in two cases in which an optic nerve bundle was affected from causes other than glaucoma.

The test appears to be promising as an additional means of provocation in suspected cases of chronic simple glaucoma, especially if some evidence of a pathological process in the optic disc exists; furthermore, as Case 12 indicates, it is felt that it should be helpful in differentiating between malformation of the optic disc and low-tension glaucoma (Perkins, 1959; Winstanley, 1959; Primrose, 1959). In cases of established chronic simple glaucoma, it is felt that judgment must be reserved regarding any prognostic significance concerning the visual loss to be expected, until long-term observations have been completed.

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