The visual field defect and ocular pressure level in open angle glaucoma

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When we inquire about the effect of ocular pressure level on the course of the visual function loss in glaucoma, we are in general offered two extreme though simple hypotheses. The first contends that the entire course can be explained by variation in ocular pressure level, and the second proclaims that the course of the visual function loss is independent of the level of ocular pressure. Despite their attractive simplicity, which accounts in a major part for their survival, neither one of these hypotheses alone can describe the reality of clinical experience.

There seems to be a general acceptance of the notion that ocular pressure level exerts its effect on visual function by compromising the blood supply to the nerve fibers sufficiently to interfere with their function or survival. Thus it follows that the effect of a given pressure level at a certain time depends upon the prevailing characteristics of the vascular bed and upon the ability of the neuronal elements to withstand the resulting deprivation. Hence the magnitude of reduction in blood supply that results from a certain level of ocular pressure will depend upon such factors as the arterial pressure of the feeding vessels and the number and caliber of the vessels comprising the vas-
cular bed. The effect of changes in these factors is such that the lower the pressure in the feeding arteries, the fewer the vessels, the smaller their caliber, the greater will be the resulting vascular effect.

We can further extend this reasoning by saying that the effect of a given reduction in blood supply on function will vary with the ability of the neuronal elements to withstand this alteration. If, to begin with, these neurones are functioning at a handicap, be it metabolic or endocrine, then their ability to withstand reduction in blood supply may be greatly reduced. Thus it is possible for a given level of ocular pressure to lead to function loss in a certain individual but not in another or in the same individual at one time but not at another. Therefore, the hypothesis becomes one in which the offender is still the ocular pressure level but its effect in any one individual or at any one instant is complicated by the operation of modifiers or factors that determine the vulnerability of the function of the optic nerve fibers. I shall emphasize in this presentation that these modifiers need not be only ocular, in fact they do not need to be limited to the vascular category, but may extend to include the metabolic and endocrine as well. Thus a broader perspective results in which the visual function in glaucoma is not dependent upon the ocular pressure level alone, but also upon other factors, ocular and extraocular, that modify this effect sufficiently to negate a simple hypothesis and to warrant their consideration together with ocular pressure level in the composite of factors that determines the course of visual function. The cases to be reported presently indicate that such factors or modifiers will include systemic hypertension, abnormal glucose tolerance test, and peptic ulcer.

If one were to describe the most salient feature of the relationship between ocular pressure level on the one hand and onset and course of visual field defect in open angle glaucoma on the other, it will have to be the marked individual variability in this respect. I shall demonstrate this in selected case reports which will reveal the inadequacy of simple hypotheses and the need to identify the modifiers and elucidate their method of operation. I shall describe and emphasize in particular the reversible nature of some glaucomatous defects and the favorable influence of reduction in intraocular pressure level.

The visual field defect in glaucoma

In order to speak of the relationship between the visual field defect in glaucoma and other parameters, one must first define the defect. Ideally such a definition should include a description of the time course of field defects in glaucoma; their early beginning and transition to the advanced stage of involvement. Unfortunately, our knowledge in this respect is sketchy and at times speculative. It is complicated by the fact that the defect, even in its advanced form, is not specific for glaucoma but has been shown to occur with other disease entities. It is more seriously handicapped by the finding that what may be considered an early field change in glaucoma is produced by a wide variety of factors whose frequency increases with age. As a result, the specificity of these defects and consequently their usefulness in establishing the diagnosis of glaucoma in the individual subject is markedly reduced. Such is the case, for instance, with peripheral contraction of the isopter; this may indeed be the most frequent and the earliest field change in glaucoma, yet it is produced by so many other factors that it cannot be profitably utilized as a criterion of glaucomatous involvement of the visual field. Involvement of the central field is more profitable in this respect. For, although the defects may occur with other disease entities, their specificity in this respect can be markedly improved by ruling out a majority of the possible causes. This can be done by the following procedures: (1) a detailed examination of the fundus through a dilated...
pupil to rule out local chororetinal lesions that can account for the defect; (2) a detailed ocular history to rule out retrobulbar neuritis; (3) x-ray examination of the skull in the region of the sella turcica and optic foramen; (4) complete neurologic examination, including serologic tests for syphilis.

When no relevant positive findings can be uncovered by the above procedures the defects are then considered glaucomatous in nature. Thus it is a diagnosis by elimination.

The following defects in the central field were considered to be glaucomatous in nature when they could be outlined by the 1/1,000 or larger white target on the tangent screen, or by the I-2-e or greater stimulus on the Goldmann perimeter, and could not be accounted for by the findings

![Figure 1](image1.png)

**Fig. 1.** Glaucomatous field defects. A, A nasal step greater than 10° and a paracentral scotoma greater than 5° outlined on the Goldmann perimeter using the I-2-e stimulus. B, A Bjerrum scotoma that breaks through the I-2-e isopter (inner isopter) and which persists with the I-3-e (light hatching) and becomes associated with a nasal step greater than 10° (intermediate isopter). With the I-4-e, the scotoma breaks into an arcuate and a paracentral scotoma (solid black) and the isopter (outermost) continues to demonstrate a nasal step greater than 10°.

![Figure 2](image2.png)

**Fig. 2.** Glaucomatous field defects. A, A Bjerrum scotoma with the I-2-e that breaks through the isopter (innermost). The I-3-e (intermediate isopter) shows a nasal step and a normal blind spot not associated with paracentral scotomas. With the I-4-e the isopter (outermost) demonstrates a nasal step. B, An arcuate scotoma greater than 45° and a depressed upper nasal boundary with the I-2-e. The scotoma is less for the I-3-e (vertical lines, intermediate isopter). The I-4-e demonstrates normal blind spot (solid black) and isopter (outermost tracing).
of any of the procedures described above: (1) an arcuate scotoma continuous with the blind spot and extending beyond 45° above the horizontal or 50° below the horizontal; (2) a paracentral scotoma greater than 5°; (3) a nasal step greater than 10°.

The procedure of selective perimetry was used in the detection of the above defects. Thus, I shall be speaking of these specific defects and of the effect of ocular pressure level on their appearance and course keeping in mind that these may be only a partial list of the types of glaucomatous involvement of the visual field.

Ocular pressure level and the onset of the field defect

The first question in this regard is the following: Is the development or appearance of these field criteria unrelated to ocular pressure level? The answer is in the negative. Approximately 4,000 subjects with normal eyes and normal visual fields participated in a long-term follow-up study which involved yearly examination of ocular pressure level by applanation tonometry and examination of the visual field by tangent screen or Goldmann perimetry. Of these, four subjects developed, during the period of observation, the glaucomatous field criteria enumerated above. These defects appear in Figs. 1 and 2 and exemplify what are considered glaucomatous field defects in this presentation. The applanation pressure readings on initial examination of these four individuals (that is, at the time when their visual field was normal), and those obtained at the time when the defect was first detected, appear in Table I. It is apparent that the development of field loss cannot be considered unrelated to ocular pressure level, for in these cases the ocular pressure level, even before the appearance of the field defect, was higher than the mean value for the sample. More significantly, when the defect was first detected, all four subjects had an applanation reading higher than 22 mm. Hg. Of the 4,000 subjects seen, only a small fraction (approximately 200) had a pressure reading higher than 22 mm. Hg. Thus the hypothesis that the appearance of these defects was unrelated to ocular pressure level has to be rejected at a high level of confidence (better than one per cent). Their appearance was restricted to eyes with high applanation pressure level.

The second question becomes: is this association a simple one? The answer is an emphatic no. For, although the defect appeared in eyes with high applanation pressure level, it could not be concluded that all eyes with a pressure level equal to or higher than that level had a field defect. Furthermore, no simple relationship could be discerned between pressure level on initial examination and the time lapse that preceded the onset of field loss. This immediately underlines the marked individual variability with respect to the vulnerability of the visual field to the effect of ocular pressure level. Not all, but some individuals with high pressure levels develop field defects. Consequently, two related questions come to the forefront: the first is, what factors other than ocular pressure level, be they ocular or extraocular, help identify individuals with increased vulnerability in this respect, and what is the mechanism of their operation? Conversely, what are those factors that can distinguish individuals with high resistance in this regard? Obviously, if open angle glaucoma is to be considered in terms of its effect on the visual field, then these questions are not irrelevant nor academic but are of the essence for our understanding and practical management of glaucoma. The factors in question become

Table I

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Applanation pressure</th>
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<tr>
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<td>In results on initial examination</td>
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<tr>
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<td>20</td>
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an integral part of the complex that determines visual field loss in glaucoma, and in that context are as significant as the ocular pressure level itself.

The effect of change in pressure level on the field defects

Significant in this respect is the effect of dexamethasone-induced pressure rise on the visual field. In eyes with normal visual fields the pressure rise produced by topical application of dexamethasone was shown to produce contraction of the isopter and enlargement of the blind spot. More significantly, it was shown to produce para-central scotomas identical with those of spontaneous open angle glaucoma (Fig. 3). The important feature in this respect was that in the production of these defects the magnitude of pressure rise was more important than the absolute value of pressure level attained. Furthermore, with increasing age a smaller magnitude of pressure rise was required to produce changes in the visual field. Thus an increase in pressure level can indeed produce field defects identical with those of open angle glaucoma and becomes more

Fig. 3. Changes in the central visual field with dexamethasone hypertension in an otherwise normal eye of a 32-year-old son of a glaucoma patient. Before dexamethasone: intraocular pressure (IOP) OD, 19 mm Hg; visual acuity (VA), 20/20. The isopter for the I-2 test object appears delineated by a solid line and the blind spot in solid black. After four weeks of dexamethasone: IOP OD, 37 mm Hg; VA 20/20. The isopter for the I-2 was not altered. Changes in central visual field are represented by stippled areas: enlargement of the blind spot and the appearance of two disconnected Seidel scotomata between the tenth and twentieth degree of field.

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Effective with advancing age, equally significant was the course of recovery of these defects. While, in general, as the pressure level was reduced on cessation of dexamethasone application, the visual field change disappeared in one to two weeks, in some cases the field defect persisted after the pressure level had been normalized and remained for two to three months before complete disappearance.11

In the glaucomatous eye with definite field defect, an increase in pressure level was shown to produce an increase in the field defect in the direction of more advanced glaucomatous loss. In this respect, the significant finding was that eyes with low tension glaucoma required a smaller magnitude of pressure rise to demonstrate a marked increase in the defect (Fig. 4). Similar findings were reported with betamethasone-induced ocular hypertension in open angle glaucoma13 as well as with the mechanically produced rise in ocular pressure.15 Thus a glaucomatous field defect can be markedly aggravated by an increase in ocular pressure level.

Is the field defect in glaucoma permanent? The answer must be in the negative. Glaucomatous field defects may show complete recovery. In general, when we
Table II

<table>
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<th>Year</th>
<th>Applanation pressure (mm. Hg)</th>
<th>Tonographic C-value (ml./mm. Hg/min.)</th>
<th>Field defect with 1/1,000 white tangent</th>
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<td></td>
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<td>OD</td>
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<td>15</td>
<td>15</td>
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speak of glaucomatous field defects as being due to the involvement of bundles of optic nerve fibers, we also emphasize the fact that the neural elements involved do not regenerate and conclude that the defect is therefore of a permanent nature. While this reasoning may be valid, when fibers are permanently destroyed, we must emphasize the fact that we cannot distinguish on visual field examination loss of nerve fiber function which is due to irreversible structural destruction of the neuronal elements from that which may result from a reversible lesion involving these elements. I shall, therefore, emphasize in the remaining part of this presentation the fact that the visual field defect in glaucoma at some stage may indeed be completely reversible and describe some of the examples that illustrate the extent and the depth of such defects and the marked individual variability in this respect also.

I shall begin by describing my first encounter with this phenomenon of reversibility.14 The findings appear in Table II. This 54-year-old Caucasian male patient refused any ocular or systemic therapy, but continued to come for repeated examinations. Note the spontaneous progressive reduction in the ocular hypertension to reach normal levels. Note also the associated changes in the field defects which consist of progressive reduction until they disappear completely. This case emphasizes the completeness of the recovery and the fact that it occurs over a long interval of time; it also emphasizes the association between reduction in ocular pressure levels and recovery of the defect, and points out that spontaneous normalization of ocular pressure and aqueous dynamics together with that of the field defect in open angle glaucoma is indeed possible. The next patient illustrates the marked variability in this regard by demonstrating that a recent visual field defect continued to progress in spite of the marked and prompt reduction in ocular pressure level. This 73-year-old Caucasian male patient had been followed at three-month intervals for ocular hypertension not associated with a field defect for a period of three years. The record of the field on the last normal follow-up appears in the upper part of Fig. 5. A week later he accompanied a relative of his to the clinic and during that time received a visual field examination which for the first time showed a significant field defect that appears in the lower part of Fig. 5. Ocular pressure level at this time was lower, not higher, than that on the preceding examination. He was immediately admitted to the hospital and subjected to daily examination of the visual field and diurnal determination of applanation pressure reading at three-hour intervals between 6:00 A.M. and midnight. He was given an intravenous injection of 500 mg. of Diamox and was maintained on Diamox sequels, 500 mg. each, taken twice daily. Ocular pressure level was promptly reduced and diurnal variation was maintained below 18 mm. Hg. Nevertheless, the visual field defect, which could not have existed for longer than one week, continued to increase rather than improve.
Fig. 5. The key for visual field tracing appears in the lower right corner. Each target size (indicated by a Roman numeral in the left column) is represented by a geometric symbol appearing next to it. Each target intensity (indicated by an Arabic numeral in the upper row) is represented by a specific line tracing indicated above it. The isopter and the boundary of scotomas will be represented by the line that corresponds to the intensity of the stimulus used in outlining them and will connect symbols representing the size of the target employed. In addition, scotomas and blind spots will be covered by an appropriate surface representing the target size and intensity used in outlining them. These will appear in the key in the square connecting the intensity and size coordinates for each stimulus and will be as follows: I-2-e dense stippling, I-3-e light stippling, I-4-e solid black, II-4-e fine vertical lines, V-4-e heavy vertical lines.
Samples of field during follow-up appear in Fig. 6. Note the increase in the defect with the I-3-e on Nov. 6, 1965, in spite of the fact that ocular pressure level was maintained below 18 mm. Hg since the first discovery of the defect. Longer follow-up failed to demonstrate any improvement.

In contrast with the above, the patient depicted in Figs. 7 and 8 illustrates how extensive field defects recovered in spite of the maintenance of a high level of ocular pressure. This 77-year-old Caucasian female patient had been followed for twelve years with an applanation reading that varied between 35 and 48 mm. Hg in either eye and maintained, nevertheless, a normal visual field. She refused to use any ocular hypotensive therapy and reported regularly every two to three months for visual field examination. The last normal field appears in the upper illustration of Fig. 7. When she was seen three months later a definite arcuate scotoma,
paracentral scotoma, and nasal step could be outlined with the I-2-e and I-3-e. The pressure reading was not higher than that found at the preceding examinations. She continued to refuse therapy and was seen at a frequency that varied between once a week and once a month. Ocular pressure continued to vary between the mid-thirties and the mid-forties, and the field defect did not increase in extent or in depth (upper illustration of Fig. 8). On later visits the defect improved slowly, first becoming less deep and then less in extent until it disappeared completely and resulted in a perfectly normal visual field on September 14, 1967, (lower illustration of Fig. 8). Throughout this time the ocular pressure level was not significantly reduced but remained above 35 mm Hg.

The patient described in Figs. 9 and 10, on the other hand, demonstrates an increase in the field defect in spite of an otherwise reasonable level of ocular pressure; the defect nevertheless reacted favor-
ably to reduction of the pressure level by Diamox therapy. This 77-year-old Caucasian male patient on first examination demonstrated an arcuate scotoma and nasal step; the pressure reading was 18 mm. Hg. He failed to report on the following week and was not seen until nine months later, at which time the arcuate scotoma had increased markedly and became associated with paracentral scotomas detected with the very high stimulus II-4-e and V-4-e. The ocular pressure level at that time was 19 mm. Hg. Pressure determinations at three-hour intervals varied between 16 and 22 mm. Hg. He was then given Diamox sequels, 500 mg., twice daily, and ocular pressure level was promptly reduced and the diurnal variation remained below 18 mm. Hg. at all times. The defect improved slowly and reached maximum improvement after three months, at which time the arcuate scotoma and all the upper paracentral scotomas had disappeared leaving only

Fig. 8. Key as in Fig. 5, for details see text.
the nasal step and the inferior scotoma. The recovery of a defect as deep as V-4-e, which is virtually an absolute defect on the Goldmann perimeter, is indeed most remarkable.

In contrast, the patient depicted in Fig. 11, a 40-year-old Caucasian male patient, illustrates how a normal visual field was maintained over a period of follow-up of eight years during which the pressure level was never recorded below 30 mm. Hg.

Finally, the patient depicted Figs. 12, 13, and 14 illustrates the great individual variability with respect to the pressure level which is consistent with normal visual fields. This 72-year-old Caucasian female patient, when first seen on October 5, 1965, demonstrated an absolute Bjerrum scotoma. The I-4-e isopter was intact. Applanation reading was 18 mm. Hg. Six months later she showed a progression of the defect as evident in the involvement of the I-4-e isopter in the Bjerrum scotoma.
and the appearance of a nasal step with the V-4-e (Fig. 12). Applanation reading was 22 mm. Hg at this time and the diurnal variation ranged between 16 and 24 mm. Hg. No therapy was given at that time and she was seen on January 12, 1967, at which time the field defect had increased further, so that the I-4-e isopter now demonstrated an upper altitudinal defect. Diurnal variation at this time fell between 16 and 21 mm. Hg. Seven months later the I-4-e isopter became extensively involved, demonstrating an almost complete upper altitudinal absolute defect (Fig. 13). Applanation pressure reading was 17 mm. Hg and diurnal variation fell between 16 and 22 mm. Hg. She was then started on Diamox sequels, 500 mg., twice daily. Ocular pressure level was promptly reduced and diurnal variation now fell between 9 and 14 mm. Hg. After one week of this therapy, field examination demonstrated a reversal of the trend and showed marked improvement in the V-4-e isopter. A week later, the V-4-e and the I-4-e isopter had become complete and attained the levels found on initial examination (Fig. 14). A slight residual increase in the Bjerrum scotoma with the I-4-e could still be seen. Thus, in this patient, pressure levels in the high teens were sufficient to produce progressive increase in the field defect which, however, could be reversed almost completely by reducing the ocular pressure level to the low teens in spite of the absolute nature of the defect.

From the above case reports one concludes that certain glaucomatous field defects are completely or partially reversible. It is this attribute that I wish to emphasize because it highlights the need to understand the conditions that lead to this reversal and to manipulate them therapeutically in our attempt to eliminate glaucomatous visual function loss. Size of the defect or its depth were not critical factors in this regard; extensive and deep defects showed complete recovery. Duration of the defect was not a critical factor either; defects known to be of recent onset failed to recover whereas those of long standing demonstrated reversibility. Thus, large and deep defects can exist for long intervals of time in a reversible state; this indicates that the lesion of the neuronal
elements can remain for a long time, one that produces modification or even loss of function and not reach the stage of irreversible destruction of the structural integrity of these elements.

Recovery of the defect was related to reduction in ocular pressure level, but this relationship was not a simple one. Recovery occurred with high levels of ocular pressure and failed to occur in spite of the marked reduction in pressure level. Thus, here again, as in the case of onset of field defect and ocular pressure level, we are presented with a complex relationship. However, from the practical standpoint the two situations differ markedly. For, although a high pressure level is in general related to the development of field defect, we hesitate, because of the complexity of this relationship, to impose a lifetime regimen of therapy on an individual with normal visual fields, whereas in the presence of field loss energetic therapy becomes mandatory irrespective
of whether the pressure dependent component of the field defect proves to be reversible or not. Thus, whenever a field defect of this nature is encountered, an energetic reduction and maintenance of a low ocular pressure level should be pursued. In fact, failure to demonstrate improvement of visual field should indicate the need for a more effective reduction of pressure level by medical means. In this manner, we can determine and treat the pressure-dependent reversible component of the defect.

Marked individual variability was a prominent feature of the relationship between ocular pressure level and the course
of the glaucomatous defect. This difference in vulnerability is thought to reflect the characteristics of the blood supply of the optic nerve head. The status of this vascular factor cannot be evaluated in the individual subject by direct measurement of relevant parameters. Some feel that the ophthalmodynamometrically determined pressure of the ophthalmic artery or its derivatives is a useful predictor of the perfusion of the optic nerve head circulation and, therefore, should be considered in evaluating its resistance to compression by ocular pressure.\textsuperscript{15,16} Others feel that a more appropriate measure is the difference between the mean arterial pressure
and the ocular pressure level and call this value the effective gradient and consider the resistance of the optic nerve to vary directly with it. The ratio between the orbital pressure and that of the brachial artery is considered to be a useful measure in this respect; a smaller ratio is more prevalent in eyes with glaucomatous function loss. A reduction in the systemic arterial pressure level induced therapeutically in the management of arterial hypertension has been shown to disturb the prevailing balance and produce a visual field defect or aggravate a previously existing one. Episodes of systemic hypotension associated with shock, prolonged

Fig. 14. Key as in Fig. 5, for details see text.
anesthesia, or massive hemorrhages were uncovered in cases with glaucomatous visual field loss that was not readily explained by the level of ocular pressure. Severe anemia will also be a factor in increasing the vulnerability of the optic nerve head. Changes in the local characteristics of the vascular bed of the optic nerve head, such as those occurring with age and resulting in reduction in number and in caliber of small vessels, can explain the greater frequency of field defects with age and the increased vulnerability to ocular pressure level.

However, the vulnerability of the optic nerve head to ocular pressure level, though it be an ocular event, may share in its determination factors involved in other systemic diseases; these factors can influence the ocular events in a general way. A knowledge of these factors will help to improve our ability to identify those individuals who are likely to develop glaucomatous field change and understand the mechanism of this change. To gain better insight into the possible nature of such factors, the four patients who developed glaucomatous field defects during the observation period were subjected to a detailed general medical history and examination. The findings clearly showed that these subjects differed significantly from the rest of the sample as well as from subjects with high ocular pressure levels but have normal visual fields. The difference was in the simultaneous presence of other systemic abnormalities. The most impressive of these were the results of the glucose tolerance tests (Fig. 15). Whereas none of the four subjects had known diabetes prior to the test, every one of them had an abnormal glucose tolerance test. Three of these subjects required specific therapy; the fourth was controlled adequately by dietary regulation. In addition, however, other abnormalities were encountered (Table III) and included systemic arterial hypertension, thyroid dysfunction, and peptic ulcer. It seems highly unlikely that, the simultaneous occurrence of these abnormalities in the four patients who developed glaucomatous function loss is due to chance alone. A significant association must be assumed. Thus vulnerability of the optic nerve to ocular pressure level becomes dependent not only on a vascular factor but also upon metabolic and endocrine factors as well. These may operate by modifying the vascular factor itself or by modifying the metabolic activity of the neuronal component and in so doing modify its ability to resist stresses imposed by the effect of ocular pressure on the vascular supply. Whatever the case may be, this significant association calls for a much broader concept of glaucomatous function loss: one in which the ocular pressure level remains the major offender but in which its efficacy or potential harm

Fig. 15. The results of the glucose tolerance test in the four subjects who developed field defect during the observation period. Glucose blood levels appear on the ordinate in mg. per 100 ml. On the abscissa is given the time in minutes after ingestion of 70 Gm. of glucose. The lines limiting the boundary of the stippled area represent the upper and lower limits of variation in normal individuals. The line connecting values for each of the four subjects is numbered 1 through 4. All subjects show values that are above the upper limit of normal. The results of urinalysis for glycosuria during the test are reported for each subject below the tracing.
Table III. Extraocular characteristics of subjects who developed glaucomatous field defect

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Sex</th>
<th>Age</th>
<th>Family history of glaucoma</th>
<th>Abnormal glucose tolerance test</th>
<th>Active (stomach) ulcer</th>
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*++* Indicates that specific therapy was necessary.

is dependent upon the interplay of a wide variety of factors—vascular, metabolic, and endocrine. Our understanding of the problem of open angle glaucoma will become intimately dependent upon our ability to identify these factors and their mechanism of action so as to influence the course of the visual loss in open angle glaucoma.

Conclusions

The onset of field defect in glaucoma is related to the presence of a high level of ocular pressure. The relationship is a complex one and permits marked individual variability. In addition, this is also related to the presence of other abnormalities—vascular, metabolic, and endocrine—that seem to influence the vulnerability of the optic nerve head.

The field defect in glaucoma may be completely reversible and its recovery is related to reduction in ocular pressure level. However, marked individual variability exists, indicating the operation of other factors. This emphasizes the need to identify these factors and elucidate their operation in influencing the onset or course of glaucomatous function loss.

Reversibility of the glaucomatous defect underlines the need for its early detection by careful examination of the visual field. It also emphasizes the need to energetically reduce by medical means the ocular pressure level when a glaucomatous defect is detected, irrespective of the prevailing level of ocular pressure. We must learn to assess the adequacy of therapy by carefully following the course of the field defect.

Finally, the marked individual variability with regard to the onset and course of visual function loss in glaucoma and the level of ocular pressure emphasizes the operation of other factors that influence markedly this relationship and underlines the need to look for and identify these factors in order to develop a more comprehensive understanding and control of the glaucomatous process.

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(End of Symposium)