significance. It seems absurd to think of two separate diseases occurring in those patients with classical low tension glaucoma in one eye and overt glaucoma in the other. It seems unreasonable to find family histories of classical open-angle glaucoma (with high pressures) in patients showing typical low tension glaucoma unless a single disease is involved. It seems unreasonable to label a patient as having glaucoma when he has had low tension glaucoma for years, just because he later develops a small rise in intraocular pressure or an "abnormal" outflow facility. Chronic simple glaucoma and low tension glaucoma are much more likely to be manifestations of a disease process in which many factors assume varying importance in interfering with perfusion of the optic nerve head.

A clearer recognition and understanding of the factors concerned in producing low tension glaucoma—some of which have been identified—and the significance of intraocular pressure as an important but by no means the only factor leading to ischaemia of the optic nerve head, may help to answer some of the enigmas of chronic simple glaucoma itself.

I should like to acknowledge the help of Dr. V. P. Sweeney, who carried out the neurovascular assessments, and to thank Dr. R. W. Morgan for epidemiological support.

COMMENTARY

TREATMENT OF LOW TENSION GLAUCOMA

A patient who presents with infarction of the optic nerve head without a preceding shock-like state should have everything done that is possible medically to try and reduce the intraocular pressure in the hope of manipulating the only factor that can be manipulated ophthalmologically. It is equally important for the cardiologists and physicians to treat the patients for other abnormalities, such as congestive cardiac failure, anaemia, and arrhythmias, in order to make the perfusion of the optic nerve head efficient. The use of strong miotics and long-acting Diamox is indicated in this condition and on occasions even surgery may be indicated for further reduction in intraocular pressure.

Open-angle glaucoma

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Statistics of the distribution of eye tension do not permit us to distinguish between normal and pathological pressure. Mathematical statistics represent a distribution of discrete values by a smooth continuous curve going on to infinity. Every point of this curve has a defined probability and only this probability distinguishes the different abscissae of the curve. 15 mm.Hg is a frequent, 40 mm.Hg a rare ocular tension. Mathematically—that is all. Nothing is said about normal or pathological. Only the connection between intraocular pressure distribution and visual field decay allows a distinction between the probable normal and the probable pathological. Not because 26 mm. is rare in the general pressure distribution curve, but because many patients with long-standing pressure around

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26 mm. show visual field defects, the probability is high that a pressure of 26 mm. in an individual case is dangerous. There is no long way from here to the statement that only when signs of specific visual field decay can be demonstrated is the diagnosis of simple glaucoma allowed.

From Drance (1967) we know that "baring of the blind spot" by an inner isopter—I insist: not an absolute arcuate scotoma—is a state through which every concentric shrinking visual field may pass, *e.g.* the field of every ageing person. It is *per se* not characteristic of glaucoma. Aulhorn and Harms (1967) insist that the first characteristic glaucomatous scotomata are generally small paracentral ones not easily detectable. Forgive me if I add, after a long experience, that good perimetry is an art which is not very common.

All these points help to promote the tendency among ophthalmologists to call an increased intraocular pressure glaucomatous only when quite remarkable paracentral field defects are found. There is a great danger that, by slowly forming the notion of "ocular hypertension" as a reality fundamentally different from glaucoma, the feeling is created that it is and will be impossible to make a diagnosis just when it should be made, *i.e.* before a visual field defect arises. There is thus a risk that we may fall back to the situation which existed 50 years ago with all its implications.

To avoid great field damage, the measurement of cup/disc relation and automatization of perimetry may be of much help. But to solve the fundamental problem of avoiding any damage at all it is necessary to find a quantitative relationship between intraocular pressure and visual field decay in every individual case. To make only a first modest step in this direction is the object of the following paper.

The hypothesis today, well supported by facts, is that which was formulated by Gafner and Goldmann (1955). It may be useful to give a translation of parts of the German text, for those of you who are unfamiliar with the language. We then wrote:
"In terms of haemodynamics it can be said that the following types of vessels are influenced by intraocular pressure:

"**(1)** Vessels which give off only tiny negligible branches outside the pressure influenced area (*e.g.* the central retinal artery).

"**(2)** Vessels giving off wider branches before their entrance into the eye; but these branches still have a circulation much smaller than the major vessel, *i.e.* their resistance is high compared with the resistance in the major vessel (some posterior uveal vessels).

"**(3)** Vessels the ramifications of which, both under intraocular pressure and outside the influence of intraocular pressure, are approximately the same size. These are the vessels from the circle of Haller to the lamina cribrosa, and the nutrient vessels of the optic nerve (François)".

After the description of an electrical model and experiments with it, we came to the following conclusion:
"If the vessels which lead to the laminar region have a greater resistance than the vessels of the uvea and if they are shunted to optic nerve vessels not under the influence of intraocular pressure then they suffer the greatest proportional loss of blood flow of all intraocular vessels when the intraocular pressure increases".

Since that was written the excellent work of Hayreh (1969a) has shown that the central artery of the optic nerve is an extreme rarity, but he, as well as Ernest and Potts (1968), has shown that nevertheless just the conditions exist on the disc which our hypothesis demanded. The vessels of the papilla lie parallel to an extraocular network of vessels

not influenced by intraocular pressure. This ramification takes place rather distally from the point where central retinal and long choroidal arteries take their origin. Therefore the papillary vessels collapse when the intraocular pressure equals the blood pressure at the point where the papillary vessels issue from the extraocular vessel network (Fig. 1). Correspondingly, the papillary and peripapillary uveal circulation is less buffered against intraocular pressure changes than the retinal and most of the choroidal circulation.

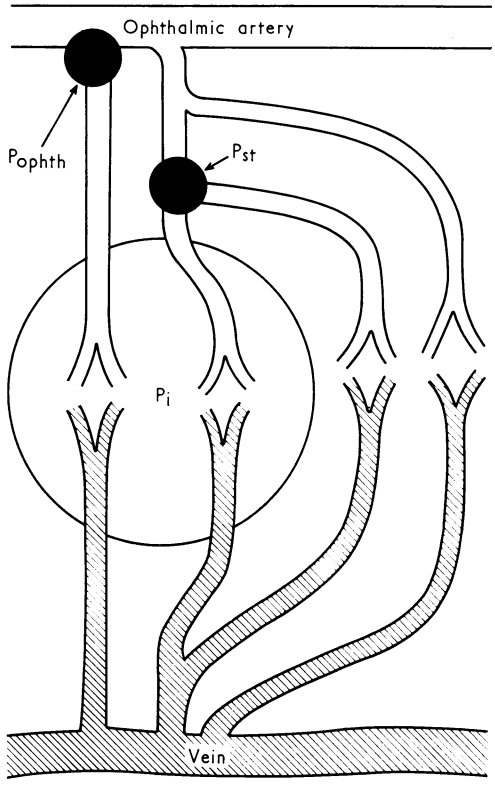

FIG. 1 *See text. Inside the eye (circle) intraocular pressure* P_i. *At the branching of vessels from the ophthalmic artery is the vascular pressure* P_{ophth}. *At the branching off of the disc vessels the pressure is* P_{st}

Therefore the circulatory hypothesis is well supported by facts. It demands the determination of the following entities:

(1) The intraocular pressure (P_i);

(2) The shunt or stop pressure, *i.e.* the blood pressure at the point where the disc vessels, influenced by intraocular pressure, branch from the extraocular vascular network free from the influence of intraocular pressure (P_{st});

(3) A quantity characterizing the sensitivity of the examined nerve fibre bundle to anoxaemia;

(4) A quantity characterizing the dependence of blood flow in the nerve fibre bundle on intraocular pressure.

We suppose that we obtain the demanded information by examination of corresponding areas of the visual field. I emphasize that values 2, 3, and 4 are specific for every point of the visual field and may be expected to be different from visual field area to visual field area, *i.e.* from fibre bundle to fibre bundle.

Our method was crude and troublesome, but it was a first attempt to grapple with our problem. It may be shortly described as follows: determination of intraocular pressure

by applanation; determination of the differential threshold with a target of constant size (I of the Goldmann perimeter) at two places in the visual field where glaucomatous eyes almost always show remarkable differences, *i.e.* nasally above or below the midline against temporally (28° eccentr.), avoiding local adaptation; correlation of ophthalmodynamometrically-induced ocular hypertension to applanation pressure. With the mentioned target, but of a luminance 1·2 times threshold, one can determine the lowest maintained pressure, at which this target disappears in the shortest time t_0. This pressure is the stop pressure P_{st} and t_0 is a measure of the sensitivity of the examined area against anoxia. Tables I and II show our first results:

(1) The stop pressure is higher temporally than nasally.

(2) The disappearance time t_0, *i.e.* the sensitivity of the examined system against anoxia, is equal nasally and temporally, its magnitude being 1 to 2 seconds.

Table I *Nasal and temporal stop pressure times*

$P_{st\ n}$	$P_{st\ t}$	Δ_{t-n}
52	60	+8
41	49	+8
49	56	+7
43	47	+4
48	54	+6
46·6	53·2	+6·6

n = nasal
t = temporal

Table II *Target disappearance times*

Nasal		*Temporal*		Δ	*Pool*		t_0	
N	M ± s	N	M ± s		N	M ± s	sec	probability
19	0·76 ± 0·869	24	0·97 ± 0·95	0·21*	43	0·88 ± 0·915	0	0·2 > P > 0·15
							1	0·5 > P > 0·45
							2	0·15 > P > 0·10
							3	0·013 > P > 0·012

* not significant N = no. of measurements M = mean

From now on two ways are possible (Goldmann, in press; Goldmann and Blok, 1971).

(1) We form a measure for "normal circulation quality," *e.g.* we determine for many normal individuals the pressure P_u at which our target disappears at the chosen place of the visual field after a *constant* time t_u. If P_{st} is the stop pressure at this point of the visual field and P_i the intraocular pressure of the untouched eye, then:

$$\left(\frac{P_{st}-P_i}{P_{st}-P_u} = B\right)$$

is a measure for "circulation quality".

Knowing by experience that pressure values below 23 to 24 mm.Hg are supported by normal individuals without damage to the visual field and calling $\overline{P_{st}}$ and $\overline{P_u}$ the statistical mean values of P_{st} and P_u of the normal population, then:

$$\left(\frac{\overline{P_{st}}-23}{\overline{P_{st}}-\overline{P_u}}(+n.6) = B_{cr}\right)$$

would indicate the limiting value of "normal circulation quality". A lower value of B than of B_{cr} found in an individual case would mean danger.

This method is very troublesome, but we would obtain statistical values which presuppose only one though very important assumption, namely that the results of this or any other short experiment are relevant for a process which goes on for years and years as the glaucomatous decay of visual functions progresses. Is it reasonable to extrapolate from our findings after 30 to 150 sec. to damage which occurs in the course of many years in simple glaucoma and from reversible changes to irreversible ones?

(2) This extremely important problem made us choose not the above-mentioned but another way (Goldmann and Blok, 1971). We reasoned that there existed the following possibilities:

(*a*) Our findings are not directly connected with the phenomena observed as visual decay in chronic glaucoma in spite of some similarities, *i.e.* they are not part of the same curve connecting pressure with visual damage time. But in spite of this shortcoming a *correlation* may exist between the results of our experiments and the behaviour of visual function in the course of glaucoma. For example, Drance and Begg (1970) have found microthromboses in the area of the disc in cases of simple glaucoma and think that they are a frequent cause of glaucomatous field defects. If this hypothesis is correct the frequency of microthromboses certainly has something to do with circulation in the nerve fibre layer as well as the results of our measurements. But between the two there cannot be more than a loose correlation.

(*b*) Let us now examine if there are indications for the existence of *one* damage time—pressure curve connecting our experiments with visual field deterioration in glaucoma. What must such a curve look like? It generally takes years to develop a characteristic glaucomatous field defect when the ocular tension is between 24 and 30 mm.Hg, and we worked at tensions between 40 and 50 mm. in minutes of time. The simplest function which may cover so wide a field should have the following properties (Fig. 2): on any point of the curve the change in damage time per change in tension is proportional to the damage time at this point of the curve, because the longer it takes to do damage the better are the chances of recovery. ΔP in the following formula is the distance from mean stop pressure (P_m). What we have called until now 'stop pressure', is a systolic pressure. By simple calculation (Goldmann and Blok, 1971), we obtain from it and the two ophthalmic pressures the mean stop pressure:

$$\frac{dt}{d(-\Delta P)} = -k.t \quad \text{or} \quad \frac{dt}{d\Delta P} = k.t \quad \ldots \quad \ldots \quad \ldots \quad \text{Formula 1}$$

If at stop pressure the disappearance time of the target is one second—as it approximately is (see Table II), then integration of Formula 1 gives:

$$\log t = k'.\,P \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad \text{Formula 2}$$

k' is $0{\cdot}434.k$. We shall call k' "circulation modulus" because it can be shown that it is a measure of the "goodness of circulation" per unit volume of active disc tissue. It can be determined from experiments with a pressure below the stop pressure (Table III).

(*c*) Our Formula 2 cannot be more than an approximation, not only because our procedure is clumsy but because it is very probable that more parameters intervene than our procedure allows us to determine. If such parameters are important, then our formula will give results far from true. If there are no other parameters or if their influence is small, our formula should be useful.

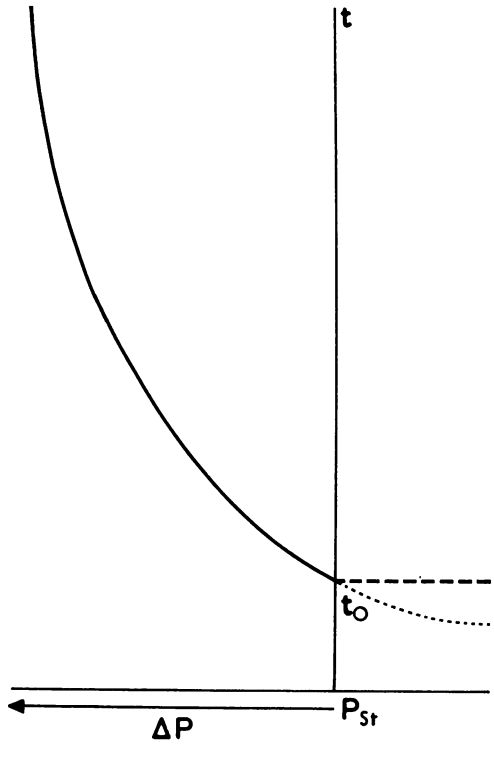

FIG. 2 *See text*

Table III

k'_n	k'_t
0·57	0·52
0·67	0·9
0·9	0·47
0·52	0·84
0·48	
0·65	
0·63	0·68

Table IV

P_{cr} n	t	Δ_{t-n}
29·5	33·5	+ 4
23·5	29·5	+ 6
26	29·5	+ 3·5
22	32	+10·0
27		
25·6	31·0	+5·9

For practical purposes we transform Formula 2 according to the following consideration: it is known that the isopters shrink with age. On the Goldmann perimeter the change is one isopter in 25 to 30 years. That is like the effect of attenuation of the target by the addition of a 33 per cent. transmission filter. This means that the target used in our experiments (1·2 times threshold) would be reduced to threshold by age alone in 5 to 6 years. To produce a beginning glaucomatous field defect the visual decay must be quicker in the predisposed areas. Log (1 year in seconds) = 7·5; log (5 years) = 8·2. Let us call A the log of the damage time in which a glaucomatous change in the visual field is produced, as distinct from the normal senile change. Then:

$$8 \geqq A \geqq 7{\cdot}5 \qquad \text{Formula 3}$$

We can now transform our Formula 2 and call "critical pressure" (P_{cr}) that pressure which just causes glaucomatous damage in an observed region of the visual field:

$$P_{cr} = P_m - \frac{A}{k'} \qquad \text{Formula 4}$$

If our experiments and our formula give P_{cr} values for normal subjects within narrow limits, then it makes sense to say that as long as $P_{cr} > P_i$ there should be no danger; an individual P_{cr} value smaller than the normal P_i values means "low tension glaucoma". Table IV shows P_{cr} values for $A = 7{\cdot}7$ and for the nasal and temporal regions.

These values seem to be reasonable. They are the result of a first tentative approach to our problem by a method which is still far from clinical practicability. We have to

improve the procedure, and it may be that we shall also have to change some of our concepts. But the chief purpose of this paper will be fulfilled even if the hypothesis and the calculations which I have presented are erroneous, for my principal aim is to show that an efficient prevention of glaucomatous field damage is only possible by a *quantitative* determination of visual field sensitivity against intraocular pressure.

Summary

The central problem of simple glaucoma is to establish the connection between intraocular pressure and visual function decay in every individual case as early as possible. Such a connection can only be a quantitative one, either by attaining statistically "normal" values achieved in certain test experiments or by evolving formulae which allow us to determine "critical limiting pressure values" for every case from a short examination.

COMMENTARY

THE APPEARANCE OF THE DISC IN EARLY OPEN-ANGLE GLAUCOMA

The harder one tries to distinguish the glaucomatous disc from the normal disc, the less one seems able to distinguish between the different types. However, one principle that Dr. Anderson and Dr. Kirsch of Miami have found very useful is that in the glaucomatous cup there is a tendency for cupping to extend towards the upper and lower portions of the disc. It does not matter whether it is a very small cup but it does seem to be the vertical orientation that is associated with glaucoma. On the other hand, a perfectly round cup with a perfectly even rim can be considered normal, even if large.

Aetiology of angle-closure glaucoma

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The shallow anterior chamber and the angle which can and does close are well-accepted properties of the eye with angle-closure glaucoma. References will be found, for example, in Shaffer (1956) and Duke-Elder (1969b). The size and shape of the anterior chamber must result directly from the characteristics of the structures bordering it, and indirectly from the influences which determine *their* size, shape, position, and function. The *marked* shallowness in angle-closure glaucoma is, I suggest, due to a *summation of quantitatively* lesser abnormalities in other individual structures which will be considered seriatim.

I. CRYSTALLINE LENS

In angle-closure glaucoma, the pupil is on a plane considerably more anterior than that of the periphery of the iris, unlike the situation in the normal eye: this produces iris *bombé* and must be due to a relatively more anterior position of the anterior surface of the lens than is usual. Three lens-related factors are very probably involved:

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