ANGLE-CLOSURE GLAUCOMA AND INVERSE ASTIGMATISM*†

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THIS investigation was initiated by the clinical observation that patients with angle-closure glaucoma appeared to exhibit a higher incidence of inverse astigmatism than normal.

In order to determine the significance or otherwise of this observation, the spectacle refraction of 150 patients with angle-closure glaucoma was noted over a period of 5 years. The diagnosis of these cases was made on the basis of the history, clinical picture, and gonioscopy findings. 60 per cent. of the patients presented with acute congestive glaucoma; 14 per cent. with symptoms of haloes; 3 per cent. with a unilateral semi-dilated pupil; 2 per cent. with retinal artery pulsation; 2 per cent. with venous thrombosis.

The patients were grouped in 10-yearly age group intervals so that a comparison with the normal could be made and a frequency polygon was constructed (Fig. 1). In general, the glaucoma patients were in the 50–79 age group, so that their refractive errors should be compared with the normal in this age group.

FIG. 1.—Age distribution of glaucoma patients.

The refractions were undertaken independently at three different hospitals and by different ophthalmic opticians in towns served by these hospitals and at varying periods before the diagnosis of glaucoma. If an accurate refraction was not possible or if the refractive findings were post-operative, then these findings were not considered in the comparison. In all, the spectacle refraction of 253 angle-closure glaucomatous eyes was noted.

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A "change of corneal astigmatism with the age" of the patient was first shown by the researches of Schoen (1887), Steiger (1895), and Pfalz (1885, 1900). The astigmatism is said to be with the rule or against the rule, according as the section of least curvature makes an angle not greater than 30° with the horizontal or vertical planes. It is found that "with advancing years the physiological astigmatism with the rule decreases, whereas the percentage of cases with corneal astigmatism against the rule increases". Related to the variation is an increase in the number of cases in which the principal meridians of the astigmatism are not the horizontal and vertical planes. Helmholtz (1856-66) believed that if the cornea were not under the influence of external forces its natural form would show astigmatism against the rule, the external forces being due to the pressure of the eyelids. With advancing years the coatings of the eyeball become more rigid and eyelid pressure diminishes; the general tone of the external forces thus decreases and the fatty matter in the socket dries up. Thus the eyelid pressure gives way and the cornea assumes its natural form. The clinical researches of Martin (1885) and Pfalz (1885, 1900) demonstrated that pressure from within (probably meaning rigidity of the ocular coats) is accompanied by corneal astigmatism against the rule. However, Eissen (1888), by inducing glaucoma in rabbits, showed that inverse astigmatism developed. Duke-Elder (1949) confirmed these findings but considered that:

"The variation of regular astigmatism with age is of considerable importance. There is a general tendency for the direct astigmatism (the usual condition at birth) to decrease or inverse astigmatism to increase, but the changes are within narrow limits."

The extent of this variation in the normal ageing eye has been considered by Hirsch (1959), who statistically analysed the spectacle astigmatism of 1,606 patients over the age of 40. The technique employed was to designate the dioptre classification with the rule as plus, against the rule as minus, while cylinders and spherical refractions were classified as zero. Hirsch confirmed the qualitative observations of Helmholtz and Duke-Elder, but added that the normal physiological increase in inverse astigmatism is approximately a quarter of a dioptre for each decade (Table).

TABLE
CHANGES IN SPECTACLE ASTIGMATISM WITH AGE (HIRSCH, 1959)

Age (yrs)	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80+
Astigmatism	+0.25 ↑	+0.09 →	+0.12 →	+0.22 →	+0.27 →	+0.41 →	+0.43 →	+0.7 →	+0.81 →

The incidence of inverse astigmatism in 150 cases of angle-closure glaucoma is compared to the normal in Fig. 2 (opposite); the percentage of eyes with inverse astigmatism increases with age in each group, but it is constantly higher at all ages in cases with glaucoma than in normal subjects.

In Fig. 3 (opposite), the magnitude of inverse astigmatism is compared for the two groups. The mean dioptric measurement of inverse astigmatism increases with age in both the normal subjects and cases of angle-closure glaucoma, but in the latter the degree is higher for a given age.

FIG. 2.—Mean incidence per cent. of inverse astigmatism with age (Hirsch, 1959).

FIG. 3.—Mean degree of astigmatism with age.

We recognize there may be insufficient glaucoma patients especially in the under 50 and over 80 age groups to facilitate an accurate comparison. It is possible, however, to make a general inference that there is a higher incidence and degree of inverse astigmatism in patients with angle-closure than in the normal eye.

In order further to interpret the clinical significance of these findings, we obtained the co-operation of the ophthalmic opticians in order to determine the changes in refraction of fifty angle-closure patients over a period of years before the diagnosis or onset of glaucoma. When this information was tabulated and analysed, it was found there were no myopes; 72 per cent. of the patients were female and acute glaucoma developed in one or both eyes in 64 per cent. of cases. In the patients with acute glaucoma, 28 out of 32 were uniocular and it was initially decided to compare the astigmatism in the acute with that in the non-acute eye. In nineteen out of 28 or just over two-thirds of the cases of uniocular acute glaucoma, the eye with the higher amount of inverse astigmatism was the one first affected.

It was considered that inverse astigmatism could not be significant in angle-closure if the spectacle refraction fell into one or more of the following categories:

- Consistently spherical refraction (6 cases);
- If direct or oblique astigmatism was manifest (5 cases);
- If the indirect astigmatism was normal and the increase with age was within normal limits (2 cases).

Therefore, the correlation of inverse astigmatism and angle-closure could be considered clinically significant in 74 per cent. of cases. In order further to investigate this result it was considered necessary to discover whether this inverse astigmatism had always been present or developed before the onset of glaucoma. Seventeen case histories were then studied in which three or more refractions were available over a minimum period of 5 years. In nine cases the initial degree of inverse astigmatism was higher than normal, but the increase was within normal limits (Fig. 4A, overleaf). In eight cases, the increase in inverse astigmatism before the onset of glaucoma exceeded the normal (Fig. 4B, overleaf).

Discussion

Since 1876 attention has been focused on the importance of the part played by mechanical obstruction in the angle of the anterior chamber in the aetiology of

FIG. 4A.—Inverse astigmatism higher than normal but unchanging in degree in nine cases compared with normal.

FIG. 4B.—Changes in inverse astigmatism before onset of angle-closure glaucoma in eight subjects compared with normal.

glaucoma. Knies (1876) and Weber (1877) substantiated the retention theory of glaucoma as distinct from the hypersecretion theory by their demonstration of the presence of peripheral anterior synechiae in eyes enucleated because of glaucoma.

Czermak (1897) contributed to our knowledge the observation that the onset of an acute attack of glaucoma was the result of pupillary dilatation blocking the angle of the anterior chamber. Further support for the retention theory came from Priestley Smith (1887), who propounded the idea that retention was due to a disproportion between the size of the eyeball and that of the lens. He argued that the volume of the lens increases with age, narrowing the anterior chamber and the space between the lens and the ciliary processes—a sudden congestion of the ciliary processes producing the attack. He also explained the relative frequency of acute glaucoma among hypermetropes as the result of the lens being relatively too large for a small eye. He considered, however, that all glaucomatous eyes had anterior chambers shallower than normal—a misconception shared by others.

Raeder (1923), on clinical observations, abandoned the concept that primary glaucoma is one single clinical entity and introduced a more reasonable sub-division into shallow chamber and normal or deep chamber. This classification was advocated and supported by Barkan (1938) who further stipulated that the mechanical blockage in the shallow chamber type was due to narrowing of the entrance to the angle.

It is an undisputed fact that the fundamental pre-requisite of angle-closure glaucoma is an anatomically narrow entrance to the angle. This narrow angle seems to be a genetically controlled developmental peculiarity, in as much as Törnquist (1953), in a study of siblings of 49 patients with acute glaucoma, revealed a statistically significant shallow anterior chamber for their age.

This pre-existing shallow anterior chamber is almost a *sine qua non* in as far as angle-closure glaucoma is concerned. However, additional features in further narrowing the anterior chamber or the entrance to the angle come into play. In reviewing these features one finds that the acknowledged relevant aetiological factors are concerned purely with the posterior wall of the angle. They may be summarized as follows:

- (1) The continuous growth of the lens throughout life (Priestley Smith, 1887).
- (2) Errors of refraction. Fuchs (1911, quoted by Elliot, 1922) stated that “a disposition towards inflammatory glaucoma appears to belong principally to hypermetropic eyes, whereas strongly myopic eyes are to be regarded as having almost complete immunity against the disease”. Sugar (1941) gave statistics of refractive error in acute glaucoma which agree with Fuchs’s statement. In a series of 47 eyes with acute glaucoma, forty were hypermetropic and seven were myopic. A second series of 140 eyes with acute glaucoma showed an average hypermetropic error of 2.72 D, only seven being myopic. Rosengren (1950) showed that there was a definite relationship between the anterior chamber depth and the refractive error of the eye.
- (3) The Curran-Chandler mechanism—physiological pupil block (Chandler, 1952). There is a higher pressure in the posterior chamber as a result of the resistance to the flow of aqueous created by the contact between iris and lens at the pupillary border.

- (4) Anterior displacement of the ciliary body due to venous engorgement resulting from neuro-vascular instability (Duke-Elder, 1926). Credence is given to this mechanism by the fact that it occurs typically in persons, usually women, in the fifth or sixth decade, who are easily excitable and have an unstable vaso-motor system.
- (5) Accommodation. Forward movement of the ciliary body may result from prolonged accommodation in reading (Higgitt and Smith, 1955).
- (6) Pupillary dilatation.
 - (a) Pharmacologically induced.
 - (b) Physiological—induced by darkness and emotion.
- (7) Sudden increase in the production of aqueous, as in the water-drinking test.

Our observations would appear to indicate that the part played by the flattening of the cornea, which constitutes the anterior part of the angle, may be of significance. It is accepted that the cornea flattens with age and, according to Wolff (1905, 1908), this flattening is greater in the vertical than in the horizontal meridian.

It is possible to approximate the angle-closure associated with a specific degree of corneal flattening, by assuming a set and constant value for the internal corneal diameter—e.g. 12 mm.—and by utilizing the constants of Gullstrand's No. 2 (exact) Schematic Eye (Fig. 5).

FIG. 5.—Gullstrand's exact schematic eye No. 2.

If the anterior and posterior corneal radii of the schematic eye flatten by say 10 per cent. vertically, then by utilizing a method suggested by Bennett (1963) it is possible to calculate that the effect is to produce almost four dioptres of inverse corneal astigmatism.

The posterior corneal plane contains the posterior corneal diameter which approximates the plane of the anterior iris, and in the diagram is taken as a datum. If the perpendicular distance from this datum along the axis of symmetry to the posterior corneal surface represents the sagittal height h , then the angle θ of the anterior chamber may be approximated by a simple application of Pythagoras in the formula:

$$\tan \theta = \frac{r - \sqrt{r^2 - \phi^2}}{\phi} = \frac{h}{\phi}$$

IN THE HORIZONTAL MERIDIAN the posterior corneal radius is 6.8 mm. in the schematic eye, so that—

$$\tan \theta_{180} = \frac{6.8 - \sqrt{6.8^2 - 6.0^2}}{6}$$

and $\theta_{180} = 30^\circ 58''$

If the cornea flattens 10 per cent. vertically, the posterior corneal radius becomes 7.48, so that—

IN THE VERTICAL MERIDIAN

$$\tan \theta_{90} = \frac{7.48 - \sqrt{7.48^2 - 6.0^2}}{6}$$

and $\theta_{90} = 26^\circ 38''$

Although it is not yet accepted that inverse astigmatism is due to an increased flattening in the vertical meridian, we are working on the assumption that a narrowing of the anterior chamber in the vertical plane does take place when inverse astigmatism is present, and according to the above calculation the angle narrows by approximately one degree per dioptre of inverse astigmatism.

Although the cornea is the principal source of ocular astigmatism, other factors (*e.g.* the crystalline lens) may contribute to the spectacle astigmatism. This non-corneal astigmatism is said to be residual and averages from half to three-quarters of a dioptre against the rule. Continuing the passage quoted above (p. 442) and referring to the role of the crystalline lens, Duke-Elder (1949) states:

“This change is partly due to a decrease in corneal astigmatism generally ascribed to hardening of the coats of the eye, but largely depends upon an increase in lenticular astigmatism which usually lies in the sense opposite to the corneal.”

It has been observed clinically and reported by the Bureau of Visual Science (1955) that residual astigmatism may increase with age, and the laboratory studies of Lowrie (personal communication), employing ultra-sonography, suggest that there is a forward tilting of the crystalline lens with age. If this is the case, it would supplement the corneal astigmatism in producing an inverse spectacle astigmatism and would certainly narrow the anterior angle above.

Conclusion

The above observations tend to show that there is firstly a greater incidence and secondly a greater degree of inverse astigmatism in patients with angle-closure glaucoma than in normal subjects. The significance of these findings could fall into these categories:

- (1) The normal flattening of the cornea with age and particularly its flattening in the vertical meridian could play a significant part in narrowing the approach to the angle of the anterior chamber, particularly above. If there is a peripheral flattening of the cornea, especially above where the entrance is normally narrower, then there will be increased resistance to the outflow of aqueous through one particular segment. The result of this could be a decrease in pressure actually within the angle, providing that the outflow of fluid through the canal of Schlemm remained constant. This lessening of pressure would presumably result in the iris being sucked forwards towards the trabeculae. Such suction on the iris as a cause of acute glaucoma was postulated by Posner (1952), although not in any relationship to astigmatism. A vicious circle would thus ensue of neighbouring parts of the last roll of the iris being

- drawn towards the posterior corneal surface. It could be that the known beneficial effect of a peripheral iridectomy would be in its equalization of the pressure throughout the anterior chamber, *i.e.* on both sides of the last roll of the iris.
- (2) It could be argued that raised pressure within the globe produces inverse astigmatism. The clinical researches of Martin (1885) and Pfalz (1885, 1900), together with the laboratory researches of Eissen (1888) demonstrated that increase in pressure in the normal eye is accompanied by corneal astigmatism against the rule. On the other hand, however, there is the evidence of more accurate observations by Bangerter and Goldmann (1941) that, in closed-angle glaucoma, the closure of the angle precedes the elevation of the ocular tension.
 - (3) Since inverse astigmatism is almost invariably found in octogenarians, it is possible that glaucomatous eyes could be regarded as being prematurely senile.
 - (4) The development of inverse astigmatism in patients with shallow anterior chambers should perhaps be regarded as an additional warning sign of the development of angle-closure glaucoma.

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