AXIAL LENGTH MEASUREMENTS AND FUNDUS CHANGES OF THE MYOPIC EYE
I. THE POSTERIOR FUNDUS*

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THE POSTERIOR FUNDUS CHANGES of the myopic eye are as striking as they are unique. They are the clinical basis for the diagnosis of pathological myopia and can effect an incapacitating loss of vision in the later stages of the disease.

These fundus changes have generally been assumed to be the consequence of increased axial elongation of the globe with the attendant mechanical tissue strain and vascular changes that occur secondary to a process of stretching. However, a definite parallel between the degree of myopia and the severity of its fundus changes has never been established. Partly because of this, the concept of a biomechanical pathogenesis for these changes has been increasingly challenged and their origin ascribed rather to abiotrophy.[1,2]

This problem is not solely academic, for the nature of these changes determines the possibility of their prevention and response to various therapeutic approaches.

The degree of refractive myopia, in general, reflects the ocular axial length. Although axial length is the primary determinant of refraction, the effect of variations in corneal power and lens power, each in the order of approximately ± 5 diopters, are such that a parallel between the degree of myopia and fundus changes due to axial elongation could be difficult to demonstrate.

Prior to the clinical use of ultrasound, the measurement of the axial length of the eye by optical or roentgenologic methods was

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complex and, in the case of the latter method, potentially dangerous. The ultrasonic method has proved at least as accurate and considerably more simple than these older methods.[3,4,5]

With the availability of this method of axial measurement it was possible to undertake a large-scale study to statistically determine the relationship between axial elongation and the fundus changes of myopia.

MATERIALS AND METHODS

The clinical material in this investigation consisted of patients referred to the Myopia Clinic of the Manhattan Eye, Ear and Throat Hospital. Approximately 100 randomly selected hyperopic and emmetropic patients were included in the study group to obtain samples in eyes of shorter axial lengths. Each eye was dilated and the fundus examined by both direct and binocular indirect ophthalmoscopy. Biomicroscopic study of the fundus was added in selected cases. The axial length of each eye was measured by ultrasound.

The ultrasonic equipment employed has been described in detail elsewhere.[6,7] Both 8 MHz and 10 MHz focused transducers were used with delay columns or, in the earlier stages of the study, in direct contact with the cornea. The ultrasonic receiving and display unit was modified so as to permit direct millimeter readouts of each axial length component. In this investigation only a composite reading was taken using an acoustic velocity of 1,532 meters per second. This introduces a small error because of the presence of the lens.[8]

In all, 1,437 eyes were examined in this manner. The eyes were divided on the basis of age into three groups. Group I consisted of eyes of patients up to and including 19 years of age; in Group II were those of patients aged 20 to 39 years inclusive; and in Group III were those of 40 years and above. All cases in which a complete examination could be performed were included in this study with no attempt made to obtain a balanced sampling of eyes. This produced a disproportionately high representation of Group I females (Table 1).

RESULTS

Five fundus changes were found to be associated with increased axial length of the eye. These were: optic nerve crescent, chorioretinal atrophy, the central pigment spot (Fuchs's), lacquer cracks, and posterior staphyloma.

TABLE 1. MYOPIA STUDY GROUP – AGE AND SEX DISTRIBUTION

	No. of patients with axial length (mm) in following ranges													
	20.0–21.4	21.5–22.4	22.5–23.4	23.5–24.4	24.5–25.4	25.5–26.4	26.5–27.4	27.5–28.4	28.5–29.4	29.5–30.4	30.5–31.4	31.5–32.4	32.5–33.4	33.5–36.6
Group I (19 years and less)	22	64	75	125	116	175	83	45	18	10	7	6	2	1
Group II (20through 39 years)	0	11	22	28	25	26	15	26	25	17	8	15	5	3
Group III (40 years and above)	9	31	48	39	35	48	44	54	39	46	30	16	6	17
Male	7	31	52	80	61	119	61	57	37	38	23	28	9	11
Female	24	75	93	112	115	130	81	68	45	35	22	9	4	10

INCIDENCE AND CHARACTERISTICS OF MYOPIC FUNDUS CHANGES

Crescent

Retraction of the lamina vitrea complex (pigment epithelium – lamina vitrea – choriocapillaries) from the optic nerve margin was seen in 1,032 eyes. The position of the crescent in relation to the optic nerve was usually temporal but was seen in all quadrants as well as extending completely around the nerve. The types of crescents seen and their prevalence are set forth in Figure 1. Chorioretinal atrophy at the optic nerve has the tendency to be peripapillary and have sharp borders. This could result in an erroneously high number of annular crescent forms. When eyes displaying atrophy at the optic nerve were eliminated from consideration, the percentage of eyes with temporal crescent increased to 71 per cent, whereas that for annular crescent decreased to 17 per cent.

The incidence of crescent formation in eyes of different axial lengths is seen in Figure 2. It demonstrates a steady rise from 0 per cent in eyes of 20.0–21.4 mm diameter to 100 per cent in all eyes of 28.5 mm length and above. The regression coefficient for the prevalence of crescent in eyes with axial lengths measuring from 21 mm to 29 mm is highly significant ($p < 0.01$).

As the axial length increased, the tendency towards annular crescent formation increased. This could be noted in the lowest age group (I) where the annular crescent became predominant over the temporal form at the axial diameter of 30.5 mm and remained so above this level. This same effect was present in the oldest age group (III) but here the temporal crescent was exceeded at 27.5 mm. This 3-mm discrepancy is probably caused by peripapillary atrophy. All crescents regardless of position tended to be larger in size as the axial length of the globe increased.

Chorioretinal Atrophy

Areas of circumscribed chorioretinal degeneration were noted most often to involve the vicinity of the optic nerve and the posterior pole. The earliest degenerative changes usually involved the crescent edge with peripapillary atrophy seen as a later development. Posterior pole involvement in the early stages consisted of focal patches of absent pigment epithelium and choriocapillaris associated with a variable amount of pigment clumping within, or on the border of, the lesion. These regions later became confluent with the formation of large geographic areas of atrophy often merging with an enlarged peripapillary degenerative zone.

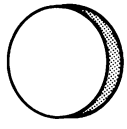
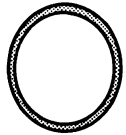
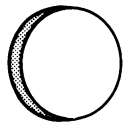
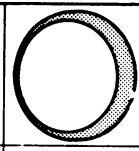
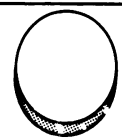
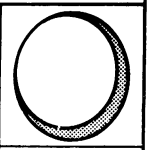
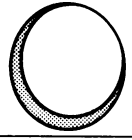
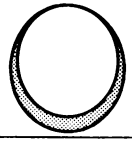
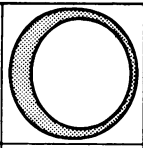
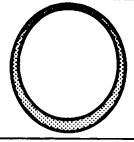
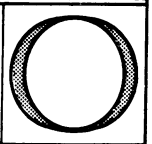
OPTIC NERVE CRESCENT: TYPE AND INCIDENCE						
APPEARANCE (LEFT EYE)						
NAME	TEMPORAL	ANNULAR	NASAL	TEMPORAL-ANNULAR	INFERIOR	TEMPORAL-INFERIOR
ALL CRESCENTS TOTAL 1032	62%	25%	3%	2.7%	2.5%	2.3%
CRESCENTS WITHOUT PERIPAPILLARY ATROPHY 841 TOTAL	71%	17%	2.9%	2.4%	2.7%	2.2%
APPEARANCE (LEFT EYE)						
NAME	NASAL-INFERIOR	TEMPORAL-INF.-NASAL	NASAL-ANNULAR	INFERIOR-ANNULAR	SUPERIOR	TEMPORAL-NASAL
ALL CRESCENTS TOTAL 1032	<1%	<1%	<1%	<1%	<1%	<1%
CRESCENTS WITHOUT PERIPAPILLARY ATROPHY 841 TOTAL	<1%	<1%	<1%	<1%	<1%	<1%

FIGURE 1

Types and incidence of crescent formation.

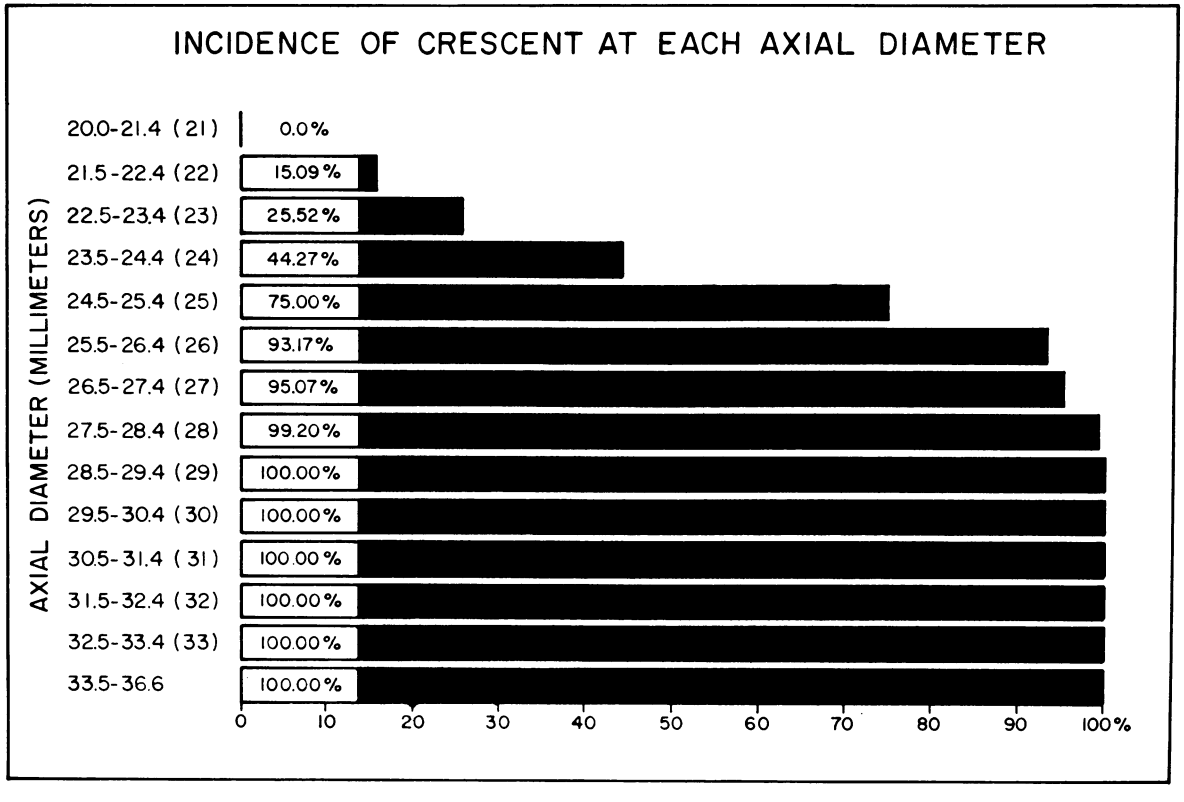


FIGURE 2
Incidence of crescent at each axial diameter.

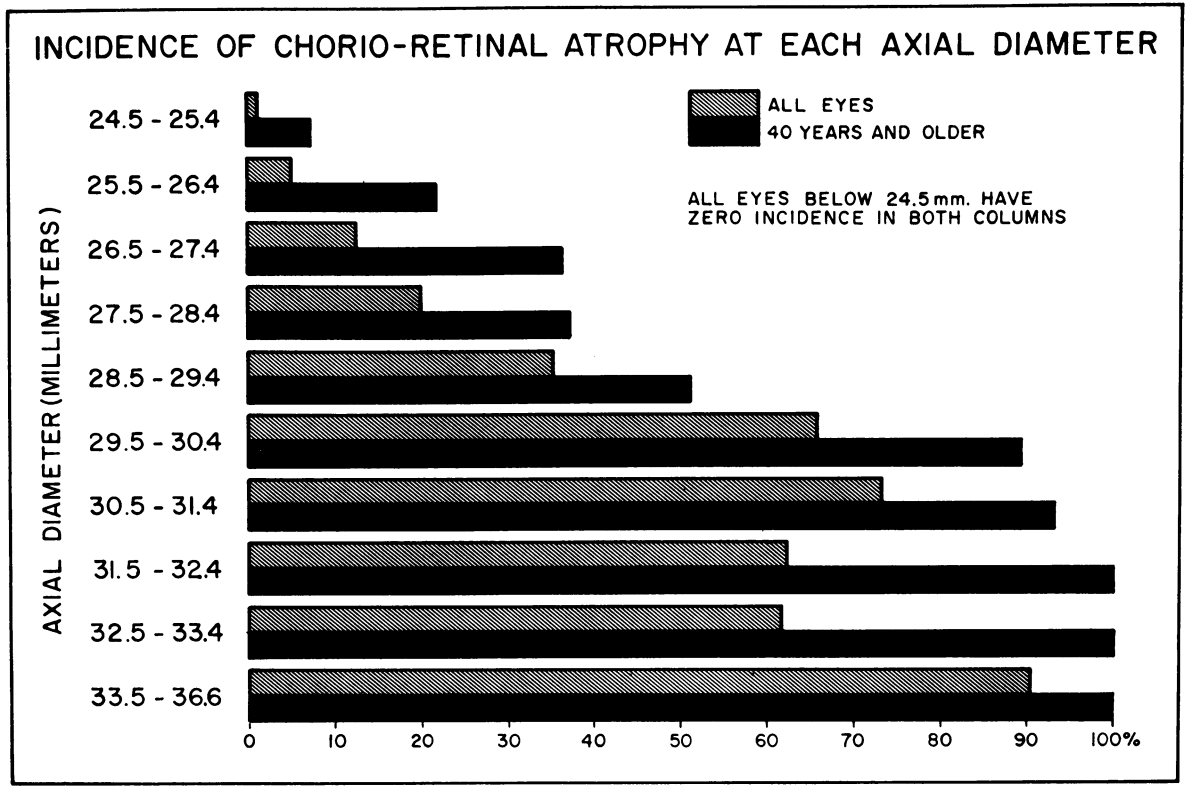


FIGURE 3

Incidence of chorioretinal atrophy at each axial diameter.

Twenty-three per cent of all eyes above 24.5 mm demonstrated atrophy with a prevalence which increased from 1 per cent in the 25-mm eye to a high of 90 per cent at the greatest diameters. The regression coefficient for the incidence of chorioretinal atrophy in eyes of this range of axial length is significant not only in Group III ($p < 0.01$) but also in Groups I and II combined ($p < 0.01$). The effect of age also was evident in the incidence of this change. Figure 3 compares the incidence of atrophy in eyes of all ages to that of Group III.

Although the effect of age can be strikingly demonstrated in this manner, it is of greater interest to consider the occurrence of atrophy in eyes of each age group at several axial diameters. Table 2 illustrates in this way the marked effect of age as well as axial elongation on the pathogenesis of this fundus change. When the regression coefficient of the prevalence of atrophy in eyes measuring from 23.5 mm to 36.6 mm for Groups I and II is compared to that of Group III, the difference between the two coefficients is statistically significant ($p < 0.01$).

TABLE 2. MYOPIC CHORIORETINAL ATROPHY–AGE GROUP INCIDENCE AT FOUR AXIAL LENGTHS

Axial length (mm)	Age Group I	Age Group II	Age Group III
27	0/76 (0%)	2/15 (13%)	16/44 (36%)
28	0/44 (0%)	5/26 (19%)	20/54 (37%)
29	1/18 (5.6%)	7/25 (28%)	20/39 (51%)
30	1/10 (10%)	6/17 (35%)	41/46 (89%)

In Group-III patients the area of atrophy was greater in eyes of highest axial diameters. The eyes of female patients displayed an 8 to 20 per cent greater incidence of atrophic changes at axial lengths below 27.4 mm. Above this diameter the incidence of such changes became only slightly greater for eyes of females. The youngest patient to demonstrate atrophy was a 14-year-old male with Marfan's syndrome.

Central Pigment Spot (Fuchs's)

A rounded, black area of variable diameter at the macula occurred in a total of 28 eyes of 26.5 mm or more axial length. This represents an incidence of 5.2 per cent in such eyes. It was bilateral in 4 patients. The eyes of females were affected more frequently than those of males in a ratio of almost two to one (18 : 10). The overwhelming

majority of cases (22) occurred in Group III. The youngest patient in which this change was noted was a 31-year-old female.

Figure 4 presents the frequency of the central pigment spot at various axial lengths. The regression coefficient for the incidence of the Fuchs's spot in eyes measuring 25.5 mm to 36.6 mm showed no statistical significance.

Lacquer Cracks

Yellow-white lines of variable orientation and diameters were seen at the posterior pole in 23 eyes of 26.5 mm diameter or more. This corresponds to an incidence of 4.3 per cent. The eyes of males were affected more often than those of females in a proportion of almost two to one (15 : 8). The greatest incidence of lacquer cracks was noted in the second age group where they were found in 13 eyes. The youngest patient to exhibit this change was a 19-year-old male; the oldest was a 51-year-old male. The lacquer cracks were bilateral in seven patients. Their occurrence at each axial length is found in Figure 4. The regression coefficient for the incidence of lacquer cracks in eyes measuring 25.5 mm to 36.6 mm showed no significance.

Posterior Staphyloma

A posterior ectasia or staphyloma is probably present in all eyes of great axial length. As a recognizable ophthalmoscopic entity, however, a sharp or abrupt edge is necessary for this classification. This fundus change was present in 102 eyes of 26.5 mm axial length or more (19 per cent). Three varieties of posterior staphyloma were seen. In one eye a peripapillary staphyloma was noted and in two others nasal staphylomas were present. In the latter instance the staphyloma edge was seen at the temporal border of the optic nerve with the ectasia involving the nasal fundus. In the remaining eyes, the staphyloma originated at, or, more commonly, one to four disk diameters nasal to, the border of the optic nerve in an abrupt out-pouching. Steplike configurations were frequently encountered. The sharp edge of the staphyloma was usually lost as it was followed temporally above and below the region of the optic nerve. In a few instances, however, a recognizable edge could be followed about the entire posterior pole. This staphyloma edge, if present temporally, was considerably less abrupt and could be followed as far anteriorly as the equator of the globe. The ectatic area of the fundus was pale with a prominently visible choroidal vasculature in comparison to the rest of the fundus.

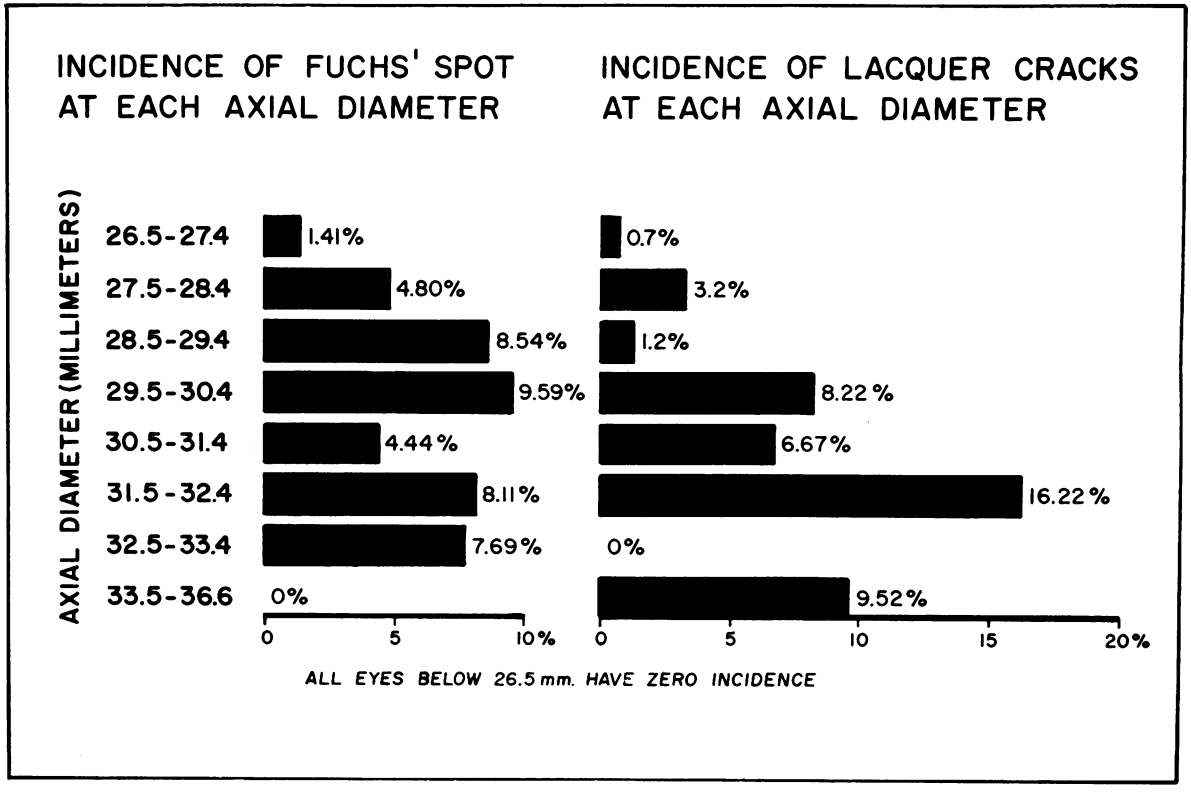


FIGURE 4

Incidence of pigment spot (Fuchs's) and lacquer cracks at each axial diameter.

Staphylomas were seen in males slightly more often than in females (55 : 47), were bilateral in 36 patients, and could be detected in all age groups. The youngest case was a 4-year-old male. The greatest prevalence was found in Group III, where 30 per cent of eyes over 26.5 mm in length exhibited this change. Group II had an incidence of 25 per cent, whereas in Group I it was seen in only 4.6 per cent of eyes.

The frequency of posterior staphyloma formation at various axial lengths of the eye is set forth in Figure 5. This increased steadily from a low of 1.4 per cent at the 27-mm diameter to a high of 71 per cent in eyes with greatest axial diameters. The regression coefficient for the incidence of staphyloma in eyes measuring 25.5 mm to 36.6 mm is highly significant ($p < 0.01$).

INTERRELATIONSHIPS OF MYOPIC FUNDUS CHANGES

The interrelationships of eyes exhibiting myopic fundus changes are set forth in Table 3.

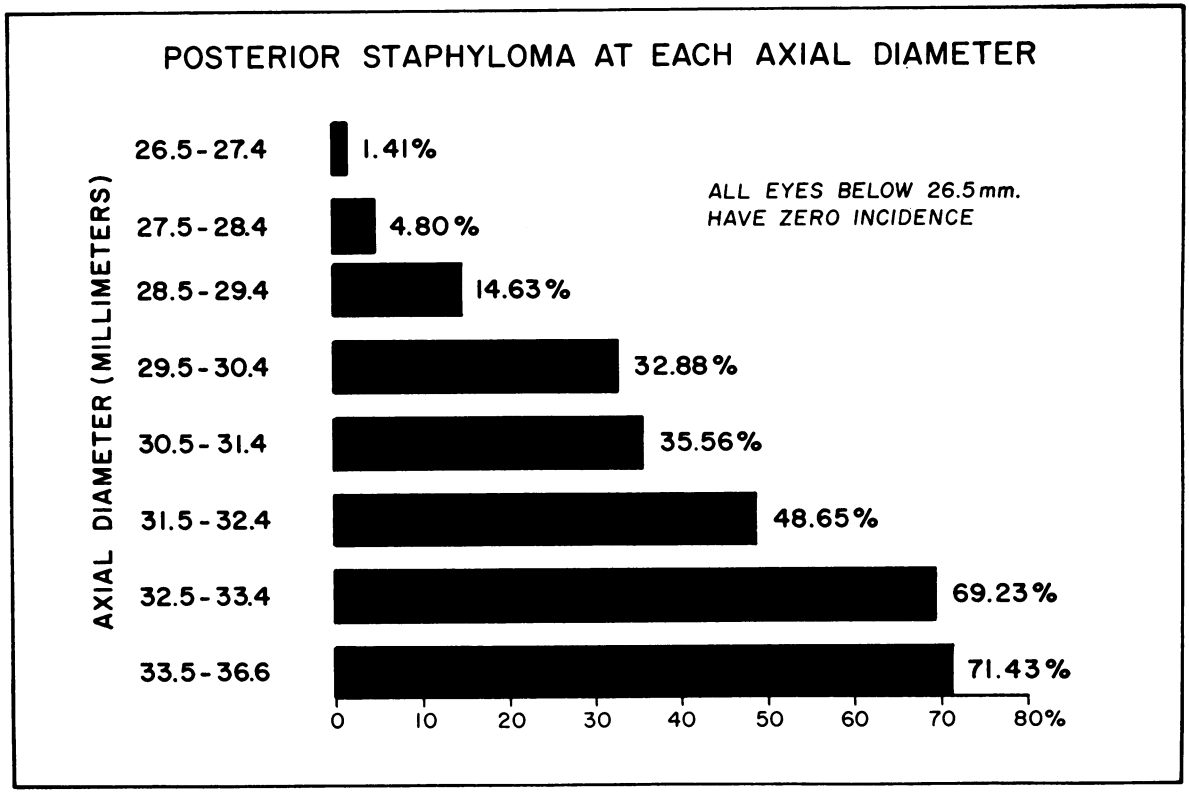
Crescent formation was present in every eye with chorioretinal atrophy. There also was noted a tendency for these atrophic changes to involve that area of the fundus ipsilateral to the sector crescent. This occurred not only at the crescent margin, but also in fundus areas beyond the immediate vicinity of the disk. Hence, the eye with temporal crescent tended to show atrophy at the posterior pole. With a nasal crescent the fundus areas nasal to the optic nerve were preferentially involved, whereas with the inferior crescent the lesions were found in the area below the optic nerve. Chorioretinal atrophy was most commonly associated with the annular type of crescent (56 per cent). In general, a positive relationship between size of crescent and degree of chorioretinal atrophy also could be discerned.

The Fuchs's spot was always accompanied by crescent formation with the annular crescent forms most commonly encountered (64 per cent).

All eyes exhibiting lacquer cracks had crescent formation. Here, however, the temporal crescent was most frequently seen (48 per cent).

All eyes with staphylomas were noted to have crescents. The annular crescent predominated and was seen in 59 per cent of these eyes.

Twenty-five of the 28 eyes with the Fuchs's spot also showed chorioretinal atrophy (89 per cent). The atrophy tended to involve both the peripapillary and posterior fundus areas concomitantly.



FIGUBE 5
Incidence of posterior staphyloma at each axial diameter.

TABLE 3. INTERRELATIONSHIPS OF MYOPIC FUNDUS CHANGES

	Crescent	Chorioretinal atrophy	Fuch's spot	Lacquer cracks	Posterior staphyloma
Eyes with crescent		218/1032 (21.1%)	28/1032 (2.7%)	23/1032 (2.2%)	102/1032 (9.9%)
Eyes with chorioretinal atrophy	218/218 (100%)		25/218 (11.5%)	9/218 (4.1%)	79/218 (36.2%)
Eyes with Fuch's spot	28/28 (100%)	25/28 (89.3%)		1/28 (3.6%)	9/28 (32.1%)
Eyes with lacquer cracks	23/23 (100%)	9/23 (39.1%)	1/23 (4.4%)		9/23 (39.1%)
Eyes with posterior staphyloma	102/102 (100%)	79/102 (77.5%)	9/102 (8.8%)	9/102 (8.8%)	

Fuchs's spots occurring in Group III eyes were associated with chorioretinal atrophy in all but one eye, that of a 48-year-old female.

Nine of 23 eyes with lacquer cracks showed atrophy (39 per cent). Seventy-nine eyes with staphyloma showed chorioretinal atrophy (77.5 per cent) which usually involved both the peripapillary area and the fundus area of the staphyloma. No eyes with staphyloma were seen without accompanying atrophy in patients above the age of 40. In the reverse relationship 36 per cent of eyes having atrophy also had staphyloma formation. Nine of 23 eyes with lacquer cracks (39 per cent) also showed staphyloma formation.

Only one case of lacquer cracks and a coincident Fuchs's spot was encountered.

COMMENTS ON RESULTS

The close association of crescent formation with increased axial diameters of the globe found in this investigation agrees with the studies of Stenstrom[9] and Otsuka and Kondo.[10] In these, however, a less abrupt rise in incidence was noted in the intermediate axial lengths. For example, in this series at 25 mm axial length, 75 per cent of eyes show crescents, whereas in the two earlier studies this figure was not reached until the eyes measured about 26.5 mm. This discrepancy is attributable to three factors:

1. Crescent classification: In this study any degree of retraction of the lamina vitrea complex from the border of the optic nerve as detected by direct ophthalmoscopy was classified as crescent formation. This alone could have the effect of considerably increasing the incidence of this change.

2. Age of the patient examined: 64 per cent of the patients in this series at the lower and intermediate axial lengths (20.0–26.4 mm) were in Group I. At the same intermediate axial length, it is more probable that an immature eye will show crescent formation than that a mature eye will. For example, at the axial length of 24 mm, 49 per cent of Group I eyes displayed crescents as compared to only 27 per cent of Group III eyes.

3. The predominance of female eyes in the low and intermediate axial lengths: Because the eye of the female is significantly shorter than that of the male,[11,12] it could be expected to demonstrate a higher incidence of crescent formation in the intermediate range of axial lengths. In this study 60 per cent of the eyes in the diameter range of from 22.5 to 26.4 mm were of female patients.

In this investigation the incidence of temporal crescents, 71 per cent

in eyes not exhibiting chorioretinal atrophy, is between the 54 per cent incidence of Vossius[13] and the 79 per cent incidence recorded by Hertel.[14] Von Szily's[15] incidence of 87 per cent also included the temporal-annular type and is considerably higher than a combined incidence of 73 per cent of the two forms for this study.

The association of increased axial length with larger temporal crescents as well as both the simple and combined forms of annular crescents agrees with a number of previous studies.[16–18] It is also in indirect agreement with studies in which a greater amount of myopia was associated with these crescent types.[19,20]

Whereas two recent studies have detected the greater association of chorioretinal atrophy with the higher grades of myopia,[21,22] this study demonstrates a close correlation of this atrophy with increasing axial length. The importance of age in the production of this fundus change has been widely recorded in the ophthalmic literature and is confirmed by the results of this investigation.

No significant difference in the incidence of atrophy between the sexes could be discerned, however. The increased incidence noted in eyes of females in the intermediate axial lengths does not indicate their increased susceptibility to this change so much as the axial length difference between eyes of both sexes. The almost equal incidence found in eyes of greater axial diameter does not confirm the purported susceptibility of the female eye to myopic chorioretinal degeneration.[23]

The incidence of Fuchs's spots in this series, 5.2 per cent of eyes 26.5 mm and above, is slightly greater than Blatt's[22] figure of 4 per cent. The correlation between Fuchs's spots and increasing axial length is not as dramatic as with other myopic fundus changes nor can a statistical significance be demonstrated between the two. This may be because this lesion changes in morphology with time to the extent that it can become unrecognizable as an ophthalmoscopic entity. Fuchs's[24] original description of the pigment spot noted an equal incidence between the sexes. The 2 : 1 plurality of the female in this series agrees more with Campos's[25] large series in which eyes of females were affected in a 4 : 1 proportion over those of males. Fuchs's report, furthermore, noted an absence of related fundus changes with the pigment spot. This study reveals an invariable association with crescent formation, an almost equally strong association with chorioretinal atrophy (89 per cent) and, to a lesser extent, one with posterior staphyloma formation (32 per cent).

Lacquer cracks were detected predominantly in males and at a younger average age than Fuchs's spots. Only 35 per cent of lacquer cracks were found in Group III eyes. Like the pigment spot it has erratic prevalence levels at increased axial diameters, so that a statistical significance with increasing axial length in the range of 25.5 mm to 36.6 mm could not be demonstrated. The coincidence of early chorioretinal atrophy in these younger eyes and the decreased incidence of lacquer cracks in older eyes suggests the clinical progression of these lesions with their eventual incorporation into larger areas of chorioretinal atrophy wherein they, like the Fuchs's spot, also become unrecognizable.

However, axial elongation of the eye would appear to play some role in the occurrence of both Fuch's spots and lacquer cracks. The regression coefficients for these fundus changes in the axial length range of 20.0 mm to 36.6 mm shows significance in both instances.

Posterior staphyloma showed a strong correlation with increasing axial length, and was considerably more common that either Fuchs's spots or lacquer cracks. It was always associated with crescent formation usually of the annular type (59 per cent) which is decidedly less than the invariable association of these two changes found in Weiss' small series.[26] The association of staphyloma with chorioretinal atrophy is particularly marked in eyes of patients over 40 years of age (100 per cent). It is the frequent occurrence of staphylomas in young eyes prior to the age at which degeneration would be expected to supervene that accounts for the fact that chorioretinal atrophy is often absent in these eyes.[27]

The increase in the incidence of staphylomas with age would concur with the natural history of the disease insofar as there is the tendency towards gradual progression of pathologic myopia during the patient's life.[23,28]

DISCUSSION

Although this study demonstrates a strong correlation between increasing axial length of the eye and at least three myopic changes of the fundus, it does not rule out the possibility of another common correlating factor. Such a factor could be a derangement of the retinal pigment epithelium. During embryologic development, the pigment epithelium induces the formation of both the choroid and sclera.[29,30] An abnormal retina may induce the formation of a sclera deficient in quantity, quality, or both with resultant ectasia under the stress of

normal intraocular pressure.[1,31] In this way abiotrophic degeneration of the retina could ensue independent of the enlargement of the scleral shell.

However, there are aspects of this study which indicate that biomechanical factors are operative to some degree in these fundus changes. The crescent, which is so strongly associated with myopia, of itself cannot be considered an abiotrophic entity. It can be seen in emmetropic and even hyperopic eyes and is usually found in the absence of ocular disease. The crescent, being closely associated with increased axial length, must be considered the result of a disparity in area between the scleral shell on one hand and the lamina vitrea complex on the other.

The origin of myopic chorioretinal atrophy is not indicated clearly by this study. The frequent onset of atrophy at the edge of the crescent and the high correlation of chorioretinal atrophy with increased axial length, crescent, and staphyloma would suggest that at least some element of biomechanics is involved in this disease process. The association of atrophy and crescent is further remarkable in that the incidence and severity of the chorioretinal degenerative changes are related to crescent type and size. In addition, the occurrence of myopic degeneration in a connective-tissue disease such as Marfan's syndrome must also be noted.[32] Although atrophic changes in myopia must continue to be considered possibly abiotrophic in nature, this study strongly suggests at least some element of biomechanical effect.

No conclusion as to the origin of Fuchs's spots or lacquer cracks can be deduced from this material. Although strongly associated with cresent and to a moderate degree with staphyloma formation, these changes could be abiotrophic in nature.

Posterior staphyloma, as previously noted, could be the result of a congenitally defective sclera and, indirectly, an abiotrophic defect. The progressive increase in incidence of this change with age as detected in this series is more compatible with the concept of a scleral disease process, however, as are their steplike configurations. The occurrence of anterior staphylomas is associated with acquired scleral disease and cannot be attributed to defects of the ciliary epithelium. In this series one eye with posterior staphyloma and two with chorioretinal atrophy were observed in three patients with Marfan's syndrome and high axial diameters. The occurrence of these changes noted in this study and the report of posterior staphyloma in the Ehlers-Danlos syndrome[33] are also more consistent with a connective-tissue disease process. In view of these considerations, it is difficult to

ascribe the occurrence of *acquired* posterior staphylomas to retinal abiotrophy.

Identical twin studies can be of considerable help in the delineation of a genetic abiotrophic disease process. Two reports in the literature are of particular interest in this relation. Orth[34] has reported identical twins, one of whom was emmetropic with no fundus abnormalities, whereas the brother's refraction was −26.00 in the right eye, and −25.00 in the left. These myopic fundi showed peripapillary crescents with stretching changes at the posterior pole of both eyes. Marchesani[35] reported uniovular twins with identical corneal power and astigmatism in all four eyes. The refraction of three of these eyes was −18.00 diopters and their fundi showed large annular crescents with degenerative changes in the macula. The remaining eye at −10.00 diopters showed only a small temporal crescent without degenerative changes in the macula. Marchesani concluded that these myopic changes were dependent upon stretching and were not heredo-degenerative in nature.

The myopia of prematurity can be of value also in resolving this question. Although at the present time these patients are too young to exhibit the late changes associated with high myopia, their posterior fundus changes are indistinguishable from pathologic myopia in the early stages of the disease. Crescent, as well as pallor, tesselation, and fine pigment mottling of the fundus all can be detected in youngsters with a history of premature birth with oxygen treatment and no family history of myopia. If other myopic changes, notably chorioretinal atrophy, supervene in later years, their biochemical pathogenesis will be strongly indicated. Prematurity is an important factor in producing a discordance of refraction in uniovular twins,[36] and it is this mechanism that possibly accounts for the refraction disparities contained in the twin studies of both Orth and Marchesani.

SUMMARY AND CONCLUSIONS

This study of the relationship of myopic fundus changes and axial length in 1,437 eyes obtained the following results.

1. Crescent formation is directly related to increased axial length in both incidence ($p < 0.01$) and type and size.

2. Chorioretinal atrophy is related to increased axial length ($p < 0.01$), but age also plays an important role in the production of this defect ($p < 0.01$).

3. Fuchs's spots and lacquer cracks, although occurring in eyes

of greater axial length, cannot be directly correlated with increasing axial length. This is possibly the result of changes in the morphology of these lesions with age.

4. Posterior staphyloma is directly related to increased axial length ($p < 0.01$).

5. Crescent formation is present in all eyes exhibiting other myopic fundus lesions.

6. Chorioretinal atrophy occurs very frequently in eyes with Fuchs's spots.

7. Chorioretinal atrophy occurs in eyes of patients above age 40 who have posterior staphyloma.

Although these results do not conclusively rule out abiotrophy in the pathogenesis of myopic fundus changes, the significant correlation of crescent formation, chorioretinal atrophy, and posterior staphyloma with increased axial length is strongly suggestive of some element of biomechanical effect. The occurrence of these myopic changes in patients with the connective-tissue diseases of Marfan's syndrome and Ehlers-Danlos syndrome also tend to indicate the involvement of biomechanical factors.

This conclusion is further supported by two previous uniovular twin studies and, tentatively, by the fundus changes in the myopia of prematurity.

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DISCUSSION

DR FREDERICK W. STOCKER. The authors presented a most thorough statistical analysis of the correlation of the various typical changes in the fundus of the myopic eye with regard to age and axial length of the bulbus.

When discussing the problem of myopia, or, for that matter, the refraction of the eye in general, there are several basic facts one always has to be aware of.

First, the variations in the refraction of the eye observed in the general population, within the range of about 5 to 7 diopters of hyperopia or myopia are plainly the result of a biologic variation, approximating a binomial curve. The refraction is the result of a combination of the refractive power of the cornea-lens system and the axial length of the bulbus. The average length of the emmetropic eye, according to Wesseley, may vary from 22 to 25 mm. If, however, one takes into consideration the higher grades, of myopia in particular, there is a definite increase of such cases, exceeding the range of the binomial curve. In this group of high myopia, the length of the axis of the bulbus is definitely the determining factor.

Second, there is no doubt an hereditary factor involved in the occurrence of myopia, but there does not exist a simple hereditary pattern. According to Franceschetti, there are several variations, such as polyallele dominant, simple recessive, and sex-linked recessive. Also, some genes appear to be quite labile as demonstrated by the occurrence of differences in identical twins.

Third, because of the enormously complex situations, extreme caution is to be exercised in interpreting statistical data, particularly in connection with possible environmental factors and therapeutic measures.

The authors do not draw definite conclusions from the results of their study as to the pathogenesis or etiology of the fundus changes. They cautiously use the term "to some degree" when they presume a so-called biomechanical factor. Taking into consideration prior publications by Dr Curtin, it is evident that they feel that an inherent weakness of the sclera, they even use the term disease, in its posterior segment may be responsible for the enlargement of the bulbus. They find some support in this belief in statistical correlations present in certain syndromes such as congenital myopia and Marfan's syndrome, in which a systemic mesodermal disorder is believed to be present. It is questionable whether findings in such comparatively rare occurrences can be applied to the vast

majority of myopes where no such disease is present. In syndromes, various individual variables may be accidentally genetically combined without necessarily being dependent on each other.

In 1943, I published a pathologic report on a rare case of myopia in a 16-year-old. The sclera was markedly thinned at its posterior pole. There were, however, no changes in either choroid or retina. This demonstrates that these changes do not occur at the time when the bulbus enlarges but later, when the process of enlargement has come more or less to a standstill. Temporal crescent and nasal super-traction of the retina were present. My observations led me to the formulation of the following theory: It is not necessary to presume that a stretching process causes the degenerative changes observed in cases of late myopia. Similar changes can be observed in other conditions, such as senile atrophy, and a crescent can occasionally be observed in emmetropic or hyperopic eyes. It seems possible that each coat of the bulbus, retina, choroid, sclera has its own inherited growth potential. While in the majority of persons there is enough co-ordination to provide near emmetropia, in myopia there would be an incongruence of growth, with the retina growing over the optic disk nasally and by its growing causing the bulbus to enlarge temporally. The degenerative changes observed much later may be due to circulatory difficulties (the vascular system is embryologically of different origin than the retina itself) or just represent an inherited tendency not unlike some senile manifestations. Waardenburg in his textbook, *Genetics and Ophthalmology* of 1961–3 seems to follow this line of thought when he says "axial myopia may be due to different genes, either by itself or as part of syndromes."

By adopting this attitude, I by no means will negate the value of therapeutic attempts, other than correcting the refractive error, to improve the situation in the highly myopic eye.

Surgical reinforcement of the posterior pole to combat further enlargement, whatever the cause of this enlargement may be, is a logical approach. Time will show how effective this measure really is. More problematic are reports of improvements obtained by medical treatments, including implantation of placenta or even injections of extracts from whole eyes as recently reported by Vance and co-workers (Bull, et mém. Soc. Franç. Opht., 82:507–24, 1970). It is difficult to evaluate statistics of that sort. A certain bias seems to be almost unavoidable, according to a sarcastic definition I recently came across: a statistician is "a man who can go directly from an unwarranted assumption to a preconceived conclusion."

I enjoyed reading this excellent and thought provoking paper, and I wish the authors success in their further pursuit of these problems.

Dr Clement McCulloch. A year ago Dr Roger West and I spent some time studying the Sioux Indians in the northern part of Ontario. The point of interest was the large percentage of high refractive errors in these people.

In general terms, a lot of Indians in the northern wastes have high myo-

pia, often high astigmatism associated with it. Secondly, at spot locations, the incidence of large refractive error is extreme. Thirdly, there are families in which high myopia is prevalent. Unfortunately it is not possible to go back several generations to prove inheritance.

I would like to ask Dr Curtin what family studies were done within their very large study?

DR CURTIN. As far as the genetic aspects of myopia are concerned, I think it plays a dominant role, although I believe some environmental aspects, such as prematurity and increased intraocular pressure do enter into the picture.

At our Myopia Clinic we have found that the incidence of glaucoma in very highly myopic eyes (and this is the same study group) goes up to 28 per cent at the greatest axial diameter, which is well above anything you would find in the normal population. In eyes below 26.5 mm we found a normal incidence of 3 per cent. So, I do think that glaucoma enters into the equation as a factor in the adult progression myopia.