CHANGES IN REFRACTION DURING LIFE*

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It is well known that the refraction of the eyes may change in the course of a lifetime, but we do not know the reasons and the rhythm that rule such changes of shape and function. The interest of research workers has long been exclusively focused on the question of the origin of short-sightedness. On this there are two opinions: the endogenous theory that considers the germ-plasma as the basis for the development of the eye and its parts, and the exogenous theory that attributes short-sightedness to the observation of close objects and other mechanical factors.

If we talk of refraction we mean total refraction. However, this does not mean a degree which is directly measurable, but a figure that results from the proportion of all optical constant values to the length of the eye's axes. This fact and the approximate value of any subjective measurement explain the difficulties that hinder work in this field.

The first step was an investigation of the frequency of different stages of refraction among certain sections of the population. Long-sightedness and short-sightedness, like all biological characteristics, exist in innumerable finely graduated degrees. The proportion of the different states of refraction found at various stages of life confirm that myopia increases more rapidly in the course of the school years than later in life. Until recently it has been maintained that learning to read and write is the chief cause of the development of myopia.

In contrast to the study of large-scale statistics, the investigation of refraction-changes during the lives of individuals has a more personal character. It is an important condition that a series of patients should visit the same oculist over a long period.

Material

In my practice in Hanover, I was lucky enough to find continuous notes made by my predecessors (Stölting and Agricola) going as far back as the year 1886. Thus I was able to draw individual curves of refraction for several patients. The horizontal scale in each graph gives the patient's age, and the vertical scale gives the refraction in dioptres.

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Fig. 1 illustrates the principle on which the graphs have been drawn. The subject, whose sight could be examined for 40 years, showed in both eyes an equal long-sightedness and a considerable astigmatism. I took only the right eye, which had full visual power. At the age of 4 years, when he came as a boy to be examined for the first time, the correction required was $+3.5$ combined cylinder $+1.0$. While the spherical component remained nearly constant during his youth, the cylindrical component rose by the 12th year from 1 to 3 dioptres, and the position of the axis of the cylinders turned from 10° to 55° . In the following years the astigmatism remained nearly constant but the spherical component went down in spite of the decline in accommodation.

FIG. 1.—Records of the right eye of a patient examined regularly from 4 to 44 years.

Fig. 2 shows a synopsis of refraction change in 110 eyes, or rather 110 meridians, of persons who could be examined periodically for at least two or three decades. As we are consulted more especially by people who show a rather complicated fault of refraction, there are many cases of astigmatism among them. In order to keep the graph clear, only the spherical component has been recorded.

The short-sighted patients are entered above the zero-line of the graph, and the long-sighted patients below. Therefore the increase of long-sightedness is graphically expressed by a descending line. The number of the hyperopes remains

far less than that of the myopes. The reason may be that the patients were selected by chance and that long-sighted people visit the oculist generally later and less frequently. In the course of a lifetime, a numerical progression can be stated on the myopic as well as on the hyperopic side of the graph, but the number of these results is too small to establish a biological law.

Observations

Fig. 2 confirms the well-known fact that with short-sighted people the curve of refraction shows a steeper ascent during the two first decades. Towards the end of growth, that is at about 20 years of age, the lines of the curves flatten. Generally, the earlier and the higher myopia begins, the greater the tendency towards a quick and steady increase, but this rule is not without exceptions. One case of myopia of 5 D begins at the age of 7 and remains constant over a longer period and others come to a standstill by the age of 13 or 15, while growth is still in progress. In these cases the curves tend to become horizontal. Blegvad (1918) demonstrated that these stationary periods at an early age are not restricted to the lower degrees of myopia, but also apply to the higher degrees of 9 or 10 dioptres and more.

On the other hand, there are degrees of refraction that after remaining stationary up to the 5th or 6th decade show a sudden and distinct increase. We shall discuss the reasons for this atypical course later on. Now and then we also see a certain lessening of myopia in the course of a lifetime, for example from 2.0 at the age of 15 to 0.5 at 65, or from 7.5 at the age of 17 to 6.5 at 54.

Some cases of excessive short-sightedness show an exceptional tendency to decrease at a later age. In one case myopia decreased in the seventh decade in one eye from 19 to 16, and in the other from 17 to 14 dioptres. With another patient we even found a decrease by steps from 17 to 8, a decline of 9 dioptres in 13 years. Such exceptions cannot be explained by a change in the length of the axis, but only by the diminution of the refraction of the cornea or the crystalline lens.

Long-sighted people show fewer dramatic changes. Here we also find steeper increases for a few years in youth, but generally the stationary forms predominate.

Individual Cases

In the entanglement of lines in Fig. 2, a good many curves coincide or cross each other, so that there is no clear view of any individual change of refraction, but we will also consider a few isolated cases.

Fig. 3 shows the curve of a slight myopia with astigmatism. It is the right eye of a doctor whose refractions on both sides were approximately equal. At the age of 11 the short-sightedness was 1.25 D; it then increased slowly to 3.5 and remained stationary from his 19th year for the following three decades. This is a classical example of the typical evolution of a simple myopia. The astigmatism was constant during all this time.

FIG. 3.—Right eye showing slight myopia with astigmatism from 11 to 47 years.

FIG. 4.—Left eye showing progressive myopia from 25 to 82 years.

As contrasted with this form of myopia which becomes stationary at the end of the period of growth, Fig. 4 shows a progressive myopia that was 6.5 D during the 3rd decade, and ascended almost continuously up to 20 D in the 9th decade. Both eyes showed not only the same refraction but also very bad myopic degeneration of the retina. The right eye was blind towards the end of the patient's life and only the left eye is represented in this graph.

Fig. 5 demonstrates the curve of a woman teacher. When she was about 30 years old we found a myopia of 6 D in the right eye and 5 D in the left. In the following decades the short-sightedness increased very slowly until her 60th year. Then there was a sudden rapid increase in both eyes, so that at 74 years of age 15 and 16 D were found. Such a result at this age cannot be explained by an increasing length of the axis but only by a change in the optical system. Indeed, just at the time of this remarkable rise there developed a cataracta

FIG. 5.—Progressive myopia in both eyes from 29 to 74 years.

FIG. 6.—Changes in an eye affected by keratoconus from 22 to 46 years.

nuclearis, that is, the crystalline lens in each eye resembled a bull's-eye window-pane. As such nuclei have a higher index of refraction, the result is an additional myopia.

Fig. 6 demonstrates the change of refraction in an eye with keratoconus, that is, accompanied by a high degree of astigmatism. At the age of 22 the refraction in the horizontal meridian was -1.0 D and in the vertical $+6.0$. In the middle of the third decade, the refraction in both meridians declined remarkably, but very soon it rose again to above the previous level, and remained at 7 D until the fifth decade.

A state of refraction, which can be demonstrated perfectly by the graphic method, is anisometropia. This form of different refraction in each eye is the very touchstone of all theories about the origin of myopia. Fig. 7 is taken from a housewife, who was already suffering at 21 years of age from a myopia of 12 D in the right eye and 5 D in the left. In this case the refraction of the better eye remained for decades absolutely constant, and in the more myopic eye, which was weak-sighted, the refraction increased only by 2 D, so that the difference remained the same during her lifetime.

FIG. 7.—Development of anisometropia from 21 to 58 years.

By contrast, Fig. 8 shows a striking “disharmony” in the change in refraction. While in the right eye the small degree of short-sightedness of 2 D remained constant during

FIG. 8.—Development of anisometropia from 14 to 62 years.

the whole of the patient's life (and even declined somewhat towards the end), the myopia of the left eye, which was higher from the beginning, ascended steeply and had attained 20 D when the patient reached the age of 62.

Such cases demonstrate that the change in refraction for each eye is determined from birth to death by endogenous factors and cannot possibly be explained by external influences.

These few examples illustrate what can be said about individual changes in refraction when the notes of many years are available. Our question was not “why” but “how” changes in refraction occur

during the lifetime of certain persons. The results therefore do not offer a basis for defending or attacking the different interpretations of the character and origin of short-sightedness. But they call for the solution of other questions seen from an ontogenetic point of view. One of these is the relation between the growth of the eye and that of the rest of the organism, especially the numerical correlations between the magnitude of the different parts of the eye and the length of the body. The refraction

curves of twins and the testing of all these problems on growing animals remain to be examined. When these questions have been explored it may be found whether outside (that is, parakinetic) influences may change the shape and function of the eye, or whether it is only hereditary factors which decide its evolution and refraction.

REFERENCE

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