THE STABILISATION OF THE REFRACTION AND ITS RÔLE IN THE FORMATION OF AMETROPIA*

BY

Professor A. KETTESY

DEBRECEN, HUNGARY

THE well-known doctrine of the late Swiss ophthalmologist Steiger about heredity in refractive errors (and in myopia) has not been generally accepted till to-day, although renowned authors supported it.

Vogt and his school played the leading party in this. On the other hand high scientific authorities opposed this and the dispute became sometimes very vigorous. Recently it was Lindner who was vehement in opposition to it, finding the cause of the "myopic process" once more in near work.

The question is of great practical significance. It is of vital importance to a young myopic patient whether he is allowed to read and study or not. As an example I can quote a case of a highly educated person, with stable myopia of 5 dioptries in healthy eyes. The myopia began 30 years ago in his 10th year. His parents were then strongly advised to place him in some rural occupation on account of his beginning myopia. It was only the sound common sense of the mother that averted such an unlucky turn in her boy's future.

As it is known, Steiger derived the refraction from the variation and combination of the optical elements, these being each separately inherited. An eye with a given axis results in hypermetropia, emmetropia, or myopia depending on the adjoined lower or higher refractive power. Since every component and the resulting refractive power has its own curve of variation, Steiger combined the 2 spherical ametropias together with emmetropia in one continuous line of refraction, H standing on the left, M standing on the right side of E.

Although Steiger founded the origin of myopia absolutely on heredity, and did not allow any exogenous factor, nevertheless near work played an important rôle in his theory. According to him myopia arose out of the curve of refraction adapting itself to the changed circumstances of life during a century of evolution, in the sense of Darwin's theory of selection and elimination. (Of course we have to complete it now with the newer concept of mutation.) With the growing importance of near

* Received for publication, November, 1947.

work the originally eliminating factor M turned, according to Steiger, into a selective value. Myopic variations could survive and multiply. Thus myopia is an acquired feature, but it was acquired by the race and not by the individual.

Neither Steiger nor his followers took into consideration the important fact that there exists between M and the two other refractions, E and H, in one respect a sharp contrast, that does not allow them to be placed straightaway into one biological unit: H, and partly E, remain mostly unchanged from early childhood, whereas M begins later and progresses sometimes till adult age. In other words, the refraction of the hypermetropic and emmetropic eye is mostly stabilized early in life, on the other hand, the myopic eye continues to change its refraction for a long time.

Therefore the separability of M from the other refractions seemed to be unquestionable. It is easy to understand that the search for exogenous causes did not cease, and that these causes were believed to be found again and again in near work.

The formation of the refraction is undoubtedly connected with the growth of the eye. The eyeball of the newborn has, an average length of 17 mm. and a capacity of 2.2 c.cm.; that of an adult is 22.5 mm. and 6.5 c.cm. It sounds strange, but we know almost nothing about the course and details of this enormous increase. Seefelder, an expert on the development of the eye, says our knowledge about the extra-uterine growth of the eye is very deficient and needs urgent completion.

About the refraction itself we know only that the newborn has an average H of 2.4 dioptries; this decreases to 1.8 D. till the third, and to 1.5 D. till the sixth year of life (Hartman, Herrnheiser, Steiger).

In the sixth year the axis of the eye has already reached the adult average. If the refractive power remained unchanged during this time, there ought to have been an average M of 14.1 dioptries.

It was Best, Wibout, Berg and others who occupied themselves with the correlations preventing this disarrangement. It was found, as it can be foreseen, that there is a positive correlation between refractive power and axis and a negative one between corneal and lenticular focal distances, but nothing was said about the substance of it.

Although it is difficult to find an explanation for a graduated correlation, it has evidently much to do with the growing of the eye. In order to maintain the right proportion between axis and focal distance during the lengthening of the axis, the refractive power has to decrease. The increase of every spherical surface is necessarily connected with an increase of the radius and therefore

with a lessening of the refractive power. The main rôle in this process must be played by the lens; its refractive power has to decrease greatly, at least 13-14 dioptres.

Any discordance in the correlation during the formation of the eye must lead to ametropia. Thus the definitive refraction must depend also on the time when growth finished, depending, of course, upon the starting values.

About the decrease of the refractive power as a consequence of growth we are not well informed, yet it seems to be finished in the 5th-6th year of life. There are two components, cornea and lens.

A decrease of the refractive power of the cornea is, as far as I know, not observed; on the contrary, if there is a noticeable change, it is an increase in the optical zone. Hence it is the lens that carries the burden of compensation. Unfortunately, the refractive power of the lens is not measurable by clinical methods. We know only that there is a gradual slowing down of production of lens fibres, compensated by a progressive sclerosis, and both are continuous during life. We have no means of expressing this process in numbers.

About the axis of the eye we are not much better informed. We know that its length varies between 20-30 mm.; it reaches even 32-34 mm. in extreme cases. In the majority of cases its growth ceases at the end of the 5th year, but in a considerable number it grows further, in some cases till the 23rd-24th year of life.

This is not very much, yet enough to supplement Steiger's doctrine about stabilisation, and to explain the formation of the refraction as follows: The two optical factors, refractive power and axis, are changing as long as their anatomical substrata are in growing. As soon as both factors come to a standstill, the refraction is stabilised. Thus the final refraction of the eye is determined:

- (1) By the combination of the inherited optical elements.
- (2) By the process of stabilisation, also inherited but independently of the optical elements.
- (3) By the correlation, this being in actual fact nothing else than the relation between the two first factors.

From the standpoint of stabilisation one can attribute to every eye a "biological value" that is independent of its refraction. On this basis all eyes may be classified into three groups:

- (1) Eyes of reduced biological value; the stabilisation takes place before the complete perfection of the refraction.
- (2) Eyes of full biological value; the stabilisation occurs together with the harmonic perfection of the eye.

(3) Eyes of surpassed biological value; the stabilisation is late, the eye is overgrowing its harmonic measures.

I might lay stress upon the important fact that theoretically every refraction is possible in every group. A hypermetropic or myopic eye is of full biological value if it is in every respect normally developed, and its ametropia is only caused by the chance combination of the optical elements. On the other hand, an emmetropic eye may be of reduced or surpassed biological value if the E is the consequence of stabilisation at a wrong time. Supposing, for example, the combination should have resulted in a H of 6 dioptres, yet stabilisation of the axis was late, the axis became therefore 2 mm. longer. The result is E, though the eye is of surpassed biological value and in its structure myopic.

Between the biological value and the time of the stabilisation there seems to be the following connection:

(1) Eyes stabilised at the end of the second year of life are of reduced biological value.

(2) Eyes stabilised at the end of the first decade are of normal biological value.

(3) Eyes that are stabilising in the 'teens are of surpassed biological value; their stabilisation may sometimes reach as far as the third decade.

The passages being continuous, the set limits are arbitrary, but they correspond well enough with the average clinical observation.

The refraction of the new-born being hypermetropic, the eyes of the first group remain in the majority hypermetropic, and those of the second, emmetropic. The clinical manifestation of the third group is necessarily myopia; it is thus easy to understand that myopia can generally appear only in the second decade, and has a progressive character—this meaning, in our view, retarded stabilisation.

The correctness of our statements could be proved only by ample material for observation, such as, for example, the observation of 1,000 eyes, each followed individually from birth till adult age, with a continuous record of the clinically measurable data: corneal and total refraction, corneal diameter, and axis. Unfortunately, there is no possibility of measuring the lens and axis. All the many mass-examinations we have at our disposal at present are only fixed cross-sections of a population. They can be treated from various statistical points of view, but they give a very uncertain insight into the happenings and changes of the individual eye.

It would be most important to find an increasing and decreasing H as well as decreasing M. It is only increasing M

that we are, to a certain degree, acquainted with. Blegvad published a remarkable paper about 64 cases of myopia observed during a long progressive period. I published some smaller material, comprising 15 cases. In both statistics there are cases when the M was stationary from early childhood: they could be explained as eyes of normal biological value, showing M of combination.

There is one definite conclusion we can draw from these tables: the majority of these eyes stabilised in the second decade.

About decreasing H we know almost nothing, though its existence is very probable. There are the cases of convergent squint which "cease" later. Such cases can be explained only by a decreasing H, when a facultative H turns into a relative one in time, that is, still at a young age, when the convergence is not yet static but dynamic. We would say that, in our view, these cases are hypermetropic refractions of slightly prolonged stabilisation. And rarely do we find in these cases E, and exceptionally even low degrees of M.

But this is only deduction and not observation. I was, and I am, continually searching after decreasing H, but up to the present time I have not found any case where I could have stated with certainty that a decrease of the refraction had occurred. Of course, this rarity can be explained by the probable assumption that hypermetropic eyes are generally very nearly stabilised in the first years of life.

Of the greatest significance, from our point of view, is the increasing H and the decreasing M (naturally with the exception of senility and disease). Such cases must be extremely rare, as we suppose that in such cases, contrary to the general rule, the axis is already fixed while the refractive power is still diminishing. Only chance could produce such a case, or very extensive mass-observations extending over many years. It was Doppel who mentioned, in the Medical Society of Vienna in 1942, eighteen cases of increasing H found in the squint-material of Lindner's clinic. His explanation was that atropine does not produce full paralysis of accommodation in children always. Lindner, who took part in the discussion, suggested a change in the refractive power.

The two deviations of the refractive curve of the adult age, the excess and the asymmetry, have been often quoted against the hereditary interpretation of myopia. By adding stabilisation to Steiger's doctrine, they seem to be explicable without giving up the hereditary basis.

It is known that the refractive curve of the new-born is a regular binomial curve, whereas that of the adult shows an

accumulation at E and a lengthening of the myopic side (Fig. 1).

Although we are far from being exactly informed about the details of stabilisation, we may assume that after birth all eyes are still growing (except the rare cases of microphthalmos). In the first 6 years about 60 per cent. are stabilised, in the following 4 years an additional 20 per cent., and the rest up to the 24th year (Fig. 2). The curve of stabilisation shows a turning point around the 6th year, with a maximum of the second differential quotient.

If we try to draw another curve, that is, showing the percentage of eyes attaining E in the marked year (of course, again only by rough estimation), we obtain a very asymmetric curve that shows a very marked maximum again around the 6th year (Fig. 3).

FIG. 1.

FIG. 2.

FIG. 3.

Collating the two curves, we see that the maximum of emmetropised cases coincides with the maximum of stabilised cases around the 6th year of life. Out of this summation an excess of emmetropia must necessarily arise. In other words, most eyes are wandering in the first decade of life from H through E towards M, but are in the majority of the cases stopped at emmetropia by the stabilisation.

From the curves the explanation of the asymmetry is apparent. At a rough guess we have to put the eyes of retarded stabilisation at 20 per cent. These are wandering from the left side of the binomial curve of the new-born gradually to the myopic side, and further, the later the stabilisation is taking place. On the left side there is a decrease, on the right side a corresponding increase, both in thickness and length.

The stabilisation being inherited too, in this interpretation the excess and asymmetry are a constituent part of the refractive curve, and typical for a given population.

The problem of stabilisation is closely connected with the general problems of growth. We know the organs and tissues of the body are subjected to very different rules of growth. For example, the central nervous system is far ahead of muscles and bones. The inner ear is already fully developed at birth.

Organs composed of various tissues must have their own particular rules of growing. The eye has, on the one hand, tissues of ectodermic origin, with rapid growth and quick development; on the other hand, tissues of mesodermic origin, with generally a very long period of growth. In the reciprocal influence they exert on each other the secret of the correlation is hidden. Primary importance must be given to the two interdependent but heterogeneous tissues, the retina and the sclera—a problem that has been already reviewed by Vogt.

Another closely related problem is that of the "extreme variant." Recent writers on the question of myopia—Tron, Scherer, Kronfeld and Devney—even if they are inclined to accept its hereditary origin, nevertheless make an exception of M of high degree, looking upon it as a common pathological process of exogenous origin. Yet extreme variants always display symptoms of morbid character. It is true a high degree of ametropia—neither M nor H—cannot be interpreted as a variant of combination, as such an eye, rightly stabilised, must be of full biological value and hence of normal function. But there seems to be no obstacle to explaining it as an extreme variant of stabilisation. Then we have as an extreme limit on the left side microphthalmos (theoretically even anophthalmos), and on the right side the highest degrees of M, both extremes showing various symptoms of degenerative and pathological character.

It is more than 10 years that I have been trying to find some practical consequences of the "doctrine of stabilisation," and to follow the fate of myopic patients from this point of view.

The parents of a child with beginning myopia are primarily interested in the prognosis. And really this is perhaps the most important practical question. To give an answer I try first to get some information upon the stabilisation of the parents and the antecedents. Then the construction of the eye itself gives some indication. Low corneal refraction (38-40 dioptries) is worse than higher values. A myopic (or even emmetropic) refraction of a child with a low corneal refraction may be already the sign of undue length of the axis, thus of a retarded stabilisation. The similar significance of the myopic conus and of the myopic fundus has been well known for a long time.

The recording of the yearly changes also gives indications. Although there are cases with periodic changes of increase and stability, the general rule is continually decreasing progression till the 20th year, and so we are able to foretell the definitive refraction approximately. Thus one commonly finds sufficient indications to judge whether the eye is already stabilised or nearly so, or is going to be an extreme variant.

In my opinion, with this judgment, and the reassurance of parents (and, of course, prescribing spectacles), the duty of the eye surgeon is fully completed; and should the case ever become an extreme variant, for the present we cannot do any more. The inherited duration of the stabilisation cannot be stopped by any means, least of all by restriction of near work. It is true the question of the origin of refraction and of the causes of myopia are far from being cleared up. Steiger's doctrine, as well as my

statements on stabilisation, are, in their uncertainty and unproved state, very open to attack. But still more so is the case with the opposite doctrine of the "school myopia." It is not only unproved, but one can bring weighty facts against it; as an example I refer to research on twins. There is, therefore, much to be said for the view that consequences of practical importance should not follow the doctrine of the school myopia, and restrictions in the near work of the young myopic patient should not be made.

Summary

Steiger's doctrine of the origin of refraction is enlarged with the conception of stabilisation.

Stabilisation is the cessation of the growing of the eye; its term varies with individuals. It plays a rôle equal to the combination and variation of the optical elements in the formation of the definitive refraction, and it is equally an inherited quality. The cause of the higher degrees of myopia is interpreted as retarded stabilisation.

The two weak points of Steiger's doctrine, the excess and the asymmetry of the variation-curve, are explained by the biological peculiarities of stabilisation.

A detailed knowledge of stabilisation will enable us to foretell the future of an early myopia.

Near work has nothing to do with the development of individual myopia, therefore restrictions in reading and studying by the young myopic patient should be put aside.

PIGMENT-ANOMALOSCOPY: A NEW PROCEDURE FOR TESTING THE COLOUR-SENSE*

BY

Professor A. KETTESY

DEBRECEN, HUNGARY

AS is known there has been up to now one method that enabled us not only to test but to measure the colour-sense in some degree: the mixing of spectral lights, as realised practically in Nagel's anomaloscope. Theory and knowledge of colour-sense and colour-blindness have been exclusively based on results obtained in this way.

The colours we see and discriminate throughout life differ from spectral lights; in opposition to the latter we call these "pigment-colours."

The important physical difference between the two forms is

* Received for publication November, 1947.