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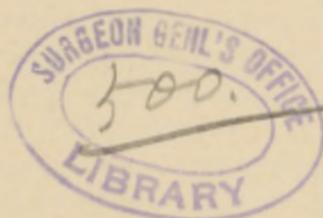
BY ✓

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THE PROXIMATE CAUSE OF
THE TRANSIENT FORM OF MYOPIA
ASSOCIATED WITH IRITIS.

WITH REMARKS ON OTHER FORMS OF SYMPTOMATIC AMETROPIA.*

BY A. SCHAPRINGER, M. D.

THE clinical fact that iritis in a considerable proportion of cases is apt to occasion a transitory myopia of low degree—about 1.5 D. to 2.0 D.—was first put on record by Dr. John Green, of St. Louis, the well-known authority on questions connected with refraction. Since the time that he read his paper on this subject before the American Ophthalmological Society in 1887 his discovery has been verified by a large number of other observers.

As to the proximate cause of the increase of refraction as a symptom of iritis Dr. Green did not offer any speculations.

In a paper on Symptomatic Myopia, read before the American Ophthalmological Society in 1888, Dr. W. F. Mittendorf advanced the theory that this transient increase

* Read before the Section in Ophthalmology and Otology of the New York Academy of Medicine, October 16, 1893.

of refraction was the result of an infiltration of the anterior portion of the vitreous body, increasing the bulk of this organ to such a degree as to push the lens system forward and with it the posterior principal focus of the eye. This theory I do not feel inclined to adopt, because as long as the tensile strength of the suspensory ligament of the lens remains unimpaired, an increase of pressure in the vitreous chamber would have to be very great indeed to displace the lens forward. The globe would feel very hard on palpation, other symptoms consequent upon increase of tension would be present, and there would be hardly any difference in the clinical pictures of iritis and acute glaucoma.

According to another theory, the increased refraction is said to be due to an augmentation in the corneal curvature produced by the increased pressure of the lids in consequence of the great photophobia. The negative results of ophthalmometric measurements completely disprove this theory.

Still another hypothesis alleges a spasmodic condition of the ciliary muscle as the cause of the symptom in question. This hypothetical cause forming the base of certain therapeutical recommendations lately made by Dr. Charles A. Oliver, of Philadelphia,* the question of the correctness of this hypothesis must henceforth be regarded as not merely of theoretical but also of practical interest.

Dr. Oliver insists that the refraction of the eye should be carefully and repeatedly tested in the course of an attack of iritis, and that instillation of strong mydriatic solutions should not be discontinued as long as the increased refraction persists, even after the pupil has been fully dilated. The presence of myopia proves the presence

* La correction exacte des vices de réfraction dans l'iritis plastique. *Annales d'oculistique*, Jan., 1893.

of spasm in the muscle of accommodation, and this being a pathological condition, it needs to be combated by the appropriate means at our disposal.

Now Dr. Green has already pointed out that the fact that the increase of refraction persists for an indefinite time after the pupil has fully yielded to the action of atropine was a proof militating against the assumption that the transient myopia was due to spasm of accommodation. Besides this argument advanced by Dr. Green, the supposition that inflammation will cause a spasmodic contraction of the ciliary muscle is contrary to all physiological experience in regard to analogous conditions elsewhere in the human organism. To bring forward only one example, let me refer to the behavior of the muscular coat of the intestine in peritonitis, where it is anything but contracted. On the contrary, it is completely paralyzed, and by its paralysis causes two of the main symptoms of the disease, tympanites and constipation. The contraction of the pupil in iritis is not due to spasm of the sphincter iridis muscle, but is only dependent on the swelling of the tissue of the iris in consequence of hyperæmia and infiltration. It is also a very significant fact in connection with the question here discussed that in the insidious forms of sympathetic iridocyclitis the first sign of impending trouble is the *diminution* of the power of accommodation, causing recession of the near point.

Having dismissed all the theories hitherto enumerated as unsatisfactory, it behooves me to substitute one against which no valid objections could be raised. I think that the transient myopia of iritis can be best explained by a temporary increase of the refractive index of the aqueous humor. That the composition of the contents of the anterior chamber in iritis differs from that in the normal state is not a hypothesis, but a fact. The formed particles sus-

pended in the aqueous humor, rendering its aspect turbid, will, of course, have no influence upon the refraction of the eye. We are here concerned only about the transparent part of the liquid. Judging from the deposits, among other substances, of fibrin upon the walls of the anterior chamber, nothing would seem more plausible than to assume that in iritis the aqueous humor contains this substance in more than the normal quantity. If this is the case, it follows from physical laws that its index of refraction must be increased. A higher index of refraction of the aqueous will produce myopia, because the beams of light which receive an inclination toward the optical axis, when refracted at the anterior corneal surface, will receive an additional inclination toward this axis at the posterior corneal surface. In the normal state there is no refraction at this surface, because then there is no difference between the refractive indices of the cornea and of the aqueous humor. The posterior wall of the cornea in iritis represents a convex refracting surface with a medium of higher refrangibility behind it than before. Consequently the optical conditions present at this surface will be the same in kind, though not in degree, as those present at the anterior surface.

If the refractive index of the aqueous humor of an emmetropic eye were lessened instead of increased, the inclination of the rays of light toward the optical axis would be diminished and the eye therefore rendered hypermetropic.

The question now presents itself, How much must the index of refraction of the aqueous be increased in order to produce a given degree of myopia?

In order to solve this problem, we have to take into consideration the factors coming into play at the refraction by the posterior corneal surface. The relation of the constants is expressed by the well known formula—

$$F_2 = \frac{n_2 r}{n_2 - n_1}$$

We wish to calculate the value of n_2 , which represents the index of refraction of the aqueous humor :

$$n_2 = \frac{F_2 n_1}{F_2 - r}$$

F_2 represents the second principal focal distance of our refractive system, consisting of corneal substance, posterior corneal surface, and iritic aqueous, or, in other words, the collective power of this system. This collective power is measured by the concave glass placed before the eye, neutralizing the collective power by its dispersive power. In substituting the reciprocal value of this glass, we have to deduct the distance of the correcting glass from the posterior corneal surface.

n_1 stands for the refractive index of the corneal substance, which is 1.3365 according to Helmholtz.

r represents the radius of curvature of the posterior wall of the cornea, which, following Tscherning, we will put down as six millimetres.

Carrying out the calculation for different degrees of myopia as met with in iritis, we find the following values of n_2 :

Degree of myopia.	Index of refraction of aqueous.
1.0 D	1.345
1.5 D	1.349
2.0 D	1.353

The increase from the normal—which is 1.336—not being considerable, the figures at least do not speak against the plausibility of our hypothesis.

If we accept this hypothesis as the true explanation of the myopia occasioned by iritis, we shall refrain from continuing the instillations of atropine after the pupil has fully yielded to its action and the congestion has

subsided, although the myopia may still be present. Our theory furnishes a contraindication against the persistent employment of atropine under these circumstances, inasmuch as a dilated pupil means an obstructed iris angle, and this means interference with the drainage of the anterior chamber. We shall not feel inclined to do anything to keep the morbid aqueous humor longer in the anterior chamber than necessary. Our hypothesis furthermore does not impose upon us the obligation of carefully and repeatedly testing the refraction of eyes affected with iritis, of which release we shall avail ourselves with avidity and earn the thanks of our patients.

If the myopia persists for some time after the subsidence of the inflammatory symptoms and we should feel called upon to hasten the work of Nature, massage of the eyeball would offer itself as a local therapeutic measure in consonance with our theory.

The two recognized varieties of myopia in general are axial myopia, which is the most common form, and myopia of curvature. If our theory as to the causation of the transient nearsightedness occasioned by iritis is verified, we shall have "index myopia" as a third type of this error of refraction.

To test the merits of the theory here propounded, very accurate measurements with instruments of precision rarely met with in the possession of practitioners will be necessary. Those who have a Helmholtz ophthalmometer, or, what seems to be a still more suitable instrument for the purpose, a Tscherning ophthalmophakometer at their disposal, will deserve the thanks of the profession by taking up this problem. The shortest way to determine the refractive index of iritic aqueous would be to tap the anterior chamber and test the liquid obtained by means of an Abbé refractometer; but the clinical indication for paracentesis

of the cornea hardly ever arises in the course of plastic iritis.

Since we are discussing the subject of index ametropia, the question naturally suggests itself, What would become of an emmetropic eye if the index of refraction of the vitreous body be increased?

The answer to this question will be found in the following consideration:

The beams of light, striking the concave anterior surface of the vitreous, are deflected toward the optical axis to join it at the place of the retina. The strength of the deflection depends upon the difference of the refractive indices of the crystalline body and the vitreous. The index of the vitreous being the lesser quantity, we will, by increasing it, lessen the difference between it and the crystalline index, or, in other words, lessen the inclination of the rays of light toward the optical axis. This means that the emmetropic eye is being rendered hypermetropic.

A change in the refractive index of the vitreous will therefore have the opposite effect from that which is produced by the same kind of change in the aqueous.

In the intervals between the prodromic attacks preceding an outbreak of inflammatory glaucoma the patients usually complain of being compelled to use increasingly strong convex glasses in rapid succession for reading purposes. This is usually explained as the result of a rapid diminution in the power of accommodation, but it seems to me not improbable that this symptom may have something to do with an increase in the refractive index of the vitreous body. Such an increase, as said before, will render an emmetropic eye hypermetropic, and a hypermetropic eye still more hypermetropic. I have met nowhere with the distinct statement from any observer that, while the near

point of the glaucomatous eye thus rapidly receded, the far point was found to remain stationary.

The myopia caused by diabetes is a form of myopia to which Hirschberg has called attention and which is probably also a type of index ametropia. I have myself recently met with two cases of this kind in succession—one of them in a lady sixty-one years old, referred to me by the kindness of Dr. S. J. Meltzer, of this city. The acquired myopia in both cases was of considerable degree, being more than 6 D. An increased curvature of the front surface of the lens I can hardly deem accountable for this, since the depth of the anterior chamber, at least as far as could be made out without the use of the instruments of precision mentioned before, seemed perfectly normal in both cases. A decrease in the refractive index of the cortical substance of the lens will, I think, best account for this type of myopia, if the myopia is to be accounted for by a change in this organ. Such a decrease will increase the refractive power of the lens system as a whole and thereby bring the posterior principal focus of the eye in front of the retina. It may sound paradoxical at first that the collecting power of a biconvex lens like the crystalline body should be increased by a decrease in the refractive index of one of its constituent parts. But it must be remembered that the cortical substance represents a system of concave or dispersive lenses which neutralize the overstrong collective power of the globular nucleus. By lowering the refractive index of the cortex we lessen its dispersive power, and the result will be a preponderance of the collecting power of the nucleus; the principal focus of the eye is thereby brought forward and the eye made myopic.

Since writing the foregoing my attention has been drawn to a paper, *On the Presence of Sugar within the Eye*

in Experimental Diabetes,* by the brothers Cavazzani, an abstract of which has recently appeared in the *Centralblatt für prakt. Augenheilkunde* (supplement for 1892, p. 496). Having made experimental researches on the functions of the pancreas, these authors report the results of the chemical analysis of the refracting media of two dogs which were afflicted with a permanent form of diabetes in consequence of the extirpation of the pancreas. The animals were killed in due season and their eyeballs enucleated. In the first dog the aqueous humor contained 0.386 per cent. of sugar. In the peripheric layers of the lens very little of this substance was found, *and none at all in the nuclear portion of the lens and the vitreous body*. In the second dog sugar in the proportion of somewhat less than 0.05 per cent. was found in the aqueous humor and none at all in the other refracting media.† The lenses were completely transparent and anatomically normal in every respect. The fact that the lens, at least of one of the animals, contained sugar, speaks against the theory enunciated by Deutschmann,‡ which says that no sugar can diffuse into the substance of the lens as long as the epithelial layer of the lens capsule remains intact.

In the investigations of the Italian experimenters, at least as far as they are reported in the *Centralblatt*, no attention was paid to the refraction of the eyes during life or to the refractive index of the media subjected to chemical examination. I hope that in future investigations of the same kind these matters will not be lost sight of. The results of the chemical analysis make it probable that the

* This is a portion of a larger work, *Le funzioni del pancreas ed i loro rapporti colla patogenesi del diabete*. Venezia, 1892.

† From the marked difference in the chemical behavior of the aqueous and the vitreous the authors draw the conclusion that the sources of nutrition of these two substances must be entirely distinct.

‡ V. Graefe's *Arch. f. Ophthalm.*, 1887, p. 229.

aqueous humor at all events plays an important part in the production of diabetic myopia. Future investigations will have to show whether the peripheric layers of the lens are also concerned in this change of refraction and how far. It is very characteristic that the vitreous body of the diabetic dogs was found absolutely free from sugar. This is in remarkable harmony with what was pointed out before on theoretical grounds—viz., that the degree of myopia produced by an increase of the refractive index of the aqueous humor will not become further increased by an increase of the refractive index of the vitreous body, but, on the contrary, an increase of the refractive index of the vitreous would neutralize, more or less, any myopia caused by a change of index of the aqueous humor.

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EDITED BY

FRANK P. FOSTER, M.D.

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