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HYPERMETROPIA AND HETEROTROPIA.

BY

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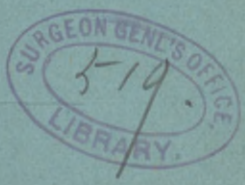

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HYPERMETROPIA AND HETEROTROPIA.[1]

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THE subject of this communication has been in part presented to the profession in two papers, "The Treatment of Internal Squint," *Annals of Ophthalmology*, January, 1894, and "A Cause of Failure in the Surgical Treatment of Internal Squint," read by title before the Pennsylvania State Society, May 16, 1894. I desire now to bring it, modified and strengthened by added experience, before this Society, with the hope that a discussion may be elicited by which its value as a statement of actual existing conditions and as an explanation of those conditions may be determined. I submit the following two propositions:

1. In constant, or monocular squint, due to hypermetropia, when one and the same eye is always used in fixation, the second eye is invariably turned upward as well as inward.

2. In concomitant, or alternating squint, due to hypermetropia, when either eye is used indifferently for fixation, the squinting eye is invariably turned upward as well as inward, and, with alternation of

[1] Read before the American Ophthalmological Society at Washington, May, 1894.

fixation, the upper deviation is transferred simultaneously with the inward deviation.

Both propositions may be included in one statement; namely, that functional esotropia, or purely lateral deviation of the visual axes, is never found alone, but is always associated with hypertropia.

I make these assertions with a certain amount of diffidence and hesitation, for, while I have been unable to find their equivalents in my reading, it seems improbable that these facts have escaped the observation of the many careful investigators who, following Donders' lead, have so thoroughly studied the etiology of functional strabismus, and I fear that my own researches into literature have been too limited.

I have found allusions to an upward squint complicating convergence, and in Stevens' studies, published in the *Archives*, attention has been called to the same condition; but in no text-book or monograph with which I am familiar is it positively stated that the two must be invariably associated, nor has any author, so far as I know, who has mentioned the upward squint as complicating the internal, suggested an explanation.

If we accept Donders' theory of hypermetropic squint, which is, I believe, universally admitted as correct, and we pursue his line of reasoning to its logical and physiologic conclusion, we shall have a satisfactory explanation not only for the existence of the hypertropia, but also a sufficiently convincing reason for the clinical fact that esotropia cannot be caused by hypermetropia unless hypermetropia also cotemporaneously develops hypertropia.

Pathologic convergence is induced through excessive action of the ciliary muscles, by reason of the anatomic and physiologic relation of the nuclei of the ciliary and interni, when the bounds of relative convergence and accommodation have been overstepped. But the nuclei of other branches of the third nerve participate with the convergent nuclei in this excitation, as is shown in the pupillary contraction.

Now, the elevators of the cornea are the superior rectus and the inferior oblique, and both are supplied with motor force from the third nerve. The depressors of the cornea are the inferior rectus and the superior oblique, only one of which receives impulse from the third. Therefore, excessive stimulation, or hypermetropic stimulation, if I may be allowed to use the expression, must induce not only overaction of the interni, but also of all the muscles supplied by the motor oculi. As the external recti cannot maintain lateral equilibrium, the elevators are for the same reason only in part antagonized by the depressors, and the cornea is rolled inward and upward. Hence, when the visual axis of one eye crosses that of the other within infinity, it must be on a higher plane.

These propositions have been verified by clinical observation. In every case, without exception, that has come under my care during the past few months, in which I have been able to induce the patient to become cognizant of the image which falls on the retina of the squinting eye, perhaps twenty-five or thirty in number, he has described its position as homonymous and on a lower level than that of the fixing eye.

No insurmountable difficulty has been encountered in discovering the false image. It has demanded, however, patience and perseverance, and the use of glasses of different colors—the true image blurred by a dark blue, and the false changed by a bright-red glass. It has occasionally been necessary in the early efforts to bring the false image nearer to the true by prisms, bases out, or to place the light nearer to the patient than twenty feet.

The final and determining tests have always been made without artificial deflection of the false image, so that no doubt as to its horizontal plane could arise from a possible oblique turning of the prism thus used. Rarely, cases may be found in which the projection is false, when, for instance, in convergence the images are heteronymous and no deduction can be drawn from the patient's answers. Here the observer must rely upon objective examination of the deviation of the squinting eye, and close inspection will reveal both esotropia and hypertropia.

Further, I believe the application of the same principle can be extended to an elucidation of the etiology of hyperphoria as well as hypertropia, and help us to a clearer comprehension than we now have of the origin of this perplexing condition. The bases of argument must again rest upon Donders' theory of hypermetropic squint. If hypermetropia in its higher grades can produce a positive deviation of the visual axes, it follows that, in the lower grades, it is responsible for tendencies to deviation, or "phorias," and, logically, the tendency cannot be limited to esophoria, but must include hyperphoria.

The influence of the ciliary muscle in overcoming the low grades of hypermetropia and astigmatism in the young is not sufficiently great to destroy the extra-ocular balance, and the visual axes are maintained in their binocular relation at a cost of nerve-energy that, in patients of neurotic disposition, is followed or accompanied by severe reflex disturbances. But it manifestly produces a corresponding overaction of the other muscles under the control of the third nerve, or a tendency to deviate from equilibrium. In persons of middle age, when presbyopia is commencing and the range of relative accommodation and convergence is limited by loss of accommodation through stiffening of the lens, the effort at contraction of the ciliary muscle, or the nerve-power, is, however, unchanged from that of earlier life, and thus the other branches of the third are stimulated in their accustomed degree. Since, as has been shown, this action includes other than the internal muscles, we have every reason to believe that the asthenopia is muscular and not accommodative, because better vision or less error of refraction in one eye will determine esohyperphoria of the other.

This hypothesis, more rational than any that has been proposed in explanation of the muscular anomalies so frequently associated with hypermetropia, receives confirmation in the effect of paralysis of accommodation and the optic correction of the error of refraction, both in heterotropia and heterophoria. In the former, the cure of the disjointed muscular action by these means alone has been perhaps the most persuasive argument to the universal

acceptance of the truth of Donders' theory; and in the latter, correction of hypermetropia and astigmatism has been amply demonstrated to be quite sufficient treatment in the great majority of cases not only for the accommodative, but for the muscular asthenopia, as is so frequently demonstrated by the gradual, and in some cases immediate, disappearance of the symptoms, and by the restoration of equilibrium according to the tests commonly employed.

Before advising operation in hyperphoria, we must therefore be convinced that it is a permanent and real condition, independent of and uninfluenced by the use of the accommodation, and cannot be transferred from one eye to the other. We may be led into error of diagnosis, even in hyperphoria, by the fact that the hypermetropic patient under the test by artificial horizontal diplopia involuntarily or unconsciously fixes at the moment with one eye, and having determined in our own mind that a right or left hyperphoria is present, all subsequent tests seem to confirm the impression. A constant hyperphoria, just as a constant hypertropia, is plainly consistent with the theory advanced, and it is only in such cases that operation on the superior rectus, either alone or in addition to operation on one or both internal, is admissible.

Recognition of the truth of the two propositions presented in the first part of this paper, and acceptance of the hypothesis offered in explanation, will necessarily modify the methods commonly employed in the surgical treatment of latent and manifest strabismus due to hypermetropia.

1. In so-called constant monocular squint, binocular fixation can be secured and maintained, unless prevented by amblyopia, only by full correction of the optic defect, tenotomy of the interni and tenotomy of the superior rectus of the squinting eye, and possibly of the inferior rectus of the fixing eye.

2. In alternating squint, vertical equilibrium will be restored by correction of the refraction and tenotomy of the interni, each muscle divided to the same extent. Direct surgical treatment of the hypertropia is not indicated under any circumstances.

The endeavor to secure a cosmetic success by confining the tenotomies to the squinting eye only, such as extensive section of the internus, conjoined with advancement of the externus, as recommended by most authorities, is greatly to be deplored.

3. In hyperphoria, operations to secure vertical equilibrium may be made only when it is clearly proved that relief from asthenopic symptoms cannot be secured by less radical measures. And the influence of hypermetropia in causing this condition, and the possibility of alternation of fixation transferring it from one to the other eye, must be borne in mind.

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