operation with possibly few exceptions is an absolutely harmless and safe one. It is doubtless true that in some of the exceptional serious accidents of vaccination that neither the operator nor the virus is at fault, but that the damaging infection takes places later as a result of carelessness, negligence and uncleanliness on the part of those vaccinated. Moreover, in many instances of eruptions occurring during or immediately subsequent to vaccination it is more than probable that they are purely coincidental, and in no way connected with or due to this procedure, either primarily or through accident secondarily. The layman, and flagrantly the anti-vaccinationists, and sometimes the physician, too, are too prone to consider all eruptions at such times as effects. In short, it should be clearly understood that eruptions occurring at such period are not necessarily vaccinal, although it is true that many of them are. It is clearly unwise and unscientific, however, even for the laudable purpose of quieting the fears of the public or for the purpose of withholding ammunition from the anti-vaccinationists, to deny that vaccination is ever responsible for any eruptive or systemic disturbance. Nothing is to be gained by smothering, concealing or perverting facts. The candid recognition of exceptional complications should lead to a study of their causes, and finally, in all probability, to their prevention or at least to their diminution. In those eruptions commonly observed—urticaria and the various types of erythema multiforme—I believe it is probable that the causative factors lie with the virus itself, either to its preparation or preservation, and possibly due to some admixture or changes in the lymph constituents, and not necessarily of extraneous origin. This seems borne out by the facts that the serums used in the treatment of certain maladies will likewise often produce similar rashes. It is well known that the serum from the horse used in the manufacture of antitoxin will provoke these eruptions, but—and this is the point which it seems to me has a pertinent bearing—according to Berg,[32] much less frequently when the serum is thoroughly and carefully filtered. It appears, moreover, extremely probable that the pemphigoid eruptions are likewise due to some toxin or microbic element derived directly from the animal from which the virus is obtained. It is in this direction, then, I believe, that the etiologic causes in the production of some of these various rashes must be sought. But even taking into consideration the exceptional accidental more serious eruptions, their occurrence weighs as nothing as compared to the benefit which vaccination has bestowed on mankind. But with proper care, however, on the part of the caretakers of the cattle, vigorous governmental inspection of the animals and extreme precaution in the collection and preservation of the vaccine, there would be but little to fear. Add to this caution and cleanliness on the part of physician and patient, both before, at and after the time of the operation, till complete healing has taken place, and the occurrence of serious accidents would be practically placed very nearly beyond the bounds of possibility.

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Original Articles.

THE DECENTERING OF LENSES FOR NEAR WORK.*

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The ideal position of lenses, when there is perfect muscle adjustment of the eyes, is such that the visual axes may cut their optical centers, and that the planes of the lenses may be parallel with the equatorial planes of the eyes. When the visual axes cut the optical centers of the lenses there can be no prismatic effect; and when the plane of the lens is parallel with the equatorial plane of the eye there can be no cylindrical effect. A want of parallelism between the plane of the lens and the equatorial plane of the eye means that there will be a cylindrical effect, for, as is well known, the strength of the lens, at right angles to the axis of tilting, is increased, while along the axis of tilting its power is unchanged. Tilting a lens 45 degrees practically doubles its refractive power for the rays that are in a plane at right angles to the axis around which it has been rotated. These laws apply with equal force to lenses that are worn for distant seeing and those that are used in near work. Infringement of these laws constitutes the chief objection to bifocal lenses. If it can be estimated just how much the visual axes must be depressed below the extended horizontal plane of the head, in reading or doing other near work, through the same number of degrees the upper border of the lenses to be used should be tilted forward to prevent cylindrical effect vertically; and each lens should have its nasal border tilted backward to the extent of half the angle of convergence, to prevent cylindrical effect horizontally. This double tilting would make the plane of each lens parallel with the equator of the eye before which it is placed.

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But the subject assigned me by our chairman is "The Decentering of Lenses for Near Work," and to this subject the remainder of this paper will be confined.

1. If there is orthophoria, presbyopic lenses should be properly centered, that is, they should be so placed that each visual axis will cut the optical center of its lens, when a point of fixation is in the extended median plane of the head. On changing the point of view in any direction, without moving the head, the prismatic effect would be the same in kind for each eye, and if the lenses are of equal strength, the degree of prismatic effect would be the same for each eye. The lenses for such eyes should never be so placed that both visual axes would cut them on either the nasal or temporal sides of the optical centers. With safety, and in some cases with positive helpfulness, the lenses may be so placed that the visual axes would cut them directly above the optical centers. As can be readily seen, this would take some work off the subvertor muscles, thus lessening the demand on the second and sixth conjugate brain centers. But orthoptic eyes will not take kindly to presbyopic lenses placed so high that the visual axes would cut them below the optical centers, for this would create an abnormally large demand on the two centers mentioned.

2. If there is uncomplicated esophoria, both presbyopic lenses should be decentered directly out, and to an equal extent, so that the two visual axes may cut the lenses to the nasal side of their optical centers, thus favoring the weak externi. This can be accomplished equally as well by making the frames wider than called for by the pupillary measurement. If the interni are properly attached, the compensating esotropia will be attended by relief, but if attached too low the compensating esotropia would develop a plus cyclophoria that might bring great discomfort. To decenter lenses in, or to have the frames narrower than called for by the pupillary measurements, for such eyes, would render the lenses more or less unbearable.

3. In esophoria complicated only by hyperphoria of one eye and cataphoria of the other, the decentering of presbyopic lenses should be confined to the lens for the hyperphoric eye, and should be down and out, so as to develop a compensating eso-hypertropia of this eye. To decenter the lens out-and-up for the cataphoric eye would result in developing a plus cyclophoria, to correct which the superior oblique would be forced into a state of abnormal tension. Slight abnormal tension is well borne by the inferior oblique, but not by the superior oblique.

4. In esophoria, complicated by hyperphoria of one eye and cataphoria of the other, with plus cyclophoria, the decentering of presbyopic lenses should be confined strictly to the lens for the hyperphoric eye, and should be down-and-out, so as to develop a compensating eso-hypertropia. The rotation in-and-up, made necessary by the prism displacement, generates a minus cyclophoria which, in such a case, would neutralize the existing plus cyclophoria, thus enabling the superior oblique to easily parallel the vertical axis of the eye with the median plane of the head. In such a case the evil-effect of decentering the lens out and up, for the cataphoric eye, comes from the compensating eso-catatropia, and is due to the plus cyclophoria that is thus generated. There being already a plus cyclophoria which the superior oblique must correct by being kept in a state of abnormal tension, the added artificial cyclophoria can do nothing but augment the discomfort of the patient.

5. In simple exophoria each presbyopic lens should be decentered directly in and to an equal extent, or what would be the same in effect, the frames should be made narrower than would be indicated by the pupillary measurement. This would develop a compensating exotropia, and the lenses would be well borne if the externi have ideal insertions or if their insertions are lower than normal; but if their insertions are higher than normal the lenses thus decentered would not be well borne for the reason that the compensating exotrophia would develop a plus cyclophoria. Presbyopic lenses, in frames that are wider than indicated by the pupillary measurement, can not be borne by an exophoric.

6. In exophoria complicated by hyperphoria of one eye and cataphoria of the other, the decentering of presbyopic lenses should be confined to the one for the cataphoric eye, and should be in-and-up. This would develop a compensating exo-catatropia; that is, the eye would be rotated out-and-down. Every such rotation of an eye develops a minus cyclophoria which the inferior oblique can correct easily. Decentering the lens in-and-down, for the hyperphoric eye, would cause a compensating rotation out-and-up, which would develop a plus cyclophoria, the correction of which would not be easily borne by the superior oblique.

7. In exophoria complicated by hyperphoria of one eye and cataphoria of the other, with plus cyclophoria, the decentering of the presbyopic lenses should be confined strictly to the one for the cataphoric eye, and should be in-and-up. The compensating exo-catatropia, that is, the rotation out-and-down, would develop a minus cyclophoria which would more or less completely neutralize the existing plus cyclophoria. To decenter the lens for the hyperphoric eye in-and-down would cause a compensating exo-hypertropia, that is, a rotation out-and-up. This would develop artificially a plus cyclophoria which, grafted onto the plus cyclophoria already existing, would only add to the discomfort of the sufferer.

8. In hyperphoria of one eye and cataphoria of the other, with or without plus cyclophoria, the decentering of presbyopic lenses should be confined to the one for the hyperphoric eye, and should be directly down. There would be caused a compensating hypertropia. This would develop a minus cyclophoria which the inferior oblique would counteract readily. To decenter the lens directly up for the cataphoric eye would cause a compensating catatropia which would develop a plus cyclophoria not easily correctible by the superior oblique. The trouble with such a lens would be emphasized if the artificial plus cyclophoria should be grafted onto an existing plus cyclophoria.

9. In double hyperphoria uncomplicated, both presbyopic lenses should be decentered directly down, and to an equal extent; or, what would be the same in effect, the nose-bridge should be made deep enough to allow the visual axes to cut the lenses above their optical centers.

10. In double cataphoria uncomplicated, if any decentering should be done at all it should be directly up.

11. If there is plus cyclophoria only, in a presbyopic case, both correcting lenses should be decentered down.

The decentering of lenses, or prisms in positions of rest, is only one way of dealing with muscle errors. I believe the rules given above are scientific, and that, if followed, comfort from the lenses will be experienced, provided the prismatic effect is not too much. The maximum vertical prismatic effect should be placed at one degree, certainly not more than two degrees, and the maximum lateral prismatic effect should be placed at two degrees, certainly not more than four degrees. In the

greater number of cases the prismatic effect should correct about one-half the manifest error, but in some cases a full correction, especially of a small vertical error, may be given.

Prisms and decentered lenses interfere with some of the visual judgments and therefore are objectionable. For this reason it is better to cure muscle errors of low degree by proper prismatic exercise. Muscle errors of high degree should be treated surgically.

THE GENESIS AND TREATMENT OF THE MYOPIC EYE.*

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There is no more interesting or important chapter in ophthalmology than that which treats of the etiology, nature and treatment of the myopic eye. The widely differing views which have been expressed regarding each of these points seem to justify a renewed presentation of the subject.

HISTORY.

The observations made in the early part of the century by James Ware regarding the relatively greater prevalence of myopia among the educated portions of the community were verified by the later examinations in the schools by many observers, who demonstrated its steadily increasing percentage as the children advanced in age and school progress. Since this was associated with a nearly equal fall in the percentage of hypermetropic eyes it required but a step to the seemingly logical conclusion that the small hypermetropic eyeball of childhood increased in size with the physiologic growth of the child into emmetropia and from this into the large myopic eyeball; in a word, that the myopic eye was a physiologic evolution. Closely allied to this was the proposition that, since only those eyes which are subjected to the demands of civilized life were observed to undergo this change of refraction, therefore it was the eye of civilization, a species of social evolution, an adaptation to the act of work.

More careful observations of the conditions, however, revealed the fact that these conclusions were untenable, since they must be maintained in face of well-known and characteristic pathologic conditions associated with myopia, which imperiled the integrity of the eye, e. g., posterior staphylomata, choroidal atrophies at the temporal border of the optic nerve and in the region of the fovea, a fluid, i. e., a degenerate vitreous with floating opacities and frequent detachment of the retina, all of which sustained with myopia an intimate and undoubted relation of cause and effect. Increasing experience and painstaking observation have served, after nearly half a century, to maintain and enforce the truth of the early teaching of Donders, to the effect that the myopic eye is a diseased eye.

Accepting the proposition as stated, it is obvious, therefore, that myopia can not be regarded as either a physiologic or sociologic evolution, but is to be viewed as one of the many baneful results of civilized life over the physical well-being of the race. The problem presented for solution is its prevention.

GENESIS OF THE MYOPIC EYE.

The observations of Cohn and others showing the rapidly increasing percentage of the disease during the educational process led thoughtful observers to inquire into its cause. Since it was evidently in some way closely related to the work in the schools it was ascribed to the bad hygienic environments of the school room, and an era of improvement was inaugurated in the lighting of the school houses, the arrangement and construction of desks and seats, the paper and type used in school books, etc. A few years later a re-examination of the same children under the improved conditions of work, much to the disappointment of these industrious and careful observers, discovered no notable diminution in either the percentage of increase or in the arrest of the progress of the myopia in those already myopic at the first examination. It was obvious, therefore, that some hitherto unnoticed factor was responsible for the genesis of the myopic eye.

The earliest suggestion with which I am familiar of the possible relation between astigmatism and myopia was made by Dr. Green[1] of St. Louis in 1867, and again[2] in a paper in 1871. He closes the latter paper with the statement that so far as he knew statistics of large numbers of eyes to establish the frequency of astigmatism were wanting. My own statistics collected in the Philadelphia schools, 1878 to 1881, were, so far as I know, the first to establish the truth of Dr. Green's suggestion, of which I was at the time entirely ignorant. In 1880 I published[3] a group of cases which, while under observation, had passed from hypermetropic into myopic astigmatism without at any time becoming emmetropic. That is to say, they passed through the turnstyle of astigmatism from hypermetropic to myopic refraction. They were all asthenopic, and the increase of refraction was associated with disease of the intra-ocular tunics. This publication was followed in 1881 by the report of the school statistics,[4] which revealed a group of interesting and important facts bearing on the genesis of the myopic eye.

First. The emmetropic eye not only remained in a nearly uniform percentage through all ages of school life, but enjoyed the highest acuity of vision and was relatively free from pain and disease.

Second. The eyes with a simple hypermetropia stood next to emmetropia in all these respects.

Third. The eyes with hypermetropic refraction, especially where astigmatism was present, manifested symptoms of asthenopia, lowered acuity of vision and pathologic states of the fundus; notably irritative and absorption changes throughout the fundus, granular changes at the fovea and crescents of choroiditis at the temporal margin of the optic disc in all stages of development from a faintly outlined crescent to far advanced atrophies which had before been regarded as physiologic anomalies when seen in hypermetropic eyes, but as pathologic if associated with myopic refraction.

Fourth. It was shown, as had been demonstrated by all observers, that not only did the myopic eye increase in percentage during school life, but that there was a closely corresponding diminution in the percentage of hypermetropia. This group of facts seemed to afford a sufficient demonstration for the conclusion that the myopic eye, with its characteristic pathologic changes in the fundus, was but a later stage of the conditions witnesses in the eyes with hypermetropic astigmatism, hence that the former was recruited from the latter.

Fifth. In the very large percentage of congenitally

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