ARE PROGRESSIVE MYOPIA AND CONUS (POSTERIOR STAPHYLOMA) DUE TO HEREDITARY PREDISPOSITION, OR CAN THEY BE INDUCED BY DEFECT OF REFRACTION ACTING THROUGH THE INFLUENCE OF THE CILIARY MUSCLE?

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AFTER a careful consideration of the above question, I have come to the conclusion: (1) That hereditary predisposition, though undoubtedly a potent cause, is not only not the sole cause, but not even the predominating cause, of progressive myopia; and (2) That the action of the ciliary muscle, taken by itself, exerts but little influence on the production of myopia, and still less on the formation of the cone.

The arguments on which these conclusions are based will be found in the following remarks:—

The belief that "like begets like" has been formulated in the speech and crystallized in the proverbs of every people since the world began, and although this uniformity of opinion exists as to hereditary predisposition, and its influence on the organism in general, a great deal of doubt has been expressed as to the degree of effect which it produces on particular and individual organs, and especially on those which fulfil the functions of special sense. Nor can it be doubted that grave objections have from time to time been raised, and many weighty arguments advanced, which would go to show that the effect of direct transmission could not be so readily detected, nor so clearly demonstrated, in regard to the special senses as to the organism as a whole. Still, in later years, as proofs have gradually multiplied and apparent discrepancies have been reconciled, the opinion has gradually become more general that, beneath a mass of apparent contradictions and almost endless exceptions, there lies a general tendency of inheritance which authorizes us in assuming that even with our special faculties there is a uniform, though perhaps, from want of knowledge, a rather flexible, law, which controls, to a degree at least, their form and character.

In none of these is it claimed that this law of transmission is made more manifest than in sight, the noblest and most intellectual of all our faculties. Thus it has been assumed by the preponderance of authorities, that the specific type of the organ of vision has its varieties of form and perception, every one of which may owe its origin to the force of heredity. Thus it is held that color blindness, strabismus, hypermetropia, and myopia, are transmissible by generation, so that according to Lucas(1)[1] there are families among which many of their members owe

[1] The numbers attached to the names of the authors refer to the bibliography at the end of the paper.

to hereditary influences alone the conformation of their optical apparatus, and the fact of their range of vision being short or long. It is further claimed that the statistical results of Funari(2), supported by the statements of Piorry(3) and Portal(4), have shown that most myopes are the sons or grandsons of myopes. It is certainly not necessary for me to remind the members of this Section that views, similar to, if not identical with, those just quoted, as to the hereditary tendency of myopia, have held sway successively in England, France, Germany, and in this country, and indeed in every country. Nor would the time allotted to this paper allow me, even if your patience would, to cite the long list of distinguished names through whose authority this wide-spread opinion has had and still has its existence; still, for the sake of the arguments which are to follow, I should like to be allowed to refresh your minds in regard to the opinions of a very few of those who in our own time have done so much towards moulding the prevailing opinion as to the hereditary nature of myopia.

Thus Stellwag von Carion(5), in discussing the causes of myopia and the elongation of the eyeball, says "the predisposition to this is of course congenital, and is, as a rule, hereditary," and adds, in speaking of the staphyloma and its relation to the scleral protuberance, that the "exquisitely hereditary character of the affection can also be brought forward as a proof of this." Jaeger(6), while emphasizing the fact that myopia is not the "prerogative of industry," declares that "posterior staphyloma is hereditary to a predominating degree." And Donders(7), in speaking of the causes of myopia, affirms that "the predisposition is almost always congenital, and in that case it is, moreover, nearly always hereditary. Beer, Jüngken, Bohn, Von Hasner, and many others, have referred to its hereditary nature, and I believe even that from time immemorial the conviction thereof has been general among the people." Many more examples quite as explicit as these might be cited here, were it necessary, since, as you are well aware, every standard work on ophthalmology is replete with them.

It would be supposed, from the force of the expressions and the weight of authority from which they come, that these opinions in regard to the predominating influence of heredity in myopia would be based on exact and extensive statistical information which would embrace, certainly, as wide a scope as from grandfather to father and from father to son. Yet, as far as I am aware, no such statistics exist, at least of such a nature as to satisfy the exacting demands of modern science. It is very true that both Donders and Jaeger have—as indeed we all have—been struck with the frequency with which myopic parents bring to us their myopic children; but, as you are well aware, the mind, in these matters, is more prone to be struck with resemblance than with dissimilarity, and no account is taken or recorded anywhere, as far as I know, of how often the reverse holds good, that is, how often children with myopia are brought by parents who, on actual examination, are shown to have normal eyes, and whose ancestors on both sides, as far as known, were never near-sighted. Moreover, I found that among 715 well-educated and intelligent persons, whom I examined for this special purpose, the percentage of those who had normal eyes themselves, but whose parents were myopic, was nearly identical with the percentage of those who were not only myopic themselves, but who had myopic parents. That is to say, that the percentage of emmetropic children from myopic parents was as high as that of myopic children from myopic parents. No one more fully

recognizes or more freely admits than I the liability to error in statistics gathered thus as it were at second-hand. Nothing but the tabulated observations of three generations at least can be of much value as regards heredity, but as such do not exist, I offer the above for what they may be worth.

These observations would, however, seem to be in accordance with those of others. Thus Cohn(8), in his investigations as to the refraction of the eyes of school children, found that out of all the myopes there were only 2.7 per cent. whose father or mother were myopic. I found that only 6.11 per cent. had either father or mother near-sighted. With both parents myopic, the percentage falls with Cohn(9) to 1.04 per cent. and with me to 1.11 per cent. Erismann(10) found, however, from his statistics that 30.6 per cent. of the myopes had one or both parents myopic. Taking, then, Erismann's percentage, which is an enormous increase over Cohn's and mine, as a basis, we find, even then, that two-thirds, or seventy per cent., of the myopic children had parents who were not myopic. This certainly does not show, as far as statistics are concerned, a marked hereditary tendency, or warrant, it would appear, the expression of so decided an opinion as that which we have quoted from the leading authorities.

There are, however, in the absence of exact statistical knowledge, several factors of less importance, it is true, but still of sufficient force to aid us materially in the solution of the problem, and amongst these the most important are necessarily those of an anatomical nature. For if in any case of disease it can be shown that an anatomical formation is peculiar to a certain disease, the presumption is that it is congenital as a rule; and the earlier it shows itself the more likely is it to be hereditary. That both of these conditions have been fulfilled in regard to myopia by the change known as posterior staphyloma, is almost universally admitted. Thus Stellwag(11) declares that posterior staphyloma is due to congenital malformation, while Jaeger(12) says that "the increase in the axis is usually accompanied with a cone which is mostly congenital, and frequently hereditary," and adds that he has seen "typical cases of it in the eyes of new-born children both in life and after death;"(13) and Donders(14) only strengthens the almost universal opinion when he declares that from the frequency with which changes in the bottom of the eye have been observed with the ophthalmoscope, myopia and staphyloma have become nearly synonymous terms. This statement he supports by a reference to Graefe(15), who had previously declared that, in myopia of $\frac{1}{6}$ to $\frac{1}{2}$, ninety per cent. showed that peculiar change in the fundus, and adds that he himself(16) believes the proportion is much nearer one hundred per cent. Still there can be but little doubt that the general and rather sweeping assertion that posterior staphyloma is synonymous with myopia, is somewhat exaggerated. For out of 500 myopic eyes I found only 20.5 per cent. affected with the crescent. Cohn(17) in 1004 cases of myopia, between the same ages, that is, from 6 to 21 years, found the cone present in only 20 per cent.; while Max. Conrad's(18) examinations in 1001 myopes showed 28.1 per cent. The average of these results would then show that only 22.3 per cent. of myopic eyes have the cone. Of my own cases there were only one-fourth which showed any change whatever, while there were three-fourths of the eyes which showed no deviation from the normal standard. This is certainly at variance with the accepted views, though it is corroborated as seen above by the extensive statistics of Cohn and Conrad, the unanimity between the results being

very remarkable. When, however, we come to the proportionate frequency of the cone in regard to the three classes of refraction, we find that it predominates to a marked degree in myopia. Thus I found of 2265 eyes examined, that of the emmetropic eyes 3.33 per cent. had cones more or less developed; of the hypermetropic 3.49 per cent.; while in the myopic the percentage rose to 20.56 per cent., or six times as much as in either of the other classes. We are forced to conclude, therefore, that there is an anatomical variation predominating in myopic over other eyes, although the frequency with which it occurs has been, it would appear, much overrated—certainly for this country. Still, too much weight should not be put upon this fact as an argument as to the congenital and hereditary character of the cone, since, in the first place, an anomaly which makes its appearance in only about one-fourth of the cases, and under the most favorable auspices for its development, that is, in school children between the ages of six and twenty-one years, cannot be said to be strongly congenital.

Moreover, it is a fact that, although, as stated by Jaeger(19) and Von Hasner, well-marked cones are found in new-born children, they are, as known to all, and admitted by them, comparatively rare; while they increase in frequency in after life, and with close application; and oftentimes in eyes which have been shown by repeated ophthalmoscopic examinations to have been previously free from the slightest trace. From these facts, and from the fact that myopia consists as a rule simply of an elongation of the globe, which would, from purely mechanical reasons, produce a crescent, a doubt has arisen, in my own mind at least, whether after all the cone was an expression of congenital malformation of the scleral protuberance, or indeed of any part of the scleral split.

For, if it had any connection with the split, we should suppose it would be in reality what both its name and alleged anatomical origin imply, that is, an actual protrusion of the sclera, or at least an excavation or limited thinning of this membrane, as is invariably the case with a true coloboma, as demonstrated as well by the ophthalmoscope as by dissections. But I hardly need remind those of you who use the upright image that there is almost never the slightest difference in level between that part of the sclera which represents the cone and the adjoining portions, and this, too, no matter how large the crescent is. The cone is, in fact, not a true staphyloma; and it is only when applied to the entire posterior portion of the eye that the name is at all applicable, and we should be forced to assume that the whole posterior part of the eye was congenitally defective, or at least that parts were so which were widely separated from the scleral protuberance.

The second anatomical variation which is supposed to be characteristic of the myopic eye, and one which has had great importance attached to it, is the peculiar conformation of the ciliary muscle, which is supposed to be congenital. From the labors of Iwanoff(20) it was shown that the ciliary muscle in highly myopic eyes varied from that in other eyes, not only in general outline, but also in its composite structure. In the drawing (Fig. 1), which is taken from Iwanoff, the shape and outline of the muscle in the three classes of refraction are contrasted with each other. The solid line represents the muscle of an emmetropic, the dotted that of a myopic, and the broken line that of a hypermetropic, eye. It will be seen that the muscle in the myopic eye extends further back along the sclera than in the normal eye, and is, moreover, broader. It will be observed also that the short side, or leg, of the triangle, which repre-

sents the muscle as a whole, forms in the normal eye nearly a right angle with the sclera, while that in the myopic eye forms an acute angle, with the apex towards the inner portion of the eye. As to its component parts, it was shown that the circular fibres found in the normal eye were

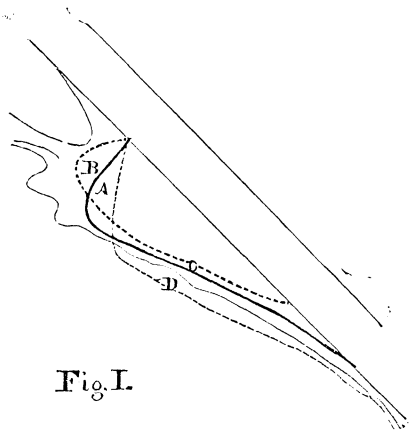


Fig. I.

either very much reduced or entirely wanting in the myopic eye, while they were increased in the hypermetropic eye. It was, therefore, assumed that, inasmuch as the myopic eye from its structure had no demands for active accommodative efforts, the reason why the circular fibres were not present was because they had become atrophied from disuse, so that the entire triangle marked A had disappeared. While, on the other hand, the circular fibres had been so increased by active accommodative demands in the hypermetropic eye as to occupy the superadded triangle B. It was further shown by Iwanoff that not only had the circular fibres become atrophic, but that the longitudinal had become hypertrophied. From this it was assumed that the action of the muscle had been changed, and that, instead of being compound, the circular fibres drawing toward the axis of the eye while the longitudinal drew directly forward, the action had become solely the latter, so that the muscle had become what the author calls a pure "tensor choroideæ." If we should accept this theory, we would have the apparent *reductio ad absurdum*, as the author himself says, in the development of a force which he supposes to produce active accommodation, in an eye which does not require or use it; for traction on the choroid and consequent relaxation of the zonula is supposed by Helmholtz and other physiologists to be the important element in the production of accommodation.

It would certainly lead me too far to enter in a discussion of this subject, but it seems as if a very important factor, and one which might produce a marked effect on the form and shape of the muscle in the myopic eye, had been neglected in the consideration of this question. This is the effect of distension of the investing membranes of the eye, which in some cases is enormous, as, for example, where progressive myopia is developed very rapidly and to a very high degree. It would, therefore, certainly seem more natural to attribute the change in form and structure to this than to any difference in function of its component parts. Thus if the posterior pole of the eye should yield, the insertion of the muscle at its choroidal end would be drawn back as seen in the drawing, and the angle

which, as we have seen, was a right angle in the normal eye would then become acute, since the insertion around the canal of Schlemm and adjacent parts would remain stationary. The few so-called circular fibres which form an anastomosing network, whose meshes run more or less perpendicular to the direction of the longitudinal fibres, would on being stretched assume precisely the opposite direction, and would then have on section the same general direction as the longitudinal, as would of course also the so-called radiating, fibres. Moreover, as the horizontal and vertical diameters also increase somewhat in these highly myopic eyes, the zonula would be stretched, and would then draw with a gentle but sustained traction on the muscle, the general direction being perpendicular to the axis of the eye. Thus the fibres of the muscle would have a tendency to draw apart, and the muscle to increase in bulk. This tension on the zonula would also account for the fact that the lens is flatter in high degrees of myopia than in the normal eye. Thus appearances hitherto supposed to be congenital might occur from a simple mechanical cause, acting during life in an eye which was not congenitally myopic, and with no hereditary tendency.

Thus while not denying the hereditary tendency towards a too yielding sclera, I have become more and more convinced that there are many cases in which the cone is simply the expression of a purely mechanical effect, distension, from which all the peculiar appearances of the crescent might result without there being the slightest connection between the cone and any congenital or hereditary tendency whatever. Does the inguinal ring never give way, or the fibres of the bladder become relaxed, or the pleura distended, or ascites occur, or even the bony cavity of the head enlarge, except through hereditary tendency? and if these can owe their origin, as they often do, to exciting causes which are purely spontaneous and fortuitous, why may not that distension of the sclera, which is myopia, take place, especially when the growing and elastic membrane is subjected to over-use, without our being compelled to attribute it "almost invariably to congenital and hereditary influences?" Clinical experience would point to such a conclusion, for it is a fact that cases occur where myopia with all its characteristic signs is produced in eyes which were previously normal; that is, in the eyes of adults, who have passed therefore the period of development, and whose family history shows no hereditary tendency. This, too, notwithstanding Donders's(21) assertion that he has never seen "myopia arise after the twentieth year in eyes which were previously normal." I have seen several such cases, and I doubt not that most practising ophthalmologists have had the same experience.

If this distension of the sclera did take place, there would be, necessarily, a change in the refraction of the eye, and this would have a decided weight in determining the question of hereditary influence. For if it could be shown that normal eyes which, according to the theory of heredity, would presuppose normal, *i. e.*, emmetropic, ancestry, or better still if it could be shown that hypermetropia, the direct opposite of myopia, with the opposite hereditary tendency, could and did frequently pass into myopia, then it would be an almost convincing proof that myopia could be and often was produced in spite of hereditary influence against it.

That an emmetropic eye can and does pass into a myopic eye, and that it has been proved to do so by direct observation, I think will be admitted by the great majority of observers. But that a hypermetropic

eye can pass into an emmetropic or normal eye, and thence into a myopic eye, is still a matter of great doubt in the minds of some of the best authorities. Thus Donders(22) says: "I have never seen a hypermetropically constructed eye become near-sighted." An opinion which he corroborates and strengthens in many other passages in his world-renowned book. Jaeger(23), however, makes a diametrically opposite statement, and declares that an interchange of refraction does take place, and that "thus an hypermetropic as well as a normally-constructed eye becomes a myopic eye through posterior staphyloma at the posterior pole of the eye." Stellwag(24) proclaims the same thing in very nearly identical terms. Various authorities too numerous to mention have ranged themselves on one side or the other of these opposite opinions, though I think that the majority of the more modern observers are tending towards the view expressed by Jaeger, that an interchange of refraction from a hypermetropic to a myopic eye can and does take place. Still it must be admitted, when it comes to actual demonstration, that the few cases which have thus far been cited are not sufficient in number or accuracy of detail to be of the slightest weight. One would certainly suppose, *à priori*, that such cases would have been observed in great numbers, and the fact that they have not, in the past decade when refraction has been so minutely studied, would seem to point very strongly to the fact that they did not exist.

In the dearth, therefore, of sufficient direct proof by observation of the passage in the same person of emmetropia into myopia, and the almost total want of such evidence of change in hypermetropia, we are forced to the consideration of collateral evidence which, it appears to me, if correctly taken and properly appreciated, is nearly as convincing as that of actual observation. I allude to the proof furnished by statistics taken from large masses of individuals, which show the proportionate rate in which the different classes of refraction occur at different times of life, and with different degrees and kind of application of the eyes. From the statistics of Ware(25), in 1813, and in later years from those of Szokalski(26), Schürmayer(27), Jaeger(28), Ruete(29), and others, very important facts were obtained. Thus it was shown by Ware that myopia was more common in cities than in the country, and that it increased in frequency and in degree with the age and amount of close application, while Ruete also called attention to the deleterious effect of insufficient and faulty illumination. These facts were afterwards corroborated by Cohn, but unfortunately Cohn's statistics, extensive as they are, are not suitable for our purpose, since, from the manner in which they were compiled, no correct idea can be obtained from them as to the frequency with which, in comparison to each other, the three different classes of refraction occur.

The statistics to which I shall call your attention are those made by Erismann(30) in Russia, Max Conrad(31) in Germany, and those by Dr. R. H. Derby and myself in this country. I choose these as examples since they are the only ones which I know of where sufficient numbers were used, and where, at the same time, the conditions of investigation were the same, or nearly the same, in each. Thus they were all upon the eyes of school children between the ages of six and twenty-one years, and the trial by glasses and Snellen's type at twenty feet were the basis of the examination; while all the more commonly occurring degrees of refraction from the lower ($\frac{1}{6}$) to the highest grades were included. Every eye was examined by the ophthalmoscope in the cases reported by Dr. Derby

and myself. Erismann's statistics were made in St. Petersburg on the eyes of 4358 scholars; Conrad's at Königsberg on 3036 eyes, and Dr. Derby's and mine on 2265 eyes in New York. The results of these statistics are arranged in a tabular form in the diagrams marked respectivly

Table I.

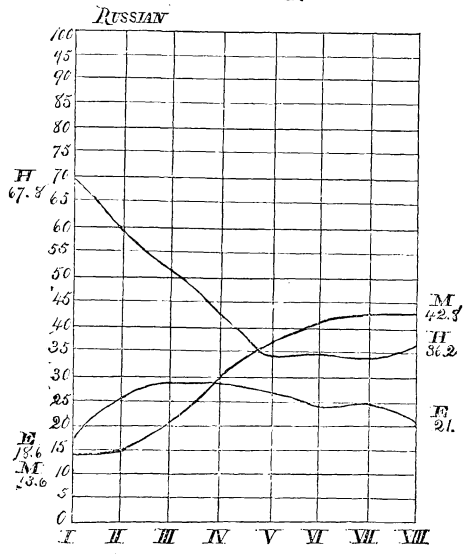
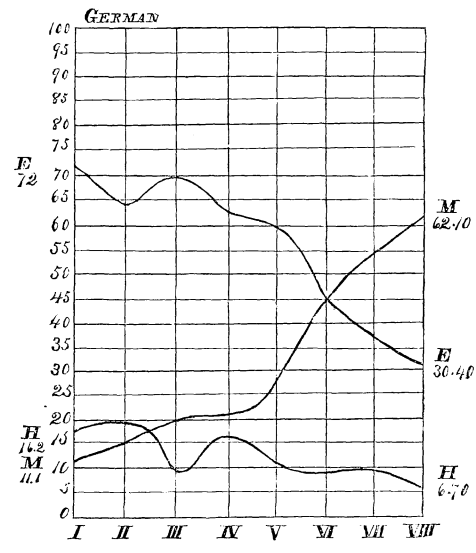


Table II.



Russian, German, and American, Tables I., II., III. The numbers at the foot of each chart represent the progression of the classes from left to right, that is, from the lowest to the highest, or, what is the same thing,

Table III.

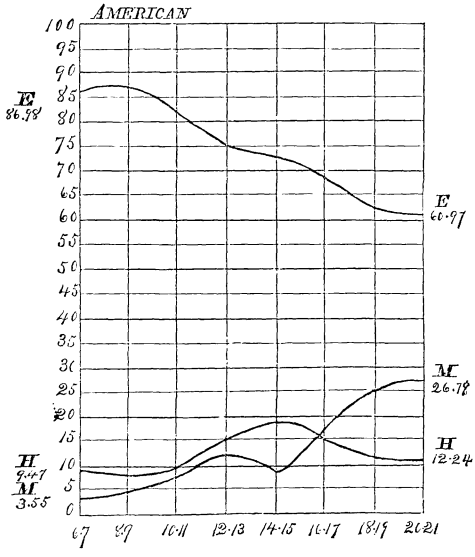
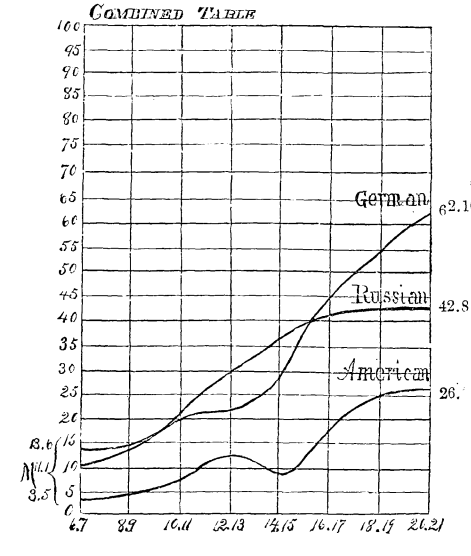


Table IV.



from the youngest to the oldest, as in the American diagram. This last is made necessary from the fact that the arrangement of the classes differs in this country from that in the foreign schools. Since, however, the successive classes correspond almost exactly to the successive ages, this can make no practical difference, especially as both the Russian and German statistics were calculated in both ways, and no material variation found between the results of the two methods. As it is the result of the entire school term, that is from six to twenty-one years, which now interests us, and which is the same in all countries, the comparison between the different nations is a just and fair one.

The numbers running longitudinally on the diagrams show the increase and decrease in percentage, each of the larger spaces representing ten, and each of the subdivisions five per cent. The line marked E represents the curve which emmetropia takes in the different years, M the myopic and H the hypermetropic curve. It will be seen on inspection, that, however much these curves differ in degree in the different diagrams, and amongst themselves, they all show that the refractive power increases with the advance in life.

Thus in the Russian diagram the frequency in which H occurs diminishes from 67.8 per cent. in the lowest class to 36.2 per cent. in the highest, while M increases from 13.6 per cent. to 42.8 per cent. Emmetropia increases a little, from 18.6 per cent. to 21 per cent. In the German[1] diagram, H descends from 16.2 per cent. to 6.70 per cent.; M rises from 11.1 per cent. to 62.1 per cent.; while E decreases from 72 per cent. to 30.40 per cent. In the American diagram, H rises from 9.47 per cent. to 12.24 per cent.; M rises from 3.55 per cent. to 26.79 per cent.; E sinks from 86.98 per cent. to 60.97 per cent. It will be seen from the Russian diagram that the emmetropia remains about the same through the entire series of classes, while the myopia steadily increases. If we assume that this increase, which is equal to 29.2 per cent., is due to emmetropia passing to myopia, we are forced to assume that the balance is maintained by hypermetropia passing to emmetropia to supply the deficiency. According to this diagram a part of the hypermetropia must become emmetropia, and if a hypermetropic eye can become emmetropic by increasing its refraction, it would be absurd to deny that it could not, by a little further increase, become myopic. So too in the German diagram it is seen that the increase in myopia, which equals 51 per cent., is greater than the decrease of emmetropia, which is 41.60 per cent., that is, is greater by 10 per cent. It must consequently have taken this increase from the hypermetropia, which is indeed seen to have decreased 10 per cent. From the American diagram it will be seen that the emmetropia decreases 26 per cent., while the myopia increases only 23 per cent., so that in this case the myopia may have been derived from the emmetropia alone. The slight increase in the hypermetropia may be explained by a small amount of latent H becoming manifest as the age advanced. The increase in refraction, however, though not so great as in the other two diagrams, is nevertheless very marked. It will, moreover, be seen in both the Russian and German diagrams that, while the lower grades of refraction E and H predominate

[1] Conrad also gives a separate curve, as determined with the ophthalmoscope. For the sake of simplicity and uniformity in the diagrams I have omitted this curve, and taken only those which were determined with glasses. The increase in refractive power is, however, more marked with the ophthalmoscope than with glasses. Still I cannot persuade myself that it is, on the whole, as accurate with the instrument as with test types.

in the lower classes, a complete change occurs, and M becomes the predominating refraction, and that this change takes place at a comparatively early age. Thus in the Russian diagram the line M crosses the line E at the fourth class, or what would correspond on the American diagram to the years 12–13. It crosses H at 14–15, when myopia becomes the predominating refraction, being in the highest class twice as frequent as the normal eye. Precisely the same thing occurs in the German diagram, where M, rapidly rising, crosses both H and E, being in the highest classes twice as common as the normal eye. In the American diagram, however, E remains all through by far the predominating refraction, the line E not only never crossing those of H or M, but remaining at a distance amounting at its lowest point in respect to M at its highest, to 34 per cent., and in respect to H at its highest, to 43 per cent.

This would show, if the numbers were great enough to prove it, and I think they are, that not only is there less myopia in this country in school children than in either Russia or Germany, but that emmetropia is the normal eye in all classes, which agrees with what Donders(32) found among the Dutch. For it may be assumed that after the age of twenty-one the refraction as a grand whole changes but very little, and that any slight change towards myopia in early adult life is more than counterbalanced by acquired hypermetropia in later life. Moreover, as we have taken as a standard the very class where myopia would be most frequent, that is in over-worked school children, it is fair to assume that the proportion of myopia would be less and not greater in other classes, especially in the lower and middle ranks of life. In order to make a better comparison between the three nations, as far as the myopia is concerned, the myopic curve of the three preceding tables has been transferred to a separate diagram, Table IV.

As an additional means of estimating the effect which the hereditary influence has on myopia, a comparison was made as to the frequency in which myopia occurred in the three principal nationalities of which our public schools are composed, that is, among the pupils of German, American, and Irish parentage. It was found that of all the German scholars 24 per cent. were myopic, of the Americans 20 per cent., and of the Irish 15 per cent.; so that even in this country, and under the same school influences, myopia occurs more frequently among the descendants of Germans than among either the Americans or Irish. The comparatively low percentage of myopia among the Irish is certainly remarkable. It is, however, in accordance with the assertions of various authors as to the immunity from myopia among the inhabitants of Great Britain. There can be no doubt that as a nation the Germans show a strong tendency towards myopia. Whatever may be the cause of the trouble, it is certainly a fact that the statistics of myopia taken in German schools show a decided increase over those taken in this country, which can be seen by comparing the statistics taken by Dr. Cheatham(33) in New York, by Dr. D. B. Williams(34) in Cincinnati, and Drs. Prout and Mathewson(35) in Brooklyn, with those taken in Germany. In comparing these statistics the different basis on which they are compiled should be taken into consideration, especially with those of Cohn, as he did not include myopia less than $\frac{1}{36}$, while in those cited above and made in this country $\frac{1}{60}$ was included.

From the evidence furnished by such statistics made in different countries and by different observers, I do not, while admitting the hereditary tendency, see how we can exclude the fact that there are many eyes

which, under prolonged tension of the accommodation, pass from hypermetropia to emmetropia, and thence to myopia with all its attending signs and symptoms, and this too in spite of hereditary influence to the contrary.

This factor of prolonged tension of the accommodation brings us at once to a consideration of the second proposition of our subject, which is whether posterior staphyloma and progressive myopia can be produced through the influence of the ciliary muscle. Before proceeding to discuss this, I would call your attention to the fact that the question does not refer to the entire act of accommodation, which implies the action of the recti muscles as well as that of the ciliary muscle, but is limited to the latter alone, and consequently I am desirous that it should be kept in mind that my remarks are confined to the share which the ciliary muscle alone takes as an agent in the production of myopia and the cone. There are two ways by which the ciliary muscle could render an eye previously not near-sighted, myopic. The first would be by increasing the curvature of the lens by active, that is, positive, contraction; and the second, by producing an elongation of the antero-posterior axis. That such an increase in curvature of the lens does, by contraction of the muscle, take place, with the effect of temporarily increasing the refraction of the eye, is now universally admitted. The only question which interests us here is whether the increased curvature of the lens through the agency of the ciliary muscle ever becomes permanent, and thus transforms an eye from an emmetropic or hypermetropic to a myopic eye. That such was the opinion of the earlier oculists I need not remind you, down even to the time when Cramer declared that there were myopic eyes in which the curvature of the lens corresponded to that in the normal eye when adjusted for near objects. Nor need I dwell on the revulsion of feeling which followed when it was learned from the labors of Helmholtz, Knapp, and Donders, that this last fact was not correct, and that the curvature of the lens in myopic eyes was shown to be if anything less than in the normal eye. A revulsion of opinion which culminated in Donders's(36) declaring, though formerly a firm believer in its occurrence, that he had never, since the true nature of refraction had been understood, been compelled to resort to an increased curvature of the lens as an explanation of myopia, while Giraud Teulon declares that there never has been a single well-authenticated case put on record.

The opinion that myopia is never due to increased curvature of the lens, has, however, been combated from the first by a few of the very best authorities, especially by Jaeger(37), who declares that "frequently the sole cause of myopia is the increased curvature of the lens," and by Stellwag(38), who in his last edition affirms that "increase in convexity of the lens is indisputably an important pathogenetic cause of short-sightedness;" and it cannot be doubted that this opinion, certainly as regards the initial cause of myopia, is gaining ground among the younger school. Still, notwithstanding the plausibility of the theory that increased curvature of the lens causes myopia, there is, it must be confessed, but little actual proof of its existence from such a cause. Indeed it must, I think, be admitted that the proof both experimentally and clinically points just the other way. Thus experimentation shows that by actual measurement the curvature of the lens is less if anything in a myopic than in a normal eye. Then comes the great clinical fact that in those cases of hypermetropia of a high degree, proved to exist in young children who are watched from year to year, the amount of the *total* hypermetropia does not decrease under

accommodative efforts, while the manifest does increase year by year in spite of such efforts. Thus the patient is compelled to go, not from stronger to weaker glasses, but from stronger to stronger, in spite of every effort to maintain the former amount of curvature of the lens. The very frequent occurrence, too, of adults with very high degrees of hypermetropia, who have been straining their ciliary muscle to the utmost for years to overcome their optical defect, is decidedly against the view that the curvature of the lens becomes permanently increased by over-tension; as is also the fact that in high degrees of hypermetropia where there is little or no manifest, the total is revealed by the ophthalmoscope, and then coincides with an amazing exactness with that produced by the full effect of atropine. Such facts as these, and many others might be quoted to sustain them, warrant us in the belief, until more convincing proof to the contrary is offered, that as a rule the action of the ciliary muscle has no effect in producing a permanent increase of curvature of the lens, and thus becoming a cause of myopia—no matter what the original condition of refraction. In regard to the second point in our investigation, that is, whether the ciliary muscle is capable of producing an elongation of the axis, thus causing myopia, it will be sufficient to call to mind that Young's(39) and Helmholtz's(40) investigations prove the impossibility of such a result.

But it may be urged—as, indeed, it has been frequently, especially since Dobrowolsky's(41) investigations—that, even if the natural, or what may be called the tonic, contractions of the muscle, do not produce myopia by directly increasing the curvature of the lens, spasmodic or clonic contractions do; and that the myopia first produced by increased curvature of the lens is followed by irritation of, and traction on, the deeper-seated membranes, which lead to true myopia—that is, to an elongation of the axis. If this were so, we should expect to find the signs of irritation more frequent where the strain of the ciliary muscle was the greatest—that is, in hypermetropic eyes. But it is an undeniable fact, as pointed out by Donders, and confirmed by every observer with the ophthalmoscope, that the injection of the nerve and the signs of irritation are not any greater than in the normal eye. Moreover, we should find, if simple traction on the choroid could produce the cone, that these would be most frequent where the traction of the muscle was the greatest; and this, too, independently of any increase of the length of the axis—that is to say, in hypermetropic eyes. For, if simple traction on the choroid could produce the cone, it would make no difference how short the axis was; all that would be necessary would be an increased action of the muscle, and this we get in hypermetropia. Now, not only clinical experience, but actual statistics, show, as has already been pointed out, that the crescent is found less frequently in hypermetropic eyes than any other. Moreover, if traction on the choroid could and did produce the cone, we should expect to find it in all eyes, no matter what their refraction, at the *inside* of the nerve, since this would be the place of all others in the circumference of the nerve where the traction would be soonest and most powerfully felt, for the simple reason that it is the shortest line between the two points of attachment. Moreover, the crescent would have a rapid tendency to become circular. But ninety-nine times out of a hundred the cone is at the outside of the nerve, and there remains.

But not only do we have positive proof in hypermetropic eyes that increased action of the ciliary muscle, *per se*, does not produce the cone,

but we have equally strong *negative* proof, in myopic eyes; for the greater the myopia, the less the positive contraction of the ciliary muscle. And many cases occur of the development of the cone towards adult life in eyes which were not at the time of development of the crescent using, nor had ever used, any active accommodation at all; that is, in eyes whose far point had always been from twelve to eight inches. Of all eyes these are the most prone, on over-use, to progressive myopia and the formation of the cone, while it is a clinical fact that, in many of these cases of rapidly increasing myopia, the myopia is at once checked by carrying out the far point by glasses which decrease the amount of convergence, but which increase sometimes to a great degree the demands on the muscle, especially where, as in young people, we completely neutralize the error in refraction. I do not mean to say that the cone is not due to irritation and traction, for I am firmly convinced that traction on the choroid in the plane of its extent, and pressure on its surface, are the principal if not the only causes of the cone. But what I do mean is that this traction takes place from extension at the posterior pole of the eye, not by the contraction of the ciliary muscle at the anterior parts.

It has never been demonstrated in the slightest degree that the ciliary muscle exerts the least traction on the posterior parts of the choroid. Indeed, the little experimental evidence which we have is directly opposed to such a supposition, as is indeed all the clinical evidence. There is, moreover, to say the least, a strong doubt whether the muscle exerts any traction even on the anterior portions of the choroid, and some of those who have been hitherto the most enthusiastic supporters of the theory of the meridional portion of the muscle being a direct "tensor choroideæ," seem to have modified, if not completely changed, their views. Thus Iwanoff (42) latterly, after describing the anatomy of the meridional portion, and its mode of insertion into the *L. suprachoroidea*, says: "Now, with such a disposition of the meridional portion of the muscle with the *L. suprachoroidea*, and with such a structure of this latter, it is self-evident that the deep layers of the choroid (choriocapillaris and middle choroid) cannot be essentially stretched. Consequently, the entire effect of the contraction of the ciliary muscle will limit itself to an extension of the ciliary body, which, as is known, is in the closest connection with the zonula of Zinn, while in the choroid itself, at the most, there will be only an extension of the L. suprachoroidea. This is perfectly evident when we bear in mind that that part of the choroid which is situated behind the *ora serrata* has no direct connection whatever with the zonula." Nevertheless, that enforced and maintained contraction of the ciliary muscle should produce that nervous exhaustion and irritation, with altered nutrition, which always occurs in the case of an overtasked muscle, is most natural. That this irritation should extend to the surrounding parts, causing increased vascularity, and, what is more, increased secretion, by which the intraocular pressure is augmented, might, I think, occur to some degree, in spite, too, of the assertion of Leber that the vascular supply to the ciliary muscle is so arranged that the circulation is not impeded by its contraction. Still, from what has already been stated, I am inclined to believe that this increased intraocular pressure, which is so potent a factor in the production of myopia, occurs much more frequently from other causes, especially from faulty convergence, than from the simple action of the ciliary muscle. This leads me to believe, although I appreciate most highly the labors of

Dobrowolsky,[1] that spasm of the ciliary muscle, so well recognized and described by the elder writers, has been somewhat exaggerated by the younger school both as to its frequency and the amount of influence it exerts in the production of myopia.

I have not the time to go into an extended discussion of this question, which it surely deserves, and must therefore content myself by pointing out the fact that it would appear that two important conditions have been ignored in the works of Dobrowolsky and his followers, Schiess-Gemuseus(43) and Dr. Derby(44) of Boston. The first is the physiological effect of atropine, which is to reduce the refraction to an amount which, according to Donders(45), would be expressed by $+\frac{1}{6}\frac{1}{0}$, or even $+\frac{1}{4}\frac{1}{0}$. The ordinary emmetropic eye then under atropine would be a hypermetropic eye of $\frac{1}{6}\frac{1}{0}$. Are we to assume, then, as Dobrowolsky does, that a decrease of refraction which amounts to only $\frac{1}{3}\frac{1}{6}\frac{1}{0}$, $\frac{1}{2}\frac{1}{4}\frac{1}{0}$, $\frac{1}{1}\frac{1}{2}\frac{1}{0}$, or even $\frac{1}{9}\frac{1}{6}$, is the result of spasm, and not the action of the drug, when the physiological action is often equal to six times the amount; and especially are we to assume this when solid atropine has been used three or four times a day for three or four weeks, or even months?

Dobrowolsky(46) gives a table of 105 cases of myopia. In 69 of these there was a decrease after the use of atropine—that is, in seventy per cent. But of these cases, when we make an allowance for the physiological effect, only thirty remain. Of these thirty, after this allowance, six show a decrease of $\frac{1}{2}\frac{1}{4}\frac{1}{0}$ or less, twelve of $\frac{1}{1}\frac{1}{2}\frac{1}{0}$ or less, seven of $\frac{1}{6}\frac{1}{0}$, three of $\frac{1}{4}\frac{1}{0}$, and two of $\frac{1}{1}\frac{1}{5}$ or less. So that there are only five cases in the entire number which show a moderate degree of spasm, and only two of these a high degree; and in all these five cases the myopia is of an excessive grade, varying between $\frac{1}{3.3}$ and $\frac{1}{2.2}$. Now, these high degrees of myopia are just the conditions in which we always expect, whether there be any insufficiency or not, an apparent increase in the refraction, caused by an excessive muscular strain at convergence. This is invariably accompanied with a displacement of the relative accommodation inward, and the slight temporary decrease in the refraction caused by the use of strong atropine invariably returns as soon as the atropine is left off, unless the strain on the convergence is relieved by suitable means. Exceedingly interesting in this connection are some of the results published by the earlier writers—Bonnet, Cuvier, Philips, and Jules Guérin—but particularly the case related by Giraud Teulon(47), of extreme myopia, in which tenotomy of the external rectus suddenly diminished the myopia by $\frac{1}{1}\frac{1}{3}$, or from $\frac{1}{5}$ to $\frac{1}{8}$. Had this amount of decrease in refraction been brought to light by the use of atropine, it would certainly have been put down by these observers as due to spasm; and it just as certainly would have returned the moment the atropine had been discontinued, unless the strain on the interni had been removed. The analogue of this, the sudden development of manifest H, which had been previously latent, after tenotomy, is too common an occurrence to need any comment whatever. It strikes me, therefore, that want of attention to these two points, namely, the physiological action of atropine and the associated action of the recti interni on the accommodation, has led these observers into a false estimate of the frequency of spasm, and its predominating influence in the production of myopia. Certainly my own

[1] I cannot agree with a recent writer, who says: "To Professor Schiess belongs the credit of utilizing the researches of Dobrowolsky for ophthalmic practice." Whatever there is of merit in this matter belongs, both theoretically and practically, entirely to Dobrowolsky.

experience does not coincide with that of these observers, and I fail to find a frequent or great diminution, even in myopic eyes of young children, after the use of atropine. This I was glad to see had been the experience of Donders(48), as expressed by him at the last International Congress.

Up to this point we have been occupied with a consideration of the action of the ciliary muscle as a whole, but a theory has been started that contraction of the muscle can take place in the direction of one meridian alone, or in different meridians in different degrees at the same time. Thus, according to Dobrowolsky(49), a complete interchange of kind and degree of astigmatism, as well as change of direction in the axis, may result from these meridional contractions, so that an eye which was originally hypermetropic in one meridian may by a counterbalancing contraction of the muscle become an emmetropic eye, or an emmetropic eye an astigmatic eye, with M in one meridian alone, or M in all meridians, and more pronounced in one than the rest; and so on with a change in all the varieties of astigmatism which could be produced through positive accommodation. It is claimed that this is a fertile source of myopia.

That a slight change in a meridian, whether of refraction or direction, should take place, would not appear strange; for there is nothing absolutely stable in the human body. This would, moreover, be in accordance with what has been supposed possible by many and corroborated in some degree by certain investigations made by Woinow(50). But any such remarkable changes as are pointed out by Dobrowolsky, either in kind or degree, I have never met with myself, nor do I know of any sufficient corroboration of them by others. For this reason I do not feel warranted in expressing an opinion in regard to them; certainly not one which would imply a belief as to their active agency in producing myopia.

These views of Dobrowolsky were followed by those of Dr. Thomson(51), of Philadelphia, based on the old theory of the antagonistic action of the two portions of the ciliary muscle. According to these views, not positive accommodation alone, as with Dobrowolsky, but even negative accommodation took an active part, through the contraction of the radial fibres of the ciliary muscle as opposed to the circular fibres. Thus a defined traction in a meridional direction was brought to bear on the choroid, which would naturally exert its influence at a corresponding point at its attachment at the circumference of the disk. This would produce the cone, which would then lie in the plane and direction of this traction.

I can only say in regard to this that our present knowledge of the action of the ciliary muscle does not support this conclusion. For notwithstanding the many attempts to prove the fact of negative accommodation, ever since the discovery of the ciliary muscle, not the slightest trace of its existence has ever been demonstrated; while on the other hand the fact of its non-existence has been, it would seem to me, incontestably shown. Until, then, the existence of negative accommodation has been proved, it would be a waste of time to speculate on what would be its effect on myopia, or rather what its effect is. We have no right to assume both the cause and effect too. But admitting the existence of negative accommodation to the fullest degree, even to the extent that it surpasses the positive, many facts would have to be explained before its effect on the cone could be admitted. First, why is it that we see the

highest degrees of myopia with immense cones, and either not a trace of astigmatism, or, if present, of such a very low degree as to preclude the idea that the cone is the result of the astigmatism? Secondly, why is it that we see such very high degrees of simple myopic astigmatism without a vestige of a cone? Thus in the past few months I have seen the following cases where there was no trace of a crescent: one case of $\frac{1}{5}$ c., two of $\frac{1}{6}$ c., one of $\frac{1}{7}$ c., two of $\frac{1}{8}$ c., one of $\frac{1}{10}$ c., three of $\frac{1}{11}$ c., and four of $\frac{1}{12}$ c.; all simple, myopic astigmatism. Thomson(52) himself has reported some cases of excessive asymmetry of the cornea, in which, to use his own words, "it is worthy of remark that in this last case, with such high degree of myopia and the use for years of glasses so unsuited, there was not the slightest appearance of the crescent at the optic disk." Again, it would have to be shown why it is that the cone sometimes runs in the same direction as the meridian of greatest curvature, and sometimes in that of the least, or again in neither. Then, too, if traction in one meridian had anything to do with the direction of the cone, why is it that it is limited so often to the outside of the nerve, and does not extend equally in the same direction on the opposite side? since it can hardly be assumed that these meridional contractions take place only in one-half of the ciliary muscle, and this, in the vast majority of the cases, the outer half.

We should have also to explain why it is that in sections of myopic eyes we find the choroidal pigment layer drawn away from the outer edge of the nerve and *over* the inner edge. In regard to the appearances of traction upon the vessels, and other details of the fundus as seen with the ophthalmoscope in astigmatism, I would say that this, as far as my experience goes, corresponds to the distortion produced by the optical defect of the refracting media, which causes the line of traction to appear to be always in the meridian of greatest curvature.

This brings me to the close of my remarks, from which I would in keeping with the condensed character of my paper make the following brief conclusions:—

I. From the fact that there is so large a percentage of children who are myopic, but whose parents are not near-sighted, while the myopia increases directly with the amount of increased tension of the eyes, and from the fact that an interchange of refraction may occur, whereby an eye which is not congenitally myopic may become so in spite of hereditary tendency, it would seem to follow that hereditary predisposition, though undoubtedly a potent cause, is not only not the sole cause, but is not even the predominating cause of myopia.

II. In regard to the second question, as to whether the ciliary muscle acting through faulty refraction can produce myopia and the cone, I conclude that the action of the ciliary muscle, taken by itself, exerts but little influence on the production of myopia, and still less on the formation of the cone.

I cannot refrain before closing from asking you once more to bear in mind that the questions submitted were not as to what the causes of myopia were, but simply the influence of two fixed and defined factors in its production. The briefest consideration of these two subjects which I could possibly make, has already led me beyond the limits of the time allowed, and I fear also of your patience. I have for these reasons been compelled to forego saying much which I am conscious that a just consideration of the subject would demand, and to discard much that I had already written.

Bibliography.

(1) Lucas, P. Traité philosophique et physiologique. Tome i. p. 391, 1847.
(2) Funari. Annales d'Oculistique. Tome x. p. 145.
(3) Piorry. Heredité dans les Maladies. p. 120.
(4) Portal. Considération sur les Maladies de Famille.
(5) Stellwag von Carion. On the Eye. American Transl., 1873, p. 354, p. 703.
(6) Jaeger. Einstellungen des diopt. Apparates, u. s. w., 1861. S. 71.
(7) Donders. Accommodation and Refraction of the Eye, 1864, p. 350.
(8) Cohn, H. Untersuchungen der Augen von 10,060 Schulkindern, u. s. w., Leipzig, 1867. S. 63.
(9) Ibid., p. 129.
(10) Erismann, F. Ein Beitrag zur Entwickelungs-Geschichte der Myopie. Græfe's Archiv, Band xvii. Ab. 1. S. 1.
(11) Stellwag. Die Accommodationsfehler des Auges, 1855. Wien Sitzungberichte der mathem-naturw. Classe der kais. Akademie der Wissenschaften, Bd. xvi. S. 225.
(12) Jaeger. Einstellungen. S. 27.
(13) Ibid., 37.
(14) Donders. Accommodation and Refraction, p. 354.
(15) Græfe. Archiv für Ophth., 1854, B. 1. S. 394.
(16) Donders. Accommodation and Refraction, p. 443.
(17) Cohn, H. Untersuchungen. S. 61.
(18) Conrad, Max. Die Refraction von 3036 Augen von Schulkindern mit Rücksicht auf den Uebergang der Hypermetropie in Myopie. Inaugural-Dissertation. S. 38.
(19) Jaeger. Einstellungen. S. 37.
(20) Iwanoff. Græfe's Archiv, B. xv. Ab. 3. S. 284.
(21) Donders. Accommodation and Refraction, p. 384.
(22) Ibid., p. 349.
(23) Jaeger. Einstellungen. S. 104.
(24) Stellwag. On the Eye. American Transl., 4th Edition, p. 723.
(25) Ware, J. C., F.R.S. Observations Relative to the Near and Distant Sight of different Persons. Phil. Trans. Royal Soc., 1813.
(26) Szokalski. Prager Vierteljahrschrift für pract. Heilkunde, 1848.
(27) Schürmayer, J. H. Handbuch der Medicin Policei. S. 61, 1856.
(28) Jaeger. Einstellungen. S. 20.
(29) Ruete. Untersuchungen über die Augenkrankheiten bei Schulkindern. Zeitschrift für Medicin, Chirurgie, u. s. w. Neue Folge B. v. Heft 4. Leipzig, 1866.
(30) Erismann. Græfe's Archiv, Band xvii. Ab. 1. S. 13.
(31) Conrad, Max. Inaugural-Dissertation, Könisberg. S. 16.
(32) Donders. Accommodation and Refraction, p. 341.
(33) Cheatham. Boston Medical and Surgical Journal, May 27, 1875, p. 633.
(34) Williams, D. B., Ibid.
(35) Prout and Mathewson, Ibid.
(36) Donders. Accommodation and Refraction, p. 394.
(37) Jaeger. Einstellungen. S. 26.
(38) Stellwag. On the Eye, 4th Edition, American Transl., p. 704.
(39) Young, Thos. Mechanism of the Eye. Phil. Trans., 1801.

(40) Helmholtz. Karsten's Encyclopædia der Physik, 1856.
(41) Dobrowolsky. Beiträge zur Lehre von den Anomalien der Refraction, u. s. w. Zehender klin. Monatsblätter. Ausserordentliches Beilageheft, 1868.
(42) Iwanoff, Græfe, und Sämisch. Handbuch der Augenheilkunde, Band i. Theil. l. S. 277.
(43) Schiess-Gemuseus. Beiträge zur Therapie der Myopie, Basel, 1872.
(44) Derby, H. Trans. American Ophth. Soc., 1874, p. 138.
(45) Donders. Accommodation and Refraction, pp. 174, 600.
(46) Dobrowolsky. Zehender ausserordentliches Beilageheft, 1868. S. 37.
(47) Teulon, Giraud. Ophth. Hosp. Reports, vol. v. p. 381.
(48) Donders. Report of fourth International Congress, London, 1872, p. 69.
(49) Dobrowolsky. Græfe's Archiv, xiv. Ab. 3. S. 50.
(50) Woinow. Græfe's Archiv, xv. Ab. 2. S. 167.
(51) Thomson, Wm. Trans. American Ophth. Soc., 1874, p. 132.
(52) Thomson, Wm. Trans. American Ophth. Soc., 1875, p. 310.

Discussion on Dr. Loring's Paper.

After the reading of the preceding paper, Dr. J. Green, of St. Louis, said:—As bearing on the question of the influence of hereditary transmission, we must remember that we scarcely ever see myopic children whose parents and grandparents have been equally subjected to the general exciting causes of myopia. Taking, therefore, into account this absence of exciting causes in the case of the ancestors, it will not do to assume the absence of hereditary predisposition to myopia even where there has been no declared myopia in parents and grandparents. Again, in all our published statistics there is great imperfection as regards the recognition of astigmatism, and yet among the causes which produce indistinctness of vision, and thus favor the development of myopia, astigmatism certainly holds an important place. The statistics are thus of less value than many suppose, in clearing up this part of the subject. It is a very significant fact that in such cases of myopia as give the patient trouble enough to lead him to consult an oculist, we find astigmatism oftener present than absent; hence, in investigating the causes of myopia, we must make more careful studies of the refraction, not merely with reference to myopia and hypermetropia, but also with reference to astigmatism.

Dr. W. Thomson, of Philadelphia, said:—I think that Dr. Green has called attention to an important fact in alluding to the large number of cases of astigmatism which complicate myopia. That myopia is very often progressive, we know, but I think from observation that cases of myopia, pure and simple, are hard to find. High grades of myopia are, as a rule, characterized by astigmatism, and therefore we ought to consider whether most cases of progressive myopia have not commenced as slight cases of astigmatism. If we accept the conclusions of the paper, the question of progressive myopia would be hopeless, and we would have nothing to do but to look on and see people growing worse day by day. If it is conceded that astigmatism is often an efficient cause of myopia, we have then a remedy in cylindrical glasses. I think that astigmatism is one of the active causes of progressive myopia.

Dr. Loring said:—I would call attention to the fact that astigmatism was not mentioned in the question for discussion, but I admit that it has something to do with myopia. Dr. Thomson takes a gloomy view of the subject. I believe that, if we go on as in Germany, the time will come when myopic refraction will predominate. In keeping children in school twelve hours a day, poring over books, a great hygienic principle is overlooked, and, as long as

this is done, so long will there be myopia. I cannot agree that the time will ever come when myopia will be prevented by the use of cylindrical glasses.

Dr. H. W. WILLIAMS, of Boston, said:—Dr. Loring says justly that myopic astigmatism is a form of myopia. I think it is conceded that hypermetropia and astigmatism are hereditary. I know several instances in which persons are myopic in one eye, and emmetropic or hypermetropic in the other, and I would ask if there is a hereditary predisposition to myopia in one eye, and not in the other? And I have seen such cases, in which at a later period the myopia has increased, and in which the hypermetropic or emmetropic eye has become myopic. I am satisfied that myopia is largely due to artificial causes.

The President, Mr. R. BRUDENELL CARTER, of London, said:—I remember the case of a young lady who was brought to me with a low degree of hypermetropia in one eye, and a high degree in the other. In the case of her father and mother both eyes were hypermetropic. I do not believe that statistics will enable us to arrive with certainty at any conclusion upon this subject. In looking back I can recall a few cases of myopia which were the result of accident. I remember at least one case in which this condition was produced by a blow.

Dr. THOMSON said:—With regard to Dr. Loring's second conclusion, I think that the whole question resolves itself into whether or not the ciliary muscle is a single or a double muscle, and that unless new light is thrown on the subject by an appeal to clinical observation, there is nothing to do but to accept this conclusion. I am convinced that the changes in the back part of the eye in myopia have something to do with the existence of astigmatism, and that the appearances which they assume are largely dependent upon the direction of the principal meridians of the astigmatic eye. If a conus is found in any part, there will be found to be a relation between the direction of the conus and that of the cylindrical glass which gives the highest vision. I believe that I have established the fact that in many cases of myopia of high grade, say one-fifth, one-sixth, or one-seventh, in which neutralizing spherical glasses have been habitually worn, the optic nerve is free from distortion, and has no displacement, but that when astigmatism, even of low grade, is found conjoined with a high degree of myopia, a conspicuous conus will be found.

Dr. E. WILLIAMS, of Cincinnati, said:—I have seen several cases which I may cite in confirmation of Dr. Thomson's remarks. I remember the cases of a distinguished lawyer and of a merchant, both over fifty years of age, who had worn glasses since they had left school, and in neither of them was conus found. When we find a patient wearing the proper glasses, and seeing perfectly with them, we rarely find a conus, but when vision, even with the glasses, is more or less indistinct, we may expect to find the conus.

Dr. S. D. RISLEY, of Philadelphia, said:—I think that we rarely see progressive myopia with conus without astigmatism, and, on the other hand, that conus is not confined to myopic eyes, but is a very frequent occurrence in hypermetropic eyes with astigmatism. I recall two cases in which conus and myopia had appeared while under observation, and were attended by marked asthenopia which was relieved, and further choroidal change arrested, by the rest afforded the ciliary muscle by a carefully adjusted, correcting glass. I am convinced, from clinical experience, that the ciliary muscle has much to do with progressive myopia and the formation of the conus.

Dr. THOMSON said:—I know of a number of cases of asthenopia from insufficiency of the internal recti, which bear upon this question. In one case, in which the asthenopia had existed over a period of thirty years, and had caused much distress, and in which the asthenopia was perfectly relieved by the use of proper prismatic glasses, there was absolutely no trace of conus in either eye.

Dr. LORING said:—The use of concave glasses often stops the development of the conus and the myopia, and, nevertheless, the use of the glasses forces the accommodation more than when they are not employed. All admit that concave glasses are useful in myopia, yet concave glasses act directly upon the ciliary muscle. That the ciliary muscle has two antagonistic functions is, to say the least, extremely improbable.