doctor any anxiety. Obviously, if a doctor or a nurse has a severe cold or a bad throat they are a danger to everyone with whom they come in contact, whether pregnant or non-pregnant. There are also elementary rules regarding coughing and sneezing which it is hoped need not be laid down as exclusively obstetrical.

CONCLUSION

1. The obvious lack in the present system is good nursing, and the carrying out of those antiseptic and aseptic principles which good nursing implies.

2. Next in order comes lack of ante-natal care, which is bound to persist until doctors, and especially those keen on the work, are called in by their patients in the first instance, and not by midwives.

Lastly, until Items 1 and 2 above are corrected, errors of judgement and technique are bound to be made, which in print seem so regrettable, but which to anyone with a practical knowledge of obstetrics, especially in industrial practice, are, under present-day conditions, in many cases not only understandable, but even excusable.

THE CONTROL OF SCHOOL MYOPIA *

BY

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As a descriptive term the name "school myopia" leaves little to be desired. It well conveys the fact that in a large group of children myopia makes its appearance in school life, generally at about the age of 11, and steadily progresses for a few years. The implication that the school is responsible for this process is, however, an assumption of considerable antiquity, for over 300 years ago Kepler first pointed out that short sight is more common among students and others who use their eyes excessively. Attempts to control school myopia have been largely based on some theory or other as to the origin of myopia. On conflicting views as to the role of accommodation in the development of myopia, under-correction and full correction of the refractive error, as also treatment by atropine or eserine, have found their exponents. Tenotomy of the internal recti has been practised by those who see excessive convergence as the responsible factor, and even tenotomy of the oblique muscles has been advocated by those to whom the myopic eye is an eye compressed by a low orbit. Iridectomy to obviate a hypothetical increased intraocular tension causing myopia has been carried out, as also has repeated paracentesis of the anterior chamber. Complete bodily rest has had its advocates, but so too have general exercises and massage of the globe. All sorts of postures to be adopted when doing near work, and all sorts of appliances to aid these postures, have been in fashion at one time or other. Subconjunctival injections of salt solutions of varying concentration, dionine, and adrenaline have all been employed, whilst of recent years radium has been brought forward as a means of cure. But it is mainly along the lines laid down by Hermann Cohn in 1867, and subsequently, that attempts at controlling myopia have been carried out. Cohn and his successor saw myopia as the result of too much close work, and they advocated hygienic measures to minimize the evils induced by excessive use of the eyes. Good illumination was needed in order to avoid unnecessary close application, with its consequent excessive accommo-

dation and convergence; furthermore, stooping over work, which was held to produce congestion of the eyes and raised intraocular tension, could also be thus obviated, especially if the light was suitably distributed over specially constructed desks and benches. The elimination of Gothic script and the use of well-printed books was likewise a corollary of these views. At the same time the advocates of these opinions stressed the necessity of general hygienic measures. Cohn pleaded for school medical services, and, whatever be the ultimate value of his ideas on the causation of myopia, his work will always remain as a pioneer effort in school hygiene.

FIFTY YEARS OF ATTEMPTED CONTROL OF MYOPIA

Over half a century has now elapsed since Cohn's advocacy has become effective. One thing is clear: myopia has not been banished, and no unequivocal answer can be given to the question whether myopia has become less frequent. Many factors have to be considered in any comparison in the frequency of the myopia fifty years ago and to-day. The data are not always strictly comparable, and the great social changes that have occurred in the interval have brought with them a readjustment in the strata of society and changes in the environment of children that are not to be lightly dismissed in any such consideration. Even so, available data are scanty and their interpretation open to dispute.

Seggel, reporting on the changes that have occurred at Munich, holds that the incidence of myopia has decreased by 10 per cent. in the case of boys and by 30 per cent. in the case of girls during twenty years. Ask, likewise, reports a decrease in Sweden: in 1906 he found the incidence of myopia in schoolboys to be 25 per cent., whereas in 1883 A. Key had found it to be about 40 to 50 per cent. Ask's conclusions were questioned by Heinonen, who could find no difference in incidence in 10,000 cases examined in Finland between 1910 and 1927; on the basis of a more detailed examination of Ask's figures he doubts their validity. Holm, reporting on Denmark, agrees with Heinonen: comparing data collected by Tscherning in 1881 with the data for 1924, he could not find any difference in the incidence of myopia. A decrease in the incidence of myopia in Portugal is ascribed by Moutinho and Mattos to general hygienic improvement. It should be noted that the problem of school hygiene and elimination of excessive close work in its bearing on the causation of myopia is quite distinct from that of myope classes for progressive myopia. Myope schools, as Harman, who is largely responsible for them, points out, are not based on any particular theory of myopia, but are intended for the child handicapped by defective vision. The evidence brought forward by Harman and others as to their beneficial influence on the course of progressive myopia is concerned with a distinctly different problem—the influence of too much close work on existing myopia.

THE GROWING EYE

The eye of the newborn infant is generally said to have an axial length of 16 millimetres; by the age of 8 the eye is held to have grown to its adult dimensions (Weiss). These are anatomical observations not based on any large series. The validity of these figures apart, it is a common experience that the eye grows during childhood. If one assumes that the anterior segment of the eye undergoes no change in its refractive power during this process, it is clear that the elongation of the eye by 8 millimetres must be accompanied by vast changes in its refraction—somewhere between 30 and 40 or more dioptries. This is hardly the case. While no unquestioned mean of refraction has been established for newborn infants, the available evidence all goes to show that the

* Read in opening a discussion in the Section of Ophthalmology at the Annual Meeting of the British Medical Association, Dublin, 1933.

majority have hypermetropia of about 3 dioptres (Herrnheiser, Schleich, Horstmann, de Vries, Dunstan). Presumably, therefore, changes of no mean order must occur in the anterior segment if, in spite of the elongation of the eye, no radical change takes place in its refractive power. According to Holm the cornea gains 4 D in refractive power during childhood. Priestley Smith found the same diameter for the cornea during the ages 5 to 9 and 10 to 19, and but a slight variation later on; while in the more recent work of Kaiser the cornea was found to reach its full diameter by the age of 6.

Rates of increase in refraction during childhood have been published by different observers. Whatever statistical merit they possess, they tend to mask the fact that all eyes do not behave alike. Not all hypermetropic eyes in childhood show increase of refraction: in fact, about 76 per cent. are stationary, according to the findings of the Special Vision Committee of the Board of Education, and 66 per cent. according to other investigations carried out over a longer period of years (Sorsby). Nor do all myopic eyes exhibit progression; 35 per cent. are stationary, 15 per cent. show slight progression, and 50 per cent. marked progression. Furthermore, as Dunstan points out, large series of investigations demonstrate that in a certain number of cases there is an increase in hypermetropia; and there is no reason to assume that these are errors of observation. Dunstan puts it: "We may classify early hypermetropes from the growth standpoint into 5 per cent. showing increase, 70 per cent. remaining stationary, and the remainder undergoing reductions—these figures having reference to the school period only." According to Tron, great variations occur in the power of the components of total refraction in emmetropia in the adult. He gives these figures:

Refraction of cornea ...	...	...	37.00-48.14 D
" " lens ...	...	...	20.19-34.09 D
" " eye ...	...	...	57.47-72.10 D
Axial length ...	...	...	20.46-25.46 D

The above figures raise the questions as to whether similarly wide excursions are to be found in the growing eye, and as to what exactly are the changes that take place in these different components during the process of growth. Obviously there must be some correlation in these changes if an increase of 8 millimetres in the axial length of the eye is not to lead to myopia of high degree in every case. The problem of the growing eye, with its relatively little disturbance in the total refraction, resolves itself into: (1) What is the value of each of the various components of the total refraction at birth? Do they show the same wide excursions as are seen in the adult? (2) What changes take place in each of these components during the growth of the eye? (3) What is the mechanism which correlates these changes in such a manner that the total refraction remains relatively undisturbed?

If one assumes that the eye has ceased to grow by the age of 8, then the solution of these problems hardly solves the problem of myopia, for myopia begins to manifest itself after this period. In such a view myopia becomes an uncontrolled continuation of the growth process, and until it is definitely known to the contrary one can hardly be justified in the assumption that the aberration is confined to the axial length. Obviously, an increase in refraction in the cornea and lens and a decrease in the depth of the anterior chamber may each produce myopia, while still remaining within the limit of normal variation, and while the axial length has not increased.

THE MYOPIC EYE

If myopia is to be regarded merely as an upset in the correlation of the various components of the total refraction of the eye, it should become manifest during the

period of the growth of the eye—that is, before the age of 8 and not, as is common experience, after this age. Moreover, the myopic eye is much more actively progressive in its refraction than the emmetropic or hypermetropic eye. A real difference in behaviour is operative here, even though, as has been shown, a large percentage of myopic eyes are stationary in childhood, and a not inconsiderable percentage of hypermetropic eyes show the same rapid rate of progression as the progressively myopic eye. It may well be, as has been argued elsewhere, that myopia represents a process of rapid increase in refraction supervening on the normal adjustment which aims at the theoretically emmetropic eye. The reason why progression is seen in myopia is not that it produces this progression, but that progression leads to a myopic eye, as any but markedly hypermetropic eyes soon become myopic as a result of this process. "Myopia no more produces progression than a glaucomatous cup produces raised intra-ocular tension" (Sorsby).

It is usual to assume that the source of increase in refraction is axial elongation, a view which can be substantiated anatomically in the case of high myopia, and is held to be supported in the case of low myopia by the presence of fundus changes. That the anterior segment of the eye has nothing to do with this increase of refraction is held to be proved by the fact that in the majority the corneal curvature falls within normal limits. But since normal limits show an excursion of about 20 D it would be of greater value to know any changes that take place in the cornea and lens during the time the myopic process is active. It must be recognized that many cases of myopia do not show any fundus changes; in fact, they are the exception rather than the rule in myopia under 3 D in adults. Furthermore, there is the contention, not altogether devoid of proof, that the more marked crescents and staphylomata at the posterior pole are congenital defects (von Szilly), and if, as Vogt says, they are regarded as abiotrophic manifestations, their absence in childhood and appearance later on does not invalidate their being considered as congenital defects. Be this as it may, it is clear that much is to be gained by analysis of the factors operative during myopic progression. Knowledge of changes in the refractive value of the various components of total refraction is as urgently needed in progressive myopia as it is in the process of growth of the eye.

VARIABILITY CURVE OF REFRACTION

The work of Steiger has emphasized the unity of refractive errors. Instead of regarding myopia as a distinct process, Steiger considered it as a physiological variation on a curve of frequency which could be constructed around a means. Several such curves have been constructed, and they show a peak consisting of refraction approaching emmetropia, and a dwindling frequency of errors the greater the deviation from this peak-refraction (Fig. 1). In the original view of Steiger it was held that myopia is simply the antithesis of hypermetropia, and that individuals are myopic or hypermetropic by the same chance variations as people are tall or short. Steiger held that the corneal refraction and axial length are disjointed, freely variable components of total refraction, that these components are determined by heredity, and that the chance union of these freely variable factors would produce the whole gamut of refractive errors. In support of such a view is, of course, the fact that an axial length which would produce emmetropia with a cornea within normal range of corneal refractions would produce myopia or hypermetropia with a cornea of different refractive power, but still within the normal range. To Steiger myopia was therefore a physiological variation. Steiger's view in its original form is hardly tenable. Quite apart from a host of clinical observations which prevent one from regarding

at least some myopic eyes as healthy eyes, and apart from the fact that during the active phase the myopic eye is of a different behaviour from the non-myopic, difficulties arise in the statistical interpretation of curves of frequency. There is also the fact that Steiger did not appreciate that the lens too, like the cornea and axial length, is not a constant but a variable. If, as he believed, errors of refraction are nothing but variables on a curve of frequency, they should plot out on a theoretically derived binomial curve, constructed on the assumption that the three main components are free variables. Such a curve is rather broad and flat, but the curves of frequency of refractive errors show two important departures from this theoretical curve, one being that there is considerable elongation of the myopic limb of the curve, with no counterpart on the hypermetropic side (Fig. 2), and the other that the peak, showing refractions approaching emmetropia, is distinctly steep (Fig. 1). As regards the elongation of the myopic limb of the curve, it has been shown

Berg has shown that if a curve based not on free variables but on correlated ones is constructed, a curve approaching in form to those actually found for the distribution of refractive errors is obtained. This view of a process of correlation is in reality a more emphatic statement of the older conception of emmetropization (Straub), which explained the great prevalence of refractions approaching emmetropia by a regulating process that aims to produce theoretically emmetropic eyes in every child.

In considering curves of frequency of refraction and their relation to binomial curves, enough attention does not appear to have been given to curves of frequency in childhood. Obviously, if myopia is a pathological process which disturbs the symmetry of the curve, symmetry should be seen in a curve before the myopic process becomes effective in late childhood. Likewise, if post-

FIG. 1.—Distribution of refractive errors seen in 2,920 secondary school pupils. Negative values = hypermetropia; positive values = myopia in dioptres. Continuous curve = actual distribution; broken curve = theoretical binomial curve (Wibaut).

that a much more symmetrical curve is obtained if myopic eyes showing crescents and other fundus changes are excluded from it (Scheerer and Betsch), or, alternatively, if eyes with myopia of over 6 D are excluded (Tron). On the basis of this it is being argued that there are two types of myopia—one a physiological variation, the other a pathological process. This argument is of significance not so much in stressing pathological myopia as in its insistence on a physiological type of myopia.

Attempts to explain the peak towards emmetropia have been fruitful in bringing forward the illuminating conception of correlation. Obviously the different components of the total refraction are not free variables, but some process of correlation must take place to bring about the excess of refractions approaching emmetropia. Wibaut, Tron, and Berg have contributed to this aspect, and various positive correlations have been suggested; indeed, Wibaut has gone as far as to suggest that the correlations in the child's eye are different from those in the adult.

FIG. 2.—Base of a refraction curve showing elongation of myopic limb and excessive incidence of myopia. Refraction indicated in dioptres of corresponding value. The broken line shows the theoretically derived binomial curve (Scheerer and Betsch).

FIG. 3.—Distribution of the refractive errors under atropine cycloplegia in a composite series of 2,398 newborn infants. Explanation as in Fig. 1 (Wibaut).

natal correlation is a process of emmetropization, the curve in newborn infants should be more truly binomial. As regards a curve of frequency in the newborn infant, Wibaut has constructed one on the rather conflicting and mainly German data of four different observers, and finds that it does indeed assume a more typically binomial form (Fig. 3). Incidentally the peak of the refraction is 2 D of hypermetropia. For children in London of the ages 3 to 8 years, a curve of frequency will be published later in the year; the peak in that series is refraction between 2.25 and 3 D. As things stand at present Steiger's work is of significance, not so much because of his views on the binomial distribution of refractive errors, but because of the attention it focused on the two disturbing factors which prevent the acceptance of his view. The excess of myopes with eyes showing myopic fundus changes, and the excess of refractions approaching emmetropia, are problems which respectively go down to the very fundamentals of pathology and physiology of refractive conditions.

HEREDITY IN RELATION TO REFRACTIVE ERRORS

That heredity plays a part in refractive errors can hardly be doubted. Not only is there the evidence of many published pedigrees, but every clinician is constantly seeing it for himself. But while heredity has previously been considered as one of the factors concerned in the development of myopia, there is a modern school which regards it as the sole factor, to the exclusion of all environmental considerations. In Steiger's view the components of refraction were hereditarily determined, free variation taking place within these limits. Extreme variations would have little chance of survival under natural conditions, and it was on this basis that he explained the greater incidence of marked refractive errors in civilized communities and in domestic animals. Racial differences were likewise to be explained, on the basis of differing environmental conditions favouring, or at least not hindering, the transmission of variations. In this unmodified form Steiger's view leaves too many questions unanswered.

The view that the myopic eye is caused by excessive elongation of the sclerotic and is a physical condition transmitted from generation to generation, much in the same way as the tallness is transmitted, hardly solves the problem. Apart from the fact that the myopic eye shows a distinct behaviour of its own during the process of development, there is the anatomical evidence that the sclerotic is thinner posteriorly. This is not exactly normal growth. In Vogt's view it is the retina which determines the length of the eye. He has shown that the thinning in the posterior pole in myopia does not affect the retina; the sclerotic adapts itself to the size of the retina and becomes thinned when the retina is too large. Experimentally, damage to the posterior pole of the retina will stop the axial growth of the eye: the retina is a sort of master-tissue controlling the sclerotic, and myopia is a hereditary condition depending upon the inheritance of large retinæ. Vogt further holds that the fundus changes in myopia are hereditarily determined characteristics. He regards them as an abiotrophic manifestation, on the ground that they show considerable similarity to senile degeneration around the disk and macula.

There are thus authorities who would deny any dualistic conception of the aetiology of myopia. To them environment is of no consequence in the production of refractive error: they view the whole process as an expression of predetermined inheritable characteristics. Low myopia is to them a physiological variation in a curve of frequency; the components of total refraction are hereditary characters, and this type of myopia is not necessarily due to axial elongation. High myopia, the so-called pathological type, is likewise a hereditary condition, and in the vast majority of cases is due to a long axis consequent on an inherited posterior staphyloma, which is either manifest at birth or becomes manifest later on. Those who adopt this extreme point of view see, of course, no therapeutic value in myope classes, though they would not deny their educational and general hygienic value.

CONTROL OF SCHOOL MYOPIA: CONCLUSIONS

Though the strictly rational control of school myopia is not yet, it is surely premature to assume an attitude of fatalism. The monistic view of the causation of myopia held by those who see myopia as a hereditary abnormality totally uninfluenced by extraneous conditions is hardly warranted by fact. If the eye is indeed fully grown by the age of 8, and for all practical purposes the variables entering into its total refraction are fully correlated and consolidated, then myopia is a process which is outside physiological variation, and such a thing as physiological myopia, which this school presupposes in the case of low myopia, is non-existent. Far too many problems press for solution be-

fore the theory that heredity is the basis of all myopia can be accepted. For one thing it is of paramount importance to know whether school myopia really develops in an eye that has completed its normal development, or whether it is an exaggerated and excessive final adjustment in the various components of refraction. In other words, when does the eye normally reach its full development? Other problems have been indicated before. It is imperative to know the range of values of the components of total refraction in infancy and childhood, and to understand the process of correlation of these changing values. More attention, too, is needed for those cases known as congenital myopia. In some of these the myopia is obviously part of widespread deformation of the eye, but in others no obvious deformity is seen. Are these cases an early failure in correlation? Are they cases of anterior segment or index myopia, or cases of myopia due to a congenital or congenitally determined long antero-posterior diameter? More, too, should be known of their behaviour so far as progression is concerned.

Galen and the copyists who followed him recognized myopia as the process in which distant objects are not seen clearly, but near objects are. They further held that this defect is due to weakness of the visual spirits secreted by the brain and supplied to the eye. If the definition is still true, we have, at any rate, passed beyond the explanation; we have learnt to recognize more than one cause and more than one variety of myopia. We appreciate the existence of a symptomatic myopia such as is seen in diabetes and trauma, and a secondary myopia as in buphthalmos and staphylomata of congenital syphilis. The main mass of myopia is, however, still eluding us, largely because our attention has been focused on the abnormalities of refraction rather than on the normal development which precedes them. When we have learnt the physiology of the growing eye we may be able "to look into the seeds of time and say which will grow and which will not." For the present it is enough to realize that all theories of myopia have led to a blind alley. If the optimism of those who regarded myopia as a product of controllable environmental conditions has not proved altogether justified, neither is there any reason for believing that the ultra-modern fatalism will prove nearer the truth. For the present, while recognizing the problems and their formidable nature, one cannot go far wrong by paying attention to measures of general and ocular hygiene in our treatment of the low myope, and by giving special considerations, such as are supplied by the myope classes, to our more advanced myopes.

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