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THE PROBLEM OF ASTHENOPIA*

BY

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IN July, 1938, I received a letter which gave me very great pleasure. It was from your House Governor, and it said that the Selection Committee had invited me to give the Middlemore Lecture for 1939. This lecture was postponed owing to the war, and perhaps this is just as well, because 1939 was the year of its jubilee, and I should have felt even more diffident about giving it then than I do at the present moment, though I could not have felt more pleased at the honour you have accorded me. In a way the Middlemore Lecture and I have grown up together because the year 1889, which witnessed its birth, is the same as that which witnessed mine.

The title of the first lecture was "The Essentials of Ophthalmic Therapeutics" and I hope that if your first lecturer, Mr. Lloyd Owen, were here now he would agree that his title might be applied to the subject we are dealing with to-day. I also hope that it would have appealed to the founder of this lectureship, who was associated with the hospital from 1828, when he was appointed assistant surgeon, until 1849, when he retired to the consulting

* The Richard Middlemore Lecture, May 7, 1940

staff. Perhaps he would be a little jealous of me because he endeavoured to start a journal of ophthalmology, but failed, owing to lack of enthusiasm among ophthalmic practitioners, whereas I have been fortunate enough to have a hand in the editing of one which was already a going concern before I came to it.

In the foundation deed of this lecture it is set out that the subject shall be connected with ophthalmic science and practice and that the lecture shall be open to all medical practitioners. I took this to mean that too technical a dissertation would not be welcomed, and some of you may wonder why I chose the subject of asthenopia, since at first sight this would appear to be a matter in which only ophthalmologists could be interested. Such is not the case, however, because although something like 80 per cent. of an eye man's work is concerned with the treatment of this complaint, it is so widespread over the population that it behoves those who are not skilled refractionists to know something about it too.

A fair, but I hope diminishing proportion of patients, when they feel their eyes ache, rush straight off to a sight testing optician, but the wiser ones will consult their doctors and be referred by them to an ophthalmologist, should they deem it necessary. It is incumbent therefore on all classes of medical men to know some of the points connected with this subject.

Although the term asthenopia, literally translated, means weak-eyedness, it has been adopted by ophthalmologists as synonymous with the word eyestrain, or to put it more exactly, since we are not sure that the eyes really *are* strained, asthenopia is a word which covers the multitude of symptoms which occur in certain persons when they use their eyes.

The exact pathological basis of these symptoms is unknown, and Parsons' dictum that "the rationale of visual fatigue in the production of ocular and systematic diseases is still a matter of conjecture" stands true. It is, however, a challenge, as are many of his provocative statements, but it was because I believe it to be true that I incorporated the word "problem" in the title of this lecture. I was tempted to preface it by the adjective "unsolved," but I felt that this was adopting rather a pessimistic attitude and perhaps putting the cart before the horse, because although you may feel at the end of this lecture that the problem is no nearer solution than it was at the beginning, it is a pity to start with that idea in our minds. The commonest manifestation of asthenopia is headache, and there seem to be two schools of thought on this subject. According to one, alluded to by Weeks of New York,³ it arises from dysfunction of the ciliary muscle, the pain being referred along the branches of the Vth nerve, in the same sort of way as angina pectoris arises from cardiac dysfunction. Michaelson is quoted as having shown that ciliary

muscle contraction, relaxation and fatigue could produce pains of varying characters, such as throbbing, dull aching, sharp and knife-like, a heavy tired, or a tight gripping feeling. On the other hand, the sympathetic nervous system may be responsible since the network of vessels in and around the eye is accompanied by delicate fibrils from this system which control vascular tone. Some of these vegetative fibres have end organs of sensation which are activated by products of fatigue or of perverted metabolism and so are responsible for producing headache when the eyes are working under unfavourable conditions.

The other school would seek to place more emphasis on changes occurring within the central nervous system itself. It has been shown,⁴ as the result of intracranial operations under local anaesthesia that stimulation of the upper surface of the lateral blood sinus of the brain produces pain at the back of the ipsilateral eye, and that in general large cerebral veins which cross the subdural space to empty into the sinuses, produce exquisite pain when traction is exerted upon them. Also although the dura is generally insensitive, it is not so at certain points where branches of the middle meningeal artery are found. The brain is therefore somewhat sensitive to circulatory changes, and as shown by experimental injections of histamine, headache can be produced by such changes. It is particularly marked when the vessels are pulsating strongly and the cerebro-spinal fluid pressure has begun to fall. The application of this to the production of an ocular headache is not so obvious as the reasoning of the first school of thought, but it is conceivable that the fatigue engendered by use of the eyes under conditions of strain might bring about vaso-motor changes in the brain of a less marked but similar character to those produced by histamine.

If we do not know for certain the exact lesion which is responsible for producing the headache of asthenopia, we can at any rate attempt to classify the causes of this condition, and the first main group is errors of refraction. I do not propose to go seriatim through the signs and symptoms associated with hypermetropia, astigmatism, and myopia, because these are already adequately described in current text books. It is rather with the problematical aspects of refractive asthenopia which are touched on lightly, if at all, that I would deal.

Cure of eyestrain by a suitable pair of glasses seems a straightforward affair, but even in this there are some interesting observations to be made. It is not uncommon, for example, when seeing a middle-aged woman for a presbyopic correction to be told by her that she has not worn glasses since childhood. She had them then for astigmatism, but the glasses "cured" this in a year or so and were then discarded. It seems an unlikely story, and when

one comes to examine the patient, one finds she still has some astigmatism, not very different perhaps from that which afflicted her in childhood, and yet over a period of years it has caused her no trouble. There are various possible reasons for this, one of which I shall give you later on in this lecture. If one considers the question from the point of view of the refraction, however, an adequate explanation would seem to be that a child at the age of 10 or 12 is beginning to do a good deal of reading, but is not yet really familiar with print and has to look separately at almost every letter. It is very much the same sort of thing as though an adult had suddenly to take up reading say Greek characters with which he was not familiar. Under such conditions the additional strain imposed by a small uncorrected error of refraction is sufficient to cause symptoms. After a year or two, however, print becomes only too lamentably familiar and the patient is able to take in whole words almost at a glance. Presumably the same degree of accurate focusing is not required and so the glasses can be discarded without recurrence of the symptoms. This explanation, which might be dubbed the last straw and the camel's back philosophy, may also be invoked to explain the temporary need for glasses which sometimes occurs at puberty and during periods of rapid growth.

The importance of recognising this type of asthenopia is threefold. In the first place one is able to reassure the anxious parent who always asks the same question, namely, "Oh, but if you give her glasses now, won't she always need them?" Secondly it enables one to give the child a very real measure of relief at what may be a critical time in its life. And thirdly, since most of these errors are trifling in amount, it emphasises the necessity for an accurate correction. It is for this reason that I always prefer to see these children under atropine before ordering glasses. Occasionally one gets surprises and finds two or more dioptries of unsuspected hypermetropia, but even this is an amount which an average child should be able to cope with without any trouble, and usually can, once familiarity with print has been established.

Other conditions besides rapid growth and lack of familiarity with print can bring about dependence on glasses, one of the most notable being convergent squint.

A particularly striking case was that of a girl aged 15 years, with a convergent squint of 28° . Before operation her refraction under atropine was approximately +2.0 D.Sph. in each eye and she seemed quite comfortable wearing +1.75 D.Sph., *i.e.* only 0.25 D. less than her full atropine correction, whereas without the glasses she promptly got symptoms of eyestrain. After operation her eyes were straight, and after going through a short period of reduction in strength of her glasses, Miss L. gave them up

altogether and reads and does everything quite happily without them, although strenuous and prolonged efforts at fusion training both before and after operation have failed to produce binocular vision.

It came as rather a surprise to me to discover that the same thing can occur in older people. A certain Miss S. came to me a few years ago to know if anything further could be done for her. She had had an unsuccessful operation for convergent squint in childhood, and when I saw her the right eye was 25° convergent and 15° up; her age at that time being 32, and her refraction roughly 4 D.cyl. in each eye. I resected 12 mm. of the right external rectus and 9 mm. of the right inferior rectus.

Fortunately this rather complicated operation went well and the ultimate result was to give her a straight pair of eyes. A month or two afterwards, she told me that she had been able to give up wearing her glasses constantly and used them only for reading or when she wanted to see anything particularly clearly.

The explanation of these two cases must I think be psychological, because in each the squint was almost as obvious with as without glasses and in neither was binocular vision obtained. My own feeling when I first needed a correction for my hypermetropia was that glasses so to speak shielded me from the outside world. It was rather like sitting in a railway carriage and looking out of the window. One felt a spectator of life rather than a participant in it, and I imagine that something of the same sort must have affected these two patients. With their glasses on, they were shielded from the prying eyes of an inquisitive world and so they felt that their deformity was less noticeable, but there was possibly another factor too. The feeling of inferiority engendered by an obvious squint may make demands on the nervous system which leaves it unable to cope with the additional strain imposed by an uncorrected error of refraction, and hence asthenopia developed when glasses were not worn.

The subject of relief of symptoms by correction of errors of refraction is of course an enormous one, and in fact in 1910 was chosen by Sydney Stephenson for one of these lectures.¹ At that time, more attention was paid to this subject by the profession as a whole than is the case to-day, and although exaggerated claims were made for the effects of correction of small errors of refraction, my feeling is that nowadays the pendulum has swung too far the other way, and that we are in danger of omitting to give certain types of people the benefit they might have from accurate correction of their refraction.

We all know that with modern methods, there is only about one person in two hundred who has a pair of emmetropic eyes.

We also know from the writings of the advocates of glasses that it was the small errors, *i.e.*, those which were present in nearly everybody, which caused the symptoms, particularly when the error was hypermetropic astigmatism with oblique axes to the cylinders. The late Dr. Leonard Williams in his admirable book "Minor Maladies"² draws attention to the fact that small astigmatisms "which the patient himself, by contracting his ciliary muscle, can adequately correct," bring about "a ceaseless and illegitimate expenditure of nervous energy" and states that this may be a predominant factor in producing nervous dyspepsia. In a characteristically amusing and racy passage this author tells how "the unfortunate patient, whose only need is a correction for his astigmatism, has his fare pared and whittled both in quantity and quality until that of Nebuchadnezzar may seem generous in comparison. Not only is his food treated in this manner, but his stomach is now soothed with papaveric caresses, and anon chastised with Chilian scorpions in the vain hope that it may be induced to make bricks without straw." You may smile as I did, when I read this passage, but practical experience made me realise that there was something in it. Whether it was the magnetic personality of Leonard Williams I do not know, but the few cases of his which I was privileged to have referred to me, with instructions to work out their refraction under a mydriatic, did remarkably well. One for example, of chronic dyspepsia, lost her symptoms and gained two stone in weight in a year as a result of constant wearing of a small correction for astigmatism. That a case of lumbago was also cured by the same method may seem to bring discredit on this form of treatment, but I do think that it is worth while to remember that in cases of obstinate dyspepsia it may be advisable to investigate the patient's refraction, and if an error is found, to try the effect of wearing a correction constantly. Why the method should work is I admit a problem, but it is also in some cases a fact. I imagine that in the same way as some people are hypersensitive to, say pollen, so are others hypersensitive to the effort required to overcome a small refractive error. It is hardly necessary to emphasise that it is usually the small errors which cause symptoms, because it is just these which the ciliary muscle makes efforts to overcome, larger errors being left alone and the sight of the eye allowed to be blurred.

Heterophoria or muscular imbalance of the ordinary type is well known as a cause of asthenopia and need not detain us. Its presence is made known by the Maddox rod and Wing tests, which are now used as a routine, and the results obtained by its intelligent correction are sufficiently satisfactory to need no more than passing mention in a paper dealing with the problematical aspects of eye strain. The reverse is the case in another aspect of extra-ocular muscle physiology.

As long ago as 1931, Stutterheim of South Africa wrote a monograph supplement to the *Brit. Jl. of Ophthal.* on the kinetic treatment of the eyes. In it he gave a striking series of cases in which asthenopia had been cured by teaching patients to overcome the diplopia produced by placing prisms base out before the eyes. The process was long and tedious and required a daily attendance of ten minutes over a period of four or five weeks. The idea was interesting, however, and seemed to merit investigation, so I began to work at it, and found out shortly afterwards that Ida Mann was on the same track. Some of you will remember her paper on the subject at a meeting of the British Medical Association. It has been published in this Journal (after delivery of this lecture), and though I do not wish to poach on it in any way, her findings coincide closely with those I reported a year or two ago to a meeting of the Ophthalmic Club. The idea behind what I think should be called Stutterheim's treatment is that convergence is phylogenetically one of the most recently acquired functions of the eyes, and that voluntary convergence, *i.e.*, the power to produce diplopia by voluntary convergence of the visual axes is a further development of this function. Curiously enough, Stutterheim designates the condition which his exercises are designed to improve as "involuntary convergence," whereas, as you will shortly see, it is the ability to perform the act voluntarily which is of real importance.

One example quoted by Stutterheim will be sufficient to show that the power of convergence is a relatively late phylogenetic acquisition. A horse has a certain amount of binocular vision, and although the eyes stand widely apart at the sides of its head, yet, if one approaches the animal with a piece of bread, it looks at it binocularly up to $1\frac{1}{2}$ to 1 metre in front of the tip of the nose. If one brings the bread nearer, the horse turns the side of its head towards the food and looks at it from now on, with one eye only, *i.e.*, the animal, although able to bring its visual axes into parallelism, cannot converge them. To return now to *homo sapiens*, here are some of the methods of treatment we have employed. If the services of an orthoptic trainer are available, the following scheme, which has been elaborated in conjunction with Miss Sheila Mayou, has been found to give good results.

In the consulting room the usual tests are made for phorias, which in the majority of cases are insignificant in amount. A vertical white strip—I generally use a folded Selvyt cloth—is then pinned on to a Bjerrum screen and a trial frame containing rotary prisms is placed on the patient's face. The amount of prism base out is steadily increased until diplopia is produced when a reading is taken. Miss Mayou then carried on as follows:

First lesson.—The patient is put on to the synoptophore which

contains two simple fusion pictures with a conspicuous lock and the arms are approximated until diplopia occurs. The process is repeated several times, in the endeavour to work up the convergence power. It is often of assistance to put in concave lenses, which make the patient accommodate and thereby increase his convergence. He is then taught to appreciate physiological diplopia by fixing on his finger and seeing an object at the end of the room double. Some patients seem to have great difficulty in doing this, and when this is so, I usually cover one eye with a red glass, switch on the Maddox rod light, and direct them to look at their finger. Two lights are then produced, one red and one white, and the diplopia is perceived. On removing the red glass, the diplopia is usually still evident. The patient is then sent home for a week and told to practise the production of physiological diplopia by fixing on his finger.

Second lesson.—The patient is again put on to the synoptophore with fusion pictures, and is encouraged to work up his convergence without the assistance of concave lenses. Some patients do better if allowed to move the arms of the machine themselves, others if the arms are moved for them by the orthoptic trainer. An endeavour is then made to get the patient to see a distant object double without using his finger for a fixation point. The best way of doing this, in my experience, is to allow the patient to fix on his finger, and then take the finger away and attempt to keep looking at the point in space which it occupied. At first the diplopia is only momentary, but if this only can be obtained it is enough for the patient to start practising on when at home, and usually after a week he is able to double distant objects by a voluntary act of convergence without the assistance of a near object for fixation. It is often helpful if two similar distant objects are used, *e.g.*, a couple of candles, and the patient tries to fuse them by convergence. This also helps to teach him to relax his accommodation and so produce a clear image of the two fused candles.

Third lesson.—The patient is introduced to “jump convergence.” In this, with the locking pictures, the arms of the synoptophore instead of being steadily approximated are pushed in suddenly about 15° , then 30° , then back to 10° . This of course produces diplopia at first, but the patient soon learns to overcome this. Then non-locking slides can also be used. For homework at this stage it is often useful to lend the patient an amblyoscope for working up his convergence and practising jump convergence. A diploscope may also be of assistance in helping him to steady the convergence and learning to hold it. Simultaneous perception slides are then put into the synoptophore, *e.g.*, the bird and cage, and the patient is directed to keep the bird in the cage while the

arms of the instrument are approximated. This is not usually possible until the ordinary fusion pictures, with a lock, can be kept single up to an angle of 40° convergence. It is well therefore to test this power first and work it up if necessary. Having obtained some power of convergence with the non-locking pictures, the next step is to go on to jump convergence with pictures of this type.

The *fourth* and *fifth* lessons are really amplifications of the third, in which efforts are made to increase the patient's power of voluntary convergence still further and to steady it until he can superimpose two dissimilar pictures when the arms of the synoptophore are 40° convergent. Voluntary convergence off the machine should also be practised, the patient having to get a wide separation between the two images and to hold his eyes steadily converged for a few seconds. Homework and active co-operation by the patient are all important and at this stage it may be helpful to supply him with some of Hamblin's stereoscope pictures which he attempts to fuse by convergence without a stereoscope. The simple words series is a good one because these have a conspicuous lock and the distance between the pictures is graded (Fig. 1). At first the patient will probably have to fix his gaze on a pencil held between him and the picture. When he does this, the latter is doubled and, taking picture No. 1 (Fig. 2) as an example, he will see four ones instead of two. By moving the pencil towards him or away from him, he can alter the positions of the ones until the middle two fuse (Fig. 3). He will then find that he has also produced a complete dog, and that the two O's are superimposed to form the word "dog." On removing

FIG. 1.

No. 1 of the Simple Words series of fusion pictures. (Hamblin).

FIG. 2.

Appearance when the two halves of the drawing have been partially approximated.

FIG. 3.

Appearance when the two halves of the drawing have been fused.

the pencil, he may find it is possible to hold the pictures fused and by practice he will learn to fuse them by an act of voluntary convergence without the aid of a pencil. The only danger with the pictures is that the patient may unwittingly fuse them by a relative divergence, but this is guarded against by starting with fixation of the pencil.

Ordinary stereoscopic photographs are not of much value, because if fused by convergence an inverse stereoscopic effect is

produced, the photographs being arranged for fusion by divergence. They can, however, be cut down the middle and pasted on to a card so that the right hand photograph is on the left and the left on the right, and then fusion by convergence will produce stereopsis.

I hope you will not think I have been too diffuse in describing the methods of training we have employed, but my excuse must be first that I wanted to prove that the convergence elicited is essentially voluntary and not involuntary in character, and secondly that I thought you might be interested to know exactly what is being done.

The job to my mind is essentially one for the orthoptic trainer rather than the ophthalmic surgeon, if only from the economic standpoint.

Turning now to consider the symptoms of asthenovergence, which is Stutterheim's name for inability to overcome 50 or more dioptries of prism base out, the most characteristic single one is I think, difficulty in following moving objects, with which of course is allied, car sickness. I have had several patients in whom this latter symptom entirely disappeared when the voluntary convergence was brought up to 80 prism dioptries. The same is unfortunately not true of sea sickness. I am only a moderate sailor myself, and yet I can go up to the limit of voluntary convergence easily. Quite a number of these asthenovergic patients complain of headache after cinemas, a symptom which would seem to be allied to the difficulty of following moving objects.

Other symptoms are really those with which we are already familiar under the heading of asthenopia, and include headache, difficulty with close work, feelings of strain, and burning in the eyes, and rather characteristic of asthenovergence, aching in only one eye in attempting close work, all these of course in the absence of any notable error of refraction or of muscle balance or in spite of wearing an adequate correction.

Coming now to consider results I feel rather shy of giving these to you because the percentage of success seems so unwarrantably high. We have in all, the records of 117 patients who have had a course of treatment, as outlined above, and of these no fewer than 89 were cured, *i.e.*, relieved of the symptoms of which they complained. Of the remaining 28, 15 reported themselves as improved and 9 were complete failures. Four did not attend for a complete course. Two of these were definitely psychological cases and lost their symptoms when the causative conditions were discovered and put right, while three others were just failures for no reason that we could find out. The sixth case was a confirmed neurotic who was never happy unless she had something wrong with her. As a result of Stutterheim treatment she was, however, able to read

for a slightly longer time, but at the end of 45 minutes she complained of feeling pricks at the back of her eyes.

Another and unexpected result has been produced in cases of anisometropia. Miss Mayou tells me that we have had through the hospital quite a number of anisometropes wearing -4 or -3.50 in one eye and a $+$ sphere or plane glass in the other eye. They are sent to the orthoptic department because they complain of headaches after wearing their glasses, but after five lessons in training their voluntary and involuntary convergence, their headaches disappear. They find, however, that they can no longer read with one eye as they used to, but have to hold their heads straight and use both eyes together.

It may be argued that the successful cases were functional and improved because of the enthusiasm and confidence with which they were treated. This may be so in some, but I don't think it can be true of all, because so many seem to follow a similar course. They are usually made worse by the first two lessons (and it is as well to warn them of this) and then when they have learnt the trick of voluntary convergence they go straight ahead and rapidly lose their symptoms. In contradistinction to this, the patients who were trained before the latter half of 1932 found that their symptoms returned after about a year. At this time we were more under the influence of Stutterheim's paper and had not discovered the importance of training the voluntary convergence and when the patients returned we found that they had lost their power of overcoming prisms base out. They were therefore re-trained and made to acquire the trick of voluntary convergence, after which there was no relapse.

It is only fair to state that the cases are to a certain extent picked, *i.e.*, if they show no interest in the work, will not practise at home, and do not co-operate in the training, this is discontinued. Such cases have not been included in the 117 I have mentioned.

In connection with this form of treatment there are three points I should like to make.

(1) That credit be given to Stutterheim for his important pioneer work in this field. Without this no one would have known anything about it and all that we have done is to elaborate his technique with the object of diminishing the number of visits and preventing recurrences by training the voluntary convergence up to 40° , *i.e.*, 80 prism dioptries.

(2) That this method of treatment is of definite value in cases of obstinate asthenopia.

(3) That in some cases it enables patients to discard their glasses, and this, in an age when everyone is clamouring for exercises instead of glasses, may be of value.

(4) A further interesting point is that a certain number of patients who have had this treatment find that their visual acuity is improved. The improvement is not usually very striking, but results such as 6/9 becoming 6/6 or 6/5 are not uncommon, and have been achieved several times. Group Captain Livingston tells me that he has found the same thing in training men in the R.A.F. and I remember Basil Lang saying something of the same kind too.

Whether any other kind of visual training would produce the same effect I do not know, but the fact of its occurrence may give a clue to the occasionally successful results which patients claim from the effects of eye exercises by some of the notorious quacks. Please do not feel that I propose entering into competition with these gentlemen, but there is no harm in learning anything they may have to teach us. Ida Mann has put the matter into a nutshell by suggesting that what we are doing in this form of treatment is to bring about the permeability of the path from the frontal cortex to the oculomotor centres by facilitation, using conscious attention as the stimulus and the fixation reflex as the source of the impulse.

Another side of extra-ocular muscle pathology was investigated a few years ago by Marlow of Syracuse. It occurred to this observer that although the eyes might appear to be in perfect muscular equilibrium when tested by the ordinary methods such might not be the case. In the same way as an unsuspected hypermetropia may be revealed by the use of atropine, so might an unsuspected error of muscle balance be revealed by keeping the eyes dissociated over a sufficiently long period of time, and I sometimes wonder whether Marlow's work has received adequate recognition, at any rate in this country. In addition to several papers this author has written a book based on his observation of 700 cases,⁵ and it is worth reading by those who are interested in ophthalmic myology. His original idea was to cover one eye continuously for a week except on the occasions when it was being tested with the Maddox rod or with the red and green test. Ideally the covering should be kept up until stability in position of the eye was obtained, as shown by its giving the same reading for error of muscle balance on two successive days. In practice, however, Marlow was of opinion that covering for a week was usually long enough. He advises that when the Maddox rod or red and green tests are being made the patient should wear whatever correction is necessary for his error of refraction. Hughes in a later paper⁶ describes three cases in each of which the covered eye tended to deviate upwards. This is said by Abraham⁸ to be related to Bell's phenomenon, the upturning of the eye on attempted closure of the lid having been first described by him

in connection with peripheral facial palsy, and it struck this observer that the tendency of an eye to deviate upwards when covered might invalidate the results of Marlow's work. In his hands it certainly seemed to do so. In 48 tests in which an eye was occluded for an average period of 23 hours, 37 showed definite hyperphoria. Of 18 tests in which the right eye was covered, all showed a right hyperphoria, while in 19 in which the left eye was covered, all but one showed a left hyperphoria, the exception being a patient who had right hyperphoria before the test, and in whom this became diminished. The amount of the hyperphoria was usually from 1° to 2° , but in some cases it went up to 3° or even 4° . Even more striking were 6 patients in whom first one eye was covered and then the other. All of these developed a right hyperphoria when the right eye was covered, and a left hyperphoria when the left eye was covered. It is also perhaps significant, in view of the tendency for a blind eye to deviate outwards, that by the occlusion test esophoria frequently becomes exophoria, but the reverse situation was never found.

Abraham therefore feels justified in concluding that the occlusion test though admirable for demonstrating the presence of Bell's phenomenon, is not a test for latent hyperphoria, but one wishes that he had maintained the occlusion for a week or more instead of for 23 hours.

In spite of these criticisms, however, Hughes⁶ is of opinion that in certain cases, the prolonged occlusion test is of value in demonstrating a definite pathological condition which is not brought out by the ordinary tests. In some instances the patient wears with comfort a prism which previous to the period of dissociation of the two eyes produced an insurmountable diplopia. He advises that the occlusion should be alternate, one eye being covered one day and its fellow the next, in order to avoid the occurrence of Bell's phenomenon. He also advises a period of 10-11 days of occlusion. I have used the method a few times myself and I can remember at least two cases in which the need for a small vertical prism was discovered and in which relief of asthenopia appeared to follow the use of such a prism. Unfortunately I have not been able to lay my hands on the notes of these cases, so I cannot give you details. Perhaps, therefore, you will excuse me if I give a brief account of one of Hughes' cases, in order to show the sort of thing which happens. The patient was one who complained of ocular discomfort and headaches, particularly in the occipital region. On the first examination 0.50^A of left hyperphoria was found. No appreciable muscle imbalance had been discovered at five previous tests by different oculists, who had all ordered approximately the same sphero-cylindrical correction. After alternating occlusion for a period

of eleven days a maximum hyperphoria of 4^{Δ} developed in the left eye, together with a marked exophoria, the latter not having been present before occlusion. Correcting prisms, equivalent to 1.5^{Δ} base down left eye and 3.0^{Δ} base in were ordered and were worn with complete comfort. A curious feature of the case was that subsequently the patient reverted to his previous esophoria and yet was uncomfortable when the prism base in was removed from his lenses. The practical difficulties in the way of carrying out this test are considerable, because it is not easy to persuade an adult patient in this country to forego the use of one or other eye and the advantages of binocular vision for as long a period as eleven days when there is no apparent disease in either eye, and this is one of the reasons why I have not employed the test lately. Another reason is that I had a patient, a nurse from the other side of the Atlantic, who showed no hyperphoria with the Maddox rod, but was attempting to wear, with considerable discomfort, a vertical prism ordered after a prolonged occlusion test. She was quite comfortable when this prism was removed. A third reason was that the procedure of keeping one eye covered for a long period of time was really unphysiological, and that defects which required such a procedure for their detection could not be of practical importance. The whole rationale of the test seemed to run counter to the ideas underlying orthoptic training and one had the feeling that one was allowing the patient to give in to a defect which he could overcome by properly planned exercises. In addition, one was condemning him to the constant wearing of glasses, whereas by training him along the lines I have already mentioned glasses can in some cases be dispensed with.

I am fully aware that this argument might be applied to the use of glasses for correction of small errors of refraction, but there is a fundamental difference between the two cases. In the case of small degrees of astigmatism we are dealing with strain on the ciliary muscle and there are no exercises I know of which will teach the owner of the muscle to overcome astigmatism by contracting one portion of it while he relaxes another. In the case of the extra-ocular muscles, however, the position is quite different and what may be called "neuro-muscular facilitation" can, as I have shown, be speedily acquired.

Another possible cause of asthenopia is aniseikonia or inequality in size of the ocular images. This is a subject which has been very fully investigated by Ames of the Dartmouth Medical School, and by his co-workers in the United States who have published many papers on it. The fundamental idea is that if the brain has to combine two images of dissimilar size, strain and asthenopia may result. Difference in size of the ocular images may be brought about in two ways. In the first place the retinal image may be

of different size in the two eyes and in the second place, although the retinal images may be equal in size, the arrangement of the retinal mosaic may differ so that a greater number of nerve elements is stimulated in one eye than in the other. Inequality in size of retinal images is due to differences in the dioptric power of the two eyes, or to put it more scientifically to differences in the distance of the nodal point from the retina. An extreme example is seen in cases of unilateral aphakia when the eye which has had the cataract removed and is wearing a suitable correcting glass sees the letters of 6/6 the same size as a normal eye sees 6/9. An extreme example of disturbance of the retinal mosaic is seen in early cases of choroido-retinitis where as a result of oedema, the rods and cones are separated more widely than is the case in the unaffected eye. The result of this is that fewer of them are stimulated and so the ocular image appears to the brain to be smaller. Ames' work is based on very much smaller differences than these and with his apparatus it is possible to detect differences of the order of 0.5 per cent. in all patients, and of 0.25 per cent. in a great many.⁷ As evidence of the importance of disparity in size of retinal images, wearing of a lens before one eye which caused a 1.5 per cent. increase in size produced a marked headache in about an hour. In 16 normal persons, 6 showed no size difference between the two eyes, and the average for the remaining 10 was 0.85 per cent. The size difference found in 96 "clinical cases" averaged 1.54 per cent. The matter is more complicated than this, however, because the distortion may not be a mere overall increase in size.

Fig. 4. This figure, taken from Ames' paper, shows some of these. The diagrams on the left show the normal image, those on the right the distorted ones.

- (a) Represents a simple overall enlargement.
- (b) An enlargement affecting only the vertical meridian.
- (c) One affecting only the horizontal meridian.
- (d) One in which there is progressive enlargement in the horizontal meridian as one passes across the visual field.
- (e) One in which the image increases in all directions from the axes of vision—so-called barrel distortion.
- (f) One in which the converse occurs—so-called pin-cushion distortion.

Optically only the first three of these are susceptible of correction, but enough has been said perhaps to show that the correction of aniseikonia is a matter for the expert, particularly as the instruments employed are complex and expensive.

The general principle of these is to present to the two eyes, looking through a modified stereoscope, two targets such as these

SUPERPOSED OCULAR IMAGES IN THE BRAIN.

FIG. 4.

(After Ames) : for explanation *vide* text.

—Fig. 5. When these are looked at binocularly the eyes fuse the central ring, but there is nothing to make them fuse the dots and the lines. If therefore the right ocular image is larger than

FIG. 5.

(After Ames) : one type of target.

FIG. 6.

(After Ames): appearance produced in stereoscope when the left eye image is larger than the right.

the left, the appearance shown in this diagram will be seen (Fig. 6). If it is smaller, the dots will lie inside the lines. If the enlargement affects only the vertical meridian, the east and west dots will lie in the gap between the two lines, the north and south ones being outside them and so on. Oblique axes of enlargement can of course be detected by rotating the targets. An estimation of the degree of aniseikonia can be obtained by putting up size lenses in front of one or other eye until all the dots lie in the spaces between the lines. Possibly some of you are wondering what size lenses are. These are lenses of zero power which produce enlargement or diminution of the object looked at through them, and the general principle upon which they are constructed can be seen from this diagram (Fig. 7). ABCD is a curved sheet of glass and it will be seen that two parallel rays of light

FIG. 7.

Diagram explaining the optics of "size lenses" (*vide text*).

entering it at E and F are bent inwards by the glass owing to its refractive effect. On emerging at H and K they are bent outwards again so as to become once more parallel, but they are now closer together. An observer therefore who looked through this sheet of glass from the concave side would see objects smaller than they really are, while if he turned it round and looked

through the convex side, they would appear larger. It is obvious that different effects can be produced according to the thickness of the glass, its refractive index, and the curvature of the surfaces, and it is by varying these that the desired degree of enlargement or diminution of the retinal image can be produced. There is thus a means of correcting what might be called regular aniseikonia, and Ames states that he has succeeded in relieving asthenopia in certain cases which had failed to respond to other methods.

Beyond having had one patient who was the unsuccessful subject of this treatment and was cured by other methods, I cannot claim to have had any clinical experience of it though I did have some size lenses made and tried them out with modified Ames' targets in an amblyoscope and found they worked.

The argument that the strain of trying to combine two unequal ocular images may conduce to asthenopia seems a rational one, though, and worthy of further study in this country.

The last chapter in the book of causes of asthenopia deals with the functional element. The psychiatrists would with some justification put it first, and T. A. Ross in his book "The Common Neuroses" tells us how important is the part played by these conditions in the genesis of asthenopia. It is of course something which we already know, and I have no doubt we could spend many hours exchanging case histories. Some of these I related at the Oxford Congress in 1934,⁹ and I have had several similar cases since and have probably missed a number too, and ordered glasses or orthoptic treatment when what was really needed was a readjustment of home life. Some of these patients are extremely difficult to tackle, because the one thing people in this country seem to resent more than anything else, is giving themselves away by admitting that what to them is a very real affliction is merely a matter of what they would call "nerves." Parents too very often dislike the thought that an onlooker may see more of the game, so far as their children are concerned, than they do. I remember very well seeing a boy, aged 19, who was training for some sort of clerical work and complaining of his eyes. There seemed to be little cause for asthenopia and his attitude toward the tests and his general conduct suggested to me that his interests were artistic. So I asked him suddenly, "didn't he want to be an artist?" His whole expression changed and he said, "how did you know?" I did not tell him, but explained that his eyes would probably go on troubling him until he achieved his ambition and thought I had done some good work. The result was, however, that two angry parents visited me next day saying that of course he did not want to be an artist; they had had a long talk with him and all he wanted was to follow his father in business. Whether the boy broke away or not, I do not know,

but if he did not, I hope he was able to get rid of his asthenopia by rationalising it. The thing can be carried too far, however, as was shown by an account in a psychological journal of a woman aged 43, who found she required glasses for seeing names in the telephone book and had her trouble put down to the fact that she suspected her husband of infidelity and was constantly looking for the other woman's name, but did not want to find it and so acquired blurred vision.

I quoted this example because although psychological causes may bring about asthenopia they are not the only cause. There is a danger nowadays that symptoms which will readily yield to ocular treatment are put down to neurosis. I was, for example, discussing the refraction part of this paper a short time ago with a senior clinical assistant at hospital, who dismissed the problem with an airy "Oh, but don't you know, that's all just suggestion." Some of it may be, but I don't think it all is. As usual, the Greeks have a word for it, and that word is *ὑβρις*. It has many translations; it is not a mortal sin, but it signifies any departure from the safe middle path prescribed by your principles. Extravagance of any kind to the Greeks was perilous. Poverty was no virtue to them, and it was just as bad to be extremely poor as to be extremely rich. The same is surely true of our approach to the problem of asthenopia. I don't say that all the methods of examination I have outlined should be applied to every patient who comes complaining of a headache when she reads. This would be *ὑβρις* and it would be equally *hubritic*, if one may coin such a word, to assume that all cases can be cured by one method. The rational procedure would seem to be, to take the simplest thing first, which to us is correction of the error of refraction, and if this fails, to proceed to other methods of examination according to the indications of the case.

In conclusion, I feel I owe an apology to those who may have come to this lecture in the hope that I would offer a universal solution to the problem of asthenopia. When deciding on its title I felt inclined to incorporate the adjective insoluble, but this might have discouraged you, it might also have been *ὑβρις* because, as I hope I have shown, although each individual patient is at first sight a problem, in the majority of cases, the problem is solvable by the methods which are now available. Thank you very much for the kind attention which you have given to me.

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SPECIAL CASE OF MELANOSIS FUNDI: BILATERAL CONGENITAL GROUP PIGMENTATION OF THE CENTRAL AREA

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MAUTHNER (1868) and Ed. v. Jaeger (1869), later on Frost, Stephenson, Dodd, Juler, Niels Hoeg (1911) described a rare (Leber) sharply characterised change of the fundus. It occurs in a sector of the fundus—very often a narrow one, but sometimes broader—covered with dark patches, round or irregularly-shaped, united in little groups. The size of the spot increases from the disc to the periphery. The area between the disc and fovea is always free. The spots lie underneath the vessels. In only two of all the cases published do the spots cover the retinal vessels, but these cases are not universally acknowledged. No retinal or choroidal change, and no functional defect have been observed.

As a sample of such a congenital anomaly—assumed to belong to the pigment epithelium, we take a picture of H. Bedriska, a 26 years old girl. External examination normal; no defect; visual acuity 6/5 in both eyes; +0.5 dptr. id.; small pale pigment ring. In the macular area of the left eye there are very tiny colloid bodies, grouped pigment patches in a narrow sector downwards at 6 o'clock (Fig. 1), the smallest near the disc, the larger and darker ones more distant. In the more central part the greyish patches have a granulation feature, the peripheral ones being deep black and nearly twice the size of the disc. They are to be seen at the utmost periphery of the indirect image. No limit of the