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COMMUNICATIONS

CONVERGENCE DEFICIENCY*

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A VAST amount of literature has grown up around the subject of heterophoria and disorders of convergence, both before the renewed interest awakened by Maddox in orthoptic treatment in this country and after. I do not propose to make any attempt at an historical survey, which would have to go back to the time of Javal or further, but there are certain practical aspects of the subject which I think may be profitably considered from the clinical point of view.

In the first place I wish to make it clear that I am not dealing with the subject of heterophoria as a whole, but with a small group of cases characterised by the fact that they show a deviation of 6° or more (exophoria) on the Maddox wing test and that when ordered to converge voluntarily without an object to fix upon they are totally unable to perform the movement, even when it has been explained and demonstrated to them. This latter sign I consider of greater importance than the readings with

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the Maddox Wing and Maddox Rod. It might, however, be objected that the movement is practised by some children as a trick ("squinting for fun") though not by all, and so one would expect to find some people proficient and not others. I did, however, enquire as to this and nearly all the patients to be described told me they had tried and always failed to do the movement in childhood. With the Maddox Wing test there is always a deviation of more than 6° , but this may vary from 6° to 22° , or the pointer may even appear to slide right off the scale, being often seen at 6° momentarily to start with. Such cases with the Maddox Rod may show orthophoria, exophoria or esophoria. I am aware that for the diagnosis of true convergence deficiency the patient should not show exophoria for distance, but I consider this distinction arbitrary. The cases I am dealing with have this in common, namely, deviation of more than 6° outwards on the Maddox Wing and failure to perform convergence as a voluntary action. The majority of them were, it is true, orthophoric or esophoric for distance, but I do not consider this essential for the diagnosis, since two separate factors come into the question, namely, the position of the eyes at rest and the amount of voluntary control over the internal recti; and these are not necessarily connected.

We are all acquainted with the type of patient who complains of suffering constantly from "eye-strain," of being unable to engage in any close work for long at a time, and of gaining little or no help from glasses. Such patients often arrive with a sheaf of prescriptions and a handbag full of spectacles and our hearts sink when these are produced. The majority of them show a very low grade hypermetropic astigmatism and nothing else, other than deviation outwards on the Maddox Rod and failure to perform convergence as a voluntary action. Some, however, show something more definite than this, such as marked anisometropia or heterophoria for distance. Myopia of high degree is rare, though low myopia may occur. Some patients are entirely emmetropic. A large number of these patients impress us from the beginning as being of the neurotic and neurasthenic types, but by no means all. Some indeed show very marked neurotic reactions, especially when asked to converge on a finger approached towards them. Head retraction, sweating, tremor and general distress may be manifested but this marked reaction is probably only given by potential true psychotics.

It has been my aim to try to bring some order and understanding into this complex and puzzling group. To this end I have analysed 6,400 case sheets from my private practice (since such cases are more easily investigated and dealt with among private patients and often escape attention in hospital work) and

have attempted to come to some conclusions. In the first place I should like to quote from the paper by Berens, Hardy and Stark on the same subject. In 1929 they went through the literature of the subject and analysed over 11,000 of their own private cases. They came to the conclusion that "A review of the literature concerning the condition . . . revealed a wide variance of opinion regarding almost all the factors connected with it." They give the following tabulation of the results of other observers up to 1929.

TABLE I

Tabulation of published opinions concerning Divergence Excess

Author	Frequency	Correlation with Refraction	Value of Exercises	Primary Treatment	Final Treatment
Wootton ...	"Very Common"	— hyperopia	Slight	Correct refraction	Tenotomy repeated
Wilkinson ...	—	—	—	Prism in lens ; exercises	Tenotomy if above 5°
Reber ...	"Common"	—	Very valuable in 75% of exophorias	Exercises : prism in lens	Tenotomy
Young ...	Rare : 1·8% of refractions	— hyperopia	Valuable	Minus sphere exercises	Recession
Bulson ...	—	—	Very valuable	Exercises (prolonged)	Advancement or resection
Lauder ...	—	—	None	Prism in lens	Tenotomy (early)
Duane ...	Common ; 33% of exophorias	None	Very valuable	Orthoptic	Tenotomy ; will require exercises as well
Dunnington	Common	None	None	Tenotomy repeated	—
Maxwell ...	—	Doubtful	Frequently valuable	Orthoptic	Tenotomy
Allen ...	—	—	Valuable and permanent	Orthoptic	Surgical

From their own practices they arrived at the following figures :—

TABLE II
Frequency of Divergence Excess in the private practice of Ophthalmology

Among 11,500 serial cases in private practice there were found 114 cases of Divergence Excess cross-filed	...	...	...	...	...	...	% 0'993
Examining every 10th record among 10,000 serial records there were found 18 cases of Divergence Excess	...	...	...	...	...	...	1'8
The frequency, therefore, is put	...	...	...	...	...	...	1—2

In my series I found a percentage of between 2 and 3 per cent. The exact number cannot be worked out more accurately as a number of cases were border line with a deviation of 6° and few or no symptoms. Undoubted cases formed 2·6 per cent. (167 out of 6,400), which is slightly higher than Berens' figure, but it is obvious that individual surgeons will vary slightly in what they include. I was myself always hunting for cases and so have probably included more border-line ones than other observers. Of the 167 cases not all were treated and not all carried out the treatment. The following conclusions are therefore drawn from the 82 cases from among them which I have more thoroughly investigated.

In considering the group as a whole one is first struck by the lack of correlation between various associated factors. The refraction shows no actual relation to the condition. Berens' series shows 54·4 per cent. hypermetropic in one or both meridians of one or both eyes, and 45·6 per cent. myopic in one or both meridians of one or both eyes. In my cases the results are as follows:—

TABLE III

Hypermetropia and hypermetropic astigmatism under 3 dioptries in highest meridian	...	...	...	...	...	% 65
Myopia and myopic astigmatism under 4 dioptries in highest meridian	...	...	...	...	...	23
High astigmatism, over 3 dioptries	...	...	...	...	...	3
High anisometropia	...	...	...	...	...	9

This is roughly the percentage of these errors in all cases in private practice and proves nothing beyond what was already known, that there is no correlation with the refraction.

I have attempted a further correlation with the age in order to ascertain if possible whether the condition was more frequent

in early life and tended to spontaneous cure or whether it arose during adult life and became progressively worse. The results of the 82 cases were as follows :—

TABLE IV

1.	Patients under 20 years	...	...	...	25*
2.	„ „ 20-30 years	...	...	...	11
3.	„ „ 30-40 „	...	...	...	14
4.	„ „ 40-50 „	...	...	...	10
5.	„ „ 50-60 „	...	...	...	8
6.	„ „ 60-70 „	...	...	...	14

*Note that this group concerns 20 years, the others 10.
Hence apparent preponderance.

The variations here are negligible and seem to show no preponderance in any age group. Again, the results of treatment need to be tabulated. I have not myself resorted to operative treatment in any case, so that the method used has always been orthoptic. Actually the exercises given have varied considerably and this will be dealt with later.

In Berens' series the results are shown in the following table, which includes three hypermetropic cases surgically treated.

TABLE V

	Cured	Improved	No change
Symptomatic—Myope ...	77·0%	—	23·0%
Hyperope ...	62·5%	37·5%	—
Myope ...	61·5%	38·5%	—
Objective—Hyperope ...	12·5%	87·5%	—

A myope is more likely to be cured than a hyperope, and a symptomatic cure is more frequent with either than an objective cure.

A myope has a better chance of being cured, subjectively or objectively, and less chance of being only improved.

Conversely, a hyperope shows more likelihood of being improved (temporarily?) and less likelihood of a cure.

Hence in this short series, our myopes predominating (13 to 8), our results will probably be more encouraging than others report.

N.B.—All our operative cases were hyperopes (Berens).

In my series the results are as follows (not all the 82 cases investigated were actually treated).

TABLE VI

Percentage results of treatment of Maddox Wing exophoria and absence of voluntary convergence

	Treated	Cured	Improv'd	No change
Symptomatic—Hypermetropes	52	37	14	1
Myopes ...	14	8	5	1
Anisometropes	5	3	2	—
Objective — Hypermetropes	52	31	18	3
Myopes ...	14	6	7	1
Anisometropes	5	3	2	—
Total ... Symptomatic...	71	48	21	2
Objective ...	71	40	27	4

These figures agree with Berens, Hardy and Stark in that they show a high percentage of cures and improvements following treatment and also in the illuminating fact that it is possible to produce apparent alleviation of all the symptoms without producing any objective cure. This is only one more argument in favour of the already recognised fact that neurasthenia and other psychological factors account for many of the symptoms, and brings us to the statement of the main clinical problem which I have tried to elucidate. This is, stated simply, the relationship of the subjective symptoms ("eye-strain," inability to read with comfort, headache and sometimes photophobia) to the objective signs (exophoria of more than 6° on the Maddox Wing and failure to perform binocular convergence as a voluntary action). That this is a definite problem is seen from the undoubted facts that (1) the objective signs can exist for years without any symptoms at all, (2) that cure of the signs does not necessarily produce disappearance of the symptoms, (3) that the symptoms may disappear entirely while the signs remain unaltered, (4) that relapse and recurrence of the symptoms may occur without reappearance of the objective signs, and finally (5) that cases exist with all the classical symptoms and no objectively demonstrable signs at all. The following cases illustrate these five points:—

Case I.—Miss ———, aged 38 years. University Lecturer and research worker. Wears glasses for myopic astigmatism

(R. $\frac{-2.25 \text{ D. sph.}}{-1.0 \text{ D. cyl. ax. } 105^\circ}$ L. $\frac{-1.75 \text{ D. sph.}}{-2.0 \text{ D. cyl. ax. } 60^\circ}$ 6/6 R. and L.)

but has not had them changed for twenty years. Has no headaches or eye symptoms whatever. Does a great deal of reading and close work and merely requested examination because she thought it was time she had new glasses, not on account of symptoms. She shows an exophoria of 22° on the Maddox Wing and no voluntary convergence, but binocular vision for distance and on the Maddox Wing does not suppress either eye though must do so when reading. This case shows that the condition can exist without symptoms.

Case II.—That cure of the signs does not necessarily produce disappearance of the symptoms is shown by the case of Mr. ———, aged 30 years, who is a clerk doing constant close work and suffering much from "eye-strain." After orthoptic treatment patient was orthophoric with full range of fusion (to 60° on synoptophore) but the symptoms were no better. The cause in this case was an obvious psychological one, as he admitted that he hated his job and wished all the time to be an engineer, but lacked the initiative to set about it.

Case III.—This was a medical woman, aged 28 years, when I first saw her. She was a typical case and felt "eye-strain" when driving a car as well as when reading. She did not bother to carry out the orthoptic treatment at all and I did not see her again for 10 years, when she reported she now had no symptoms whatever. She was married and had children, but was not worried or overworked and could read and drive a car with comfort. She still showed 12° exophoria on the Maddox Wing, no voluntary convergence and only about 20° amplitude on the synoptophore with the easiest slide (the rabbit). In this case the symptoms have disappeared with the patient's greater sense of security.

Case IV.—Mr. ———, aged 18 years. This patient was treated for convergence deficiency by an ophthalmic surgeon abroad, well acquainted with the condition, who had produced a cure, both subjective and objective. The patient then came to live in England and all the symptoms returned. He was referred by the former surgeon, who sent me full notes. On examination he was orthophoric with a fusion amplitude of 70° on the synoptophore and I could find nothing wrong at all and yet the symptoms had returned. This shows relapse of symptoms without re-appearance of signs.

Case V.—Married woman, aged 42 years. Complains of excessive eye-strain and inability to do any close work whatever for more than a minute or two. Is so worried by her inability to read that she is sure she is going blind and is learning Braille. On examination she has slight hypermetropic astigmatism (R. $\frac{+ .75 \text{ D. sph.}}{+ .75 \text{ D. cyl. ax. } 85^\circ}$ L. $\frac{+ .75 \text{ D. sph.}}{+ 1.0 \text{ D. cyl. ax. } 90^\circ}$) and a visual acuity of 6/6 right and left. She has perfect binocular vision and stereopsis, is orthophoric on the Maddox Wing and has a fusion amplitude of 65° on the synoptophore. She admits to marital difficulties and to a previous illness with abdominal pain for which she had a laparotomy and nothing was found. This patient is a purely psychological case, probably a true hysteria, and the symptoms bear no relation to the condition of the eyes.

It is thus obvious that the treatment of the symptoms of eye-strain is not so simple as one might imagine. We should actually be justified if from the foregoing five cases we argued that there is no proof of connection between the signs and symptoms whatever and it would be possible to argue that we are dealing with entirely separate conditions which in certain cases happen to co-exist. However, if this were actually the case we should not expect to find the high percentage of cases (in Tables V and VI) in which disappearance of the symptoms was associated with removal of the objective signs. There must be some connection, but it seems probable that it is not the direct one of cause and effect which many surgeons (*e.g.*, Stutterheim, Reber, Duane and Allen) have considered possible.

I propose to tackle this problem by a study of certain cases in the different age groups with the idea of explaining the origin of the objective signs, since if these are understood it may throw light on whether they alone are responsible for the symptoms.

It is well known that from an evolutionary point of view increasing binocular overlap of the fields of vision appears to be correlated with increasing neopallial development in the central nervous system. This has been pointed out again and again by anatomists such as Brouwer and Elliot Smith. That the correlate of binocular overlap is neopallial increase and not increase in complexity of the eye is shown by the condition in the primitive reptile *Sphenodon* which has a good macula, but a primitive brain and no binocular overlap. Now the overlap increases until stereoscopic vision becomes possible with superposition of the central areas of both fields. The final and highest form of binocular vision can therefore be considered to arise when by voluntary muscular action the animal can increase the

overlap of the fields so as to obtain stereoscopic vision at the near point. This occurs so late in evolution that it is probable that only man shows it and certain that not all individuals attain it. It is also well known that certain movements which are primarily largely reflex and involuntary can by practice be brought under voluntary control. Elliot Smith, Wood Jones and other anatomists have shown that such voluntary control is dependent on the existence of cerebral connections whereby the lower centres are linked with the cortical areas controlling such movements. In most people such connections exist, though in many they are not consciously used but can be made use of by practice. The exact cerebral localisation of function such as was envisaged by Sherrington, Horsley and Beevor, and Campbell is probably not capable of complete proof, while the work of Poljak seems to show that there is in the production of even the simplest efferent impulse a generalised activity of the whole cortex. In Gordon Holmes' most valuable paper "The Cerebral Integration of the Ocular Movements" we have a most lucid account of the present state of our knowledge of the anatomical pathways involved. Beevor and Horsley's centre for eye movements in monkeys lies in the frontal lobe separated by unexcitable cortex from the pre-central gyrus. This appears to be also the case in man, though destruction, if unilateral, does not produce palsy. In addition to this centre there is another in the occipital lobe, close to but not within the area striata. Fibres from here pass to the anterior corpora quadrigemina in the neighbourhood of the oculo-motor nuclei. They descend medial to the optic radiations and pass through the pulvinar and anterior brachium to the tectum of the mid-brain. Thus there are two cortical areas (frontal and occipital) involved in eye movements. The frontal area appears to govern voluntary movements and if it is destroyed the patient cannot look in a certain direction to command though he can follow a moving object, since his fixation reflex is mediated by the intact occipital centre. If the occipital centre is destroyed the fixation reflex is lost though the patient can perform voluntary movements of the eyes. Fixation is largely reflex but requires the intervention of consciousness. All authors from Ferrier onward have stressed the necessity for active "attention" in the acts associated with vision. Gordon Holmes says: "It is only the object which occupies attention which excites the fixation reflex." The movements of convergence are of the same nature, both conscious and reflex, and for the attainment of binocular stereoscopic vision of a near object both centres come into play. Gordon Holmes says of the frontal centre, "through it we can by an effort of will look or turn our eyes in any direction and converge them on

a near object. By it, too, we keep our eyes directed on any object which interests us, though this is largely a function of the occipital cortex." In the cases under consideration it is likely that what we are doing or trying to do is to increase the permeability of the frontal path by "facilitation," using conscious attention as the stimulus and the fixation reflex as the source of the impulse. This is, of course, much older and very much more firmly established phylogenetically than the frontal side of the complete reflex.

Other bodily movements have no area of cortical re-representation whatever and no amount of practice will bring them under voluntary control, while others are not usually controlled but may become so. As an example we can consider the contractions of skeletal muscles. In most people the neurologists' conception that integrated movements and not individual muscles are represented in the cortex is true, but it is also well known that by practice individual muscles may be made to contract voluntarily also, without movement being associated. (Anatomists and artists' models often become good at this.) On the other hand the movements of the heart, the contractions of the ureters, and most of the contractions of the alimentary canal are quite unrepresented in the cortex and cannot be controlled voluntarily. The contraction of the bladder occupies an intermediate position since it is not voluntary at birth and control of it is acquired slowly and at very varying rates and with varying success in different children. In the same way the contraction of both internal recti together to produce convergence may never be under voluntary control, or it may be under partial voluntary control in that it occurs in association with accommodation when called forth by the fixation reflex when the child wishes to look at a near object or it may from a quite early age be both reflex (when looking at a near object) and purely voluntarily when it is done as a trick from the frontal centre alone. This, the power of using convergence either as part of the reflex of complicated co-ordinated movements required in reading, or as a purely voluntary single movement, represents the highest possible development of the area of cortical re-representation of the eye movements in man.* A study of a series of children has seemed to show that it is acquired normally at about the age of six, but may be delayed. If it is delayed, or if the child does much close work before it is developed, symptoms may

* There is one further step in the development of cortical control of the eye movements which is difficult and perhaps impossible for a large number of people, namely, voluntary dissociation of the accommodation-convergence link. This can often be acquired by practising the fusion of stereoscopic pictures without a stereoscope by alternately diverging and converging, keeping them in focus in both positions.

result. A few cases may make this clear. K.N. is a little girl aged 6 years. She complained of "eye ache" when learning to read and when first tested showed +.75 hypermetropia and a visual acuity of 6/6 part. On the synoptophore she fused at +5° only and had no amplitude at all. No treatment was given beyond glasses for reading, but in six months she had grown and was altogether better. She had full (60°) amplitude of fusion, was orthophoric and had a visual acuity of 6/5 right and left. A year later she reported free from symptoms with and without glasses. This case I take to be one of normal development.

A.B. was also a girl aged 6 years. She was similar to K.N., but in addition had high hypermetropic astigmatism and anisometropia. (Rt. +5.0D. sph.

+4.5D. cyl. ax. vert. 6/24 Lt. +2.0D. sph.
+1.5D. cyl. ax. vert. 6/9).

She had no idea of convergence, though in spite of the apparent amblyopia in the right eye she had fusion and stereopsis at 0°. She was given glasses and a stereoscope and Wells charts, but no pure convergence exercises as the anisometropia seemed likely to be the cause of the delayed development. In five months she improved to an amplitude of convergence of 40° and a visual acuity of 6/9 and 6/6. Here the development of the convergence has occurred normally when the anisometropia was corrected.

P.M., aged 5 years, 3 months, was brought because she held things close. She was slightly hypermetropic and rather underdeveloped and difficult to test. She had binocular vision. At nine she complained of headaches and was given glasses (+ 1.5 D.sph. R. + 1.25 D.sph. L.) for reading but no exercises, as I thought she would develop normally. Four years after, after two changes of glasses, she still had headaches on reading and I put her on to convergence exercises. She had 8° of exophoria on the Maddox Wing. A month later she was orthophoric, with amplitude of 60° on the synoptophore, a visual acuity of 6/4 right and left without glasses and no symptoms. This seems to be a case of delay of the final stage of development which just required the stimulus of the exercises to make it complete.

Another girl, V.N., aged 7 years, had an interesting history of a convergent squint in infancy. When I saw her she was straight, but had no amplitude of convergence. In a year on orthoptic exercises she had 60° voluntary convergence, binocular stereoscopic vision and no symptoms. As an example of the rapidity with which the cortical pathway can be opened up in an intelligent child, M.C., aged 9 years, can be cited. She noticed that she saw double on reading when tired and had a jerky power of adduction to 20° only at times. I explained carefully what was wrong and gave her exercises, and in four weeks she was

orthophoric with 60° voluntary convergence and had improved from 6/5 to 6/4 right and left. She was emmetropic and seemed to be purely a failure of cortical appreciation of what was wrong and of what to do when she saw double. These cases are all, I think, due to slightly delayed opening up of the cortical pathway. That the association tracts were there is shown by the rapid response to treatment.

Twelve similar cases in children under 13 were treated and in all the expected development occurred and the symptoms disappeared, although in some cases glasses were necessary for errors of refraction. One, a girl aged 13 years, is interesting in that I gave her at first prisms base in for reading simply to relieve the symptoms. She did not report again for eight years and the condition was unchanged (exophoria and convergence deficiency). I then gave her exercises and she became orthophoric. She took up photography and did very minute retouching work for a year without any symptoms. This shows that the cortical pathway was intact, but previously unused.

One of the children was definitely psychopathic and came from a Child Guidance Clinic with an anxiety neurosis. She obtained fusion to 40° and improved from 6/9 to 6/5, but I am sure will produce other symptoms later. She is of the type from which the neurasthenic patients in the older group come and the convergence deficiency is possibly fortuitous. All the other children seemed mentally normal, many of them extremely intelligent. Many of them were watched for three years and no relapse occurred.

Among children therefore, the condition of convergence deficiency seems to be part of a delayed use of cerebral pathways which are anatomically present, certainly after the sixth year. Exercises develop the child's awareness of the use they can make of their eyes and the control once gained is apparently stable. It is doubtful whether without exercises development occurs after 7 or 8.

As we have seen that there is no correlation with age whatever it is not necessary to divide adult cases into groups. This is to be expected if the condition is one of arrested development (of function, not of anatomical structure).

Most of the patients complained of difficulty in close work as the chief symptom. Some were distinctly neurotic and anxious in type. Others apparently normal, but overworked. One, a young man aged 21 years, did not know that anything was wrong till he failed to pass the Air Force test. He was very keen to get better and worked so hard at the exercises that he improved his convergence amplitude from 26° to 65° in twelve days and his wing test from -10 to $+4$. He was passed by the Air Force,

but could only do restricted flying as he had anomalous colour vision. Four years later he was still all right.

A typical case in a young adult was brought to me by the head of a teachers' training college. She was a brilliant and well balanced girl aged 18 years with 4 dioptres myopia and the Maddox Wing arrow was right off the scale. She had "awful headaches" nearly all day and there was a question of her giving up studying. I saw her three times only and she worked hard at exercises at home. In three weeks there was an improvement and in four months she was orthophoric for distance and near with 60° voluntary convergence. She wrote some months later, saying, "I have been doing a great deal of reading and sewing and the eyes have been behaving very well."

With one exception (an art student of neurotic type who did not continue treatment) all the patients up to 24 years obtained normal convergence and Maddox Wing tests. At 24 and over one began to find here and there patients in whom as well as the convergence deficiency there was definite mental instability. If this was marked no treatment was given, as in the case of a young married woman who with a little persuasion produced the delusion that she was a hermaphrodite and therefore could not look people straight in the face. In such cases I feel that the connection between the mental state and the convergence deficiency is a purely fortuitous one, though the pre-existing arrest of functional development of the eyes is probably the reason why a lot of the neurotic and psychopathic symptoms are referred to the eyes.

Some of these marked psychological cases are interesting, though for an ophthalmic surgeon, very difficult. On the whole they were more common in my series in older people. There were only three under 40 years. One, aged 33 years, was a medical woman who almost immediately after I had noted the convergence deficiency had a serious mental breakdown. Another, a man aged 34 years, began treatment and stopped as soon as he showed any improvement. He did not want to be cured. The fourth, aged 38 years, was a telephone operator who hated her work and took no trouble over her treatment and derived no benefit. Fourteen patients over 40 years showed intense anxiety neurosis or hypochondria or had a bad family history. One had an epileptic, two suicides and a paranoia in her immediate family and lived with the epileptic. In spite of this she was anxious to try treatment and worked very hard at exercises. She reached a stage in which she was comfortable for long periods, but had relapses always under extra strain. Another was intensely worried by the strain of house hunting; another, aged 48 years, had broken down from uncongenial work; one, a distinguished Army Officer,

was an intense hypochondriac and "enjoyed thoroughly rotten health." He refused treatment, as did three others whom I labelled "does not wish to get well," one of whom even refused to look at the tests. I must, however, in justice say that nearly all these patients were over 60 years and found any new ideas hard to acquire. This (age) does not necessarily mean that treatment will not be successful as the following case shows. The patient was a married woman aged 62 years, with marital difficulties and a neurotic history. She had a marked functional tremor of head and hands and complained of great pain in her head and eyes and many functional symptoms besides. She was given exercises and in four weeks she could converge 40° on the synoptophore. At this stage all the tremor disappeared quite suddenly and she reported feeling very fit and much happier. She continued well for thirteen months and then had a slight return of symptoms under strain. I do not think the exercises did anything except make her feel that something was being done at last.

In addition to these definitely pathological personalities there are the large number of adults who did do the treatment and were cured or improved. How many of them would have revealed psychological symptoms as well on examination I do not know. Many of them were overworked school teachers, people afraid of losing their jobs, people with family maladjustments, etc., but often they appeared quite normal. The majority responded very well to treatment and were cured or improved. Some I have no doubt whatever were cases of delayed development of function which had not been treated in infancy and had started to give symptoms when the patient began to study. B. W., aged 30 years, is a case in point. She was a medical student, normal, healthy and successful, but complaining of migraine and eye-strain. She had convergence deficiency, slight exophoria for distance and tended to neglect the right eye.

(Rt. $\frac{+0.25 \text{ D. sph.}}{+0.25 \text{ D. cyl. ax. } 25^\circ}$ Lt. $\frac{+0.25 \text{ D. sph.}}{+0.5 \text{ D. cyl. ax. vert.}}$) She found fusion exercises difficult, but worked hard. She worked for about six months and obtained orthophoria and convergence of 60° and discarded her glasses. The migraine occurred about every six weeks to two months but did not now come after studying. Eighteen months later she reported, "No effort in reading, no need to wear glasses. Feels quite different."

The length of time needed to get the maximum result is least in the intelligent children and the young adults. In older adults, as one would expect, it is longer. The maximum time taken to produce a fusion at 60° on the synoptophore and a Maddox Wing deviation of less than 6° , was a year in an adult aged 63 years,

while the shortest time was twelve days in the Air Force candidate mentioned above. Most other cases took between one and two months. On the other hand this does not mean necessarily more visits. About four visits are usually required, but in non-co-operative, unintelligent patients obviously more. Practically all the cases I treated myself. Some of the children and a few adults were sent to orthoptic trainers, but in these cases the average number of attendances was much greater, partly because some of them were also exophoric for distance and required more treatment, and partly because a slightly different method was used, with, I think, less direct explanation of what was required.

Treatment.—From the preceding consideration of cases we can possibly draw some conclusions which will assist in prognosis and treatment. In the first place it seems likely that the reason for the objective signs is arrest of the full functional development of the paths to and from the area of cortical re-representation of eye movements. The reason for the delay cannot be stated beyond the fact that the function is normally the last to be acquired. The reason for the symptoms is not simple. In some cases the condition is of no inconvenience whatever, if the individual is well balanced and not working under poor conditions or undue strain. Such cases should not be treated since we know from experience that they can be cured at any age if they do break down, and many may never break down. If, however, a poor psychological make-up is associated then the existing slight visual handicap is magnified and used as a peg on which to hang endless symptoms. These cases should be treated since they often do well. The concentration necessary to master the exercises is beneficial and the overcoming of the disability not only improves the condition of the eyes but often gives the patient fresh confidence in other directions. Only in extreme cases does the mental condition make the case unsuitable for treatment. These cases will, of course, be liable to reproduce the original symptoms or others later under strain.

By far the majority of cases, however, are normal individuals with an arrested development of function which only begins to bother them if they are doing excessive close work. All these cases should be treated, as they give excellent results and the patients are most grateful, not only on account of the increased comfort on reading, but also of the fact that many of the low hypermetropes under 40 years find that they can discard their glasses.

We now come to the question of the exact details of treatment. It follows that as we are trying to establish a movement which is normally dual in nature, being both part of an unconscious reflex and also capable of being performed voluntarily, two methods of

approach are possible. We can increase the power and amplitude of the reflex until it becomes conscious or we can endeavour to show the patient how to perform the voluntary action first and to relegate it to the level of a reflex again later when it has been mastered. Both methods appear to give identical results and one may be better than the other in certain cases. The first is applicable to children and to adults without much insight. The second to my mind is preferable as it is in my experience invariably shorter and more satisfying to the patient. Actually I always use a combination of the two in varying proportions. The first method includes graduated exercises in fusion on the synoptophore (Maddox and others) exercises with prisms to produce convergence for distance (kinetic treatment of Stutterheim) and exercises with a stereoscope and figures of graduated spacing and difficulty (as in the Hamblin series). All these methods increase the involuntary range of convergence.

The second method aims at stimulating the patient's appreciation of the proprioceptive impulses from the eye muscles so that he learns first to recognise the position of his eyes and thus quite soon to be able to control this. The principal procedures are the education of the patient in the appreciation of physiological diplopia and the control of this; exercises with the small diploscope and exercises in the fusion of stereoscopic cards without a stereoscope. If retinal neglect is present the Wells G. series of cards in a stereoscope is used until it disappears. The advantage in beginning with the second method is the amount of time saved. I first became interested in the whole problem as the result of some correspondence with Dr. Stutterheim of Johannesburg, the author of "Eye Strain and Convergence." I then used his method of fusion of a distant object against prisms base out and also an arrangement of rotating prisms in a trial frame which produced a somewhat similar result. The treatment took sometimes some months; Stutterheim himself states that twenty-four visits are usually necessary, but as many as ninety may be required, though I never actually saw a patient more than fourteen times. Even this I considered far too high and therefore attempted by education of the proprioceptive sense to shorten the treatment and to give the patient more to work on at home. I then found that if one began this way round the average number of visits was four and the time one to two months or less.

I will now outline the procedure as it seems to me to give the quickest results.

After the condition has been diagnosed and the patient shown on the Maddox Wing what is wrong, the treatment is explained. It is to my mind essential for the patient to understand what is being done, and I find it usually necessary to explain the difference

between binocular convergence and squinting as some patients will not do the exercises because they think a "squint" is produced. I next endeavour to demonstrate physiological diplopia, making the patient conscious of seeing two fingers when he holds a finger in front of his eyes and looks at a distant object (usually a small light). There is often difficulty about this as it is a surprise to many people and they will tend to neglect rather than to admit they see double. When this has been mastered, and it is extremely important, the patient is then shown how to produce diplopia for distant objects by looking at his finger. This may be impossible at the first visit and he is sent away with the following typed instructions and told to practise at home for a week or more.

"Convergence Exercises"

These exercises should be carried out in a dark room with a small light, such as a candle, at a distance of approximately six yards. Both eyes should be kept open all the time.

1. Hold up the finger vertically at arm's length in line with the light. Look at the light and notice that when the gaze is concentrated on the light the finger appears double. Arrange this (by moving the finger if necessary) so that the light appears midway between the two fingers. Now look directly at the finger, which will appear single, notice that the light is now double, and one light appears on either side of the finger.

2. Concentrate the gaze of the finger all the time and move it slowly from arm's length towards the nose. The finger should appear single all the time and the two lights should get slightly farther apart as it approaches the eyes. Repeat this, moving the finger slowly backwards and forwards. Care should be taken to concentrate on the finger all the time as this must never be seen double during this exercise.

3. When exercises 1 and 2 have been thoroughly mastered this exercise should be attempted, but not before. Hold the finger at a distance of approximately one foot from the eyes, and concentrate the gaze in the air just above the top of it which should be between the two lights. Without moving the eyes, lower the finger. If the eyes have not moved, obviously the light will still be seen double and in the same position. Endeavour to maintain the two lights at the same distance apart for as long as possible. This is at first very difficult, but becomes easier with practice.

4. Perform exercise 3, but without using the finger at all. Simply imagine that it is in the air, and double the light by looking at a point in the air about one foot from the eyes.

When exercise 2 has been mastered, usually in a week, the patient is given a treatment on the synoptophore with very easy fusion pictures (the rabbit and some of the stereoscopic ones) and it is explained that the effect of decreasing the angle on the instrument is the same as moving the picture nearer and he must try to follow it. It is essential that the patient tries to understand what he is doing. Exercise 3 then becomes just possible and the patient is given a stereoscope and Wells charts. The G series

is used first to ensure that there is no more suppression and then the E and C and I series. These latter should first be viewed through the stereoscope and then taken out and fused by holding a thin pencil vertically between the eyes and the card and endeavouring to converge on the pencil while noting what happens to the picture. Further treatment with the synoptophore on harder pictures is then given and finally the small diploscope and pointer for home use and the A series of Wells charts, which having no lock are the hardest. The pencil is dispensed with and if the patient holds the parrot steady in the cage by voluntary convergence without the stereoscope the cure is complete. If they are then tested with Stutterheim's prisms and the synoptophore they will have from 60° to 75° of convergence amplitude and the symptoms will have disappeared in all cases except the marked psychopaths and frequently in them also.

In conclusion, I wish to emphasize the clinical importance of absence or weakness of voluntary convergence and to point out the fact that it is among the most easily treated of the heterophorias.

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