

# Management of microtropia

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Microtropia or microstrabismus may be briefly described as a manifest strabismus of less than  $5^\circ$  with harmonious anomalous correspondence. Three forms can be distinguished: primary constant, primary decompensating, and secondary.

There are three situations in which the ophthalmologist may be confronted with microtropia:

(1) Amblyopia without strabismus;

(2) Hereditary and familial strabismus;

(3) Residual strabismus after surgery. This may be called secondary microtropia, for everyone will admit that in most cases of convergent strabismus perfect parallelism and bifoveal fixation are not achieved even after expert treatment.

Microtropia and similar conditions were not mentioned by such well-known early practitioners as Javal, Worth, Duane, and Bielschowsky. The views of Maddox (1898), that very small angles were extremely rare, and that the natural tendency to fusion was much too strong to allow small angles to exist, appear to be typical.

The first to mention small residual angles was Pugh (1936), who wrote:

“A patient with monocular squint who has been trained to have equal vision in each eye and full stereoscopic vision with good amplitude of fusion may in 3 months relapse into a slight deviation in the weaker eye and the vision retrogresses”.

Similar observations of small residual angles have been made by Swan, Kirschberg, Jampolsky, Gittoes-Davis, Cashell, Lyle, Broadman, and Görtz. There has been much discussion in both the *British Orthoptic Journal* and the *American Orthoptic Journal* on the cause of this condition and ways of avoiding it.

Pugh incriminated the tendency to suppression, Jampolsky an imperfectly cured amblyopia, Bedrossian an insufficient surgical correction, and Gittoes-Davis an insufficient correction of hyperopia. Swan believed that such residual deviations could be avoided by intensive postoperative orthoptic treatment, and Cüppers wrote that residual deviations would be prevented by treating anomalous correspondence with after-images. Nowadays, of course, prisms seem to be the panacea, even against microtropia.

In reviewing all these theories, it becomes clear that one has fallen once again into the old trap. Before knowing the nature of a condition one tries to cure it, and before trying to elucidate a condition one screens its origin with the smoke of therapeutic effort.

It is perhaps more helpful to start the investigation not with patients who have undergone a lengthy course of treatment but with the more interesting cases of primary microstrabismus, in whom no overt strabismus has been noticed, in whom no treatment has changed the original sensory conditions, and in whom more insight may be gained into the primary binocular pathology.

**Personal observations**

In the course of 15 years, 26,762 patients have been examined for primary microtropia in my practice. This total includes every patient with every kind of ocular disorder, not only those with amblyopia or disturbances of binocular vision. It was discovered that, among 1,789 patients with convergent and 515 with divergent strabismus, there were 755 cases of convergent microtropia but only nineteen of divergent microtropia. In the group with convergent strabismus, microtropia comprised 42.2 per cent.: 338 cases (18.8 per cent.) of primary constant microtropia and 417 cases (23.3 per cent.) of decompensated or secondary microtropia.

**PRIMARY CONSTANT MICROTROPICIA**

Among the 388 cases of primary microtropia there were 123 children. A careful statistical study was made of 120 cases, recording the age at diagnosis, the degree of deviation of the cover test, refraction, fixation, correspondence, visual acuity, familial incidence, and results of treatment.

On the unilateral cover test the average deviation was 3.8<sup>Δ</sup>, and this increased on the alternating prism cover test to an average deviation of 6.7<sup>Δ</sup>.

70 per cent. were isometropic and the remaining 30 per cent. anisometropic. Refraction in the isometropic cases showed a more or less normal distribution. In the anisometropic group there were 28 cases of anisohypermetropia and six of anisomyopia. In the cases of anisohypermetropia, the microtropic eye was always more hypermetropic.

Before treatment, fixation was central in 50 per cent., unstable central in 5 per cent., and eccentric in 45 per cent.

The visual acuity was, of course, best in patients with isometropia and central fixation, and worst in those with anisometropia and eccentric fixation, those with isometropia and eccentric fixation or with anisometropia and central fixation coming in between (Fig. 1). From this it follows that amblyopia in primary microtropia covers a wide spectrum.

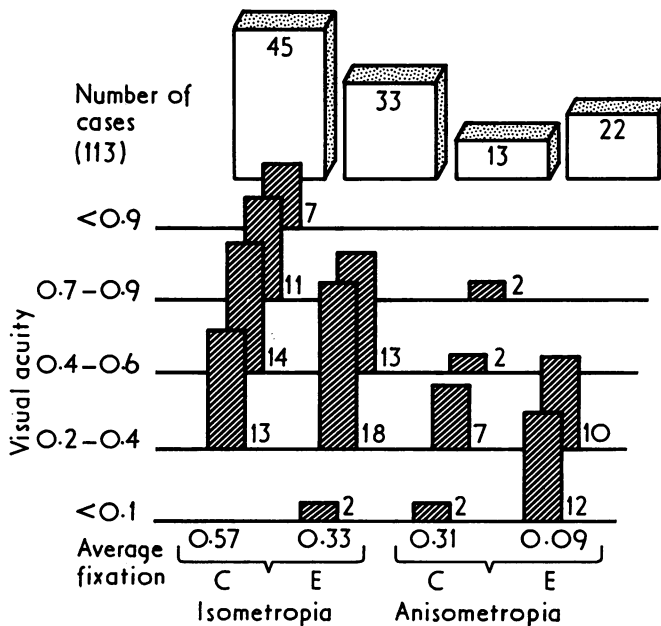


FIG. 1 Visual acuity in 113 children with primary constant microtropia

C = central fixation  
E = eccentric fixation

Microtropic amblyopia shows a characteristic which must be respected in treatment. Even those in whom distance vision seems to be good may complain of additional difficulties in reading, such as single letters of a word being fuzzy, disappearing, or getting mixed up with others. This "crowding" phenomenon, which exists even in patients with central fixation, is due to a temporal scotoma which can be demonstrated on the Amsler charts, and this explains why in microtropia of the right eye the final letters of a word seem fuzzy whereas in microtropia of the left eye the initial letters seem fuzzy.

An analysis of the degree of the angle of anomaly (Fig. 2) showed that small angles were more frequent, the curve showing almost one half of a binomial form.

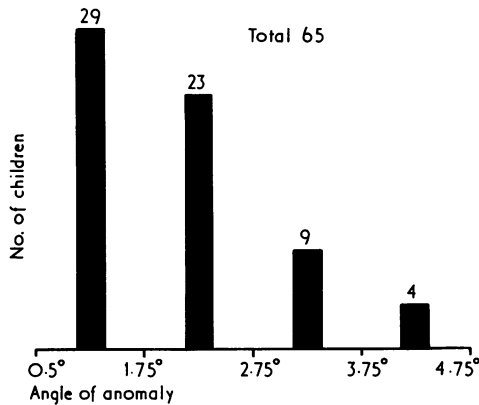


FIG. 2 Distribution of angle of anomaly in 65 children with primary constant microtropia

There were many other interesting findings, which can not be discussed here, but one is of particular importance, *i.e.* the familial incidence of microtropia. The constant finding in such cases is a small anomalous correspondence, fixation being central or eccentric.

#### PRIMARY DECOMPENSATING MICROTROPIA

In the examination of children with intermittent squint, a careful cover test soon after the onset of the squint may show that, even in the apparent parallel position, the eyes are not perfectly straight. The same is true when an amblyopia with eccentric fixation is present with apparent parallelism. We must therefore make sure that this decompensation arises not from the parallel position but from microtropia. We have found this condition in 128 children. The refraction of 84 children with constant primary microtropia was compared with that of 66 with decompensating primary microtropia. In the latter hypermetropia was more pronounced (Fig. 3, overleaf).

An important question is why a primary microtropia develops into a larger angle of deviation. The effect of therapy may give some clues to the answer. We found hypermetropia to be the decompensating factor in 21 per cent. of cases, and an essential convergent deviation or convergent position of rest was assumed to be the cause in another 21 per cent. In 8 per cent. amblyopia, and in 5 per cent. a high AC/A ratio or convergence excess, was thought to be the cause. In the remaining 45 per cent. two or more of these factors were involved.

This development can be shown to take place in a cycle (Fig. 4, overleaf). Primary microtropia develops into a manifest convergent squint by esopetal forces, such as hypermetropia, convergent position of rest, convergence excess, or amblyopia. Since anomalous correspondence pre-exists, sensory adaptations to the new angle are easily made. After therapy,

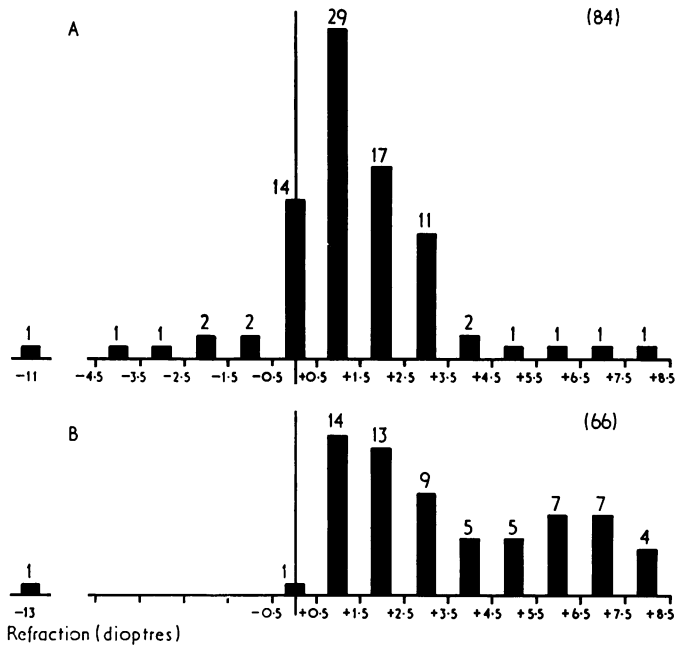


FIG. 3 Distribution of refraction (a) In 84 isometropic cases of primary constant microtropia (b) In 66 isometropic cases of primary decompensating microtropia

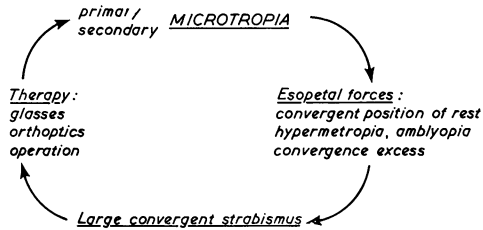


FIG. 4 Development from primary microtropia to large-angle strabismus and to secondary microtropia

not parallelism but the pre-existing microtropia shows up again. This supplies a simple answer to our question why, even after careful treatment, not parallelism but a secondary microtropia usually occurs.

From this another important finding may be deduced. Hitherto anomalous correspondence was always thought to be the consequence of motor disturbances. According to Bredemeyer (1968) and Bullock (1968), anomalous retinal correspondence is a sensory change that occurs only as a result of strabismus, but we can now arrive at a more precise definition. In cases of primary microtropia the deviation is so small that it should be corrected to parallelism by normal fusion, but a deviation is maintained by anomalous correspondence and we may therefore assume that not the motor but the sensory factor is the chief cause. There is much to be said for the view that, in cases of microtropia, strabismus is the result of anomalous retinal correspondence.

**SECONDARY MICROTROPIA**

The primary constant and primary decompensating forms of microtropia are well-defined clinical entities, but secondary microtropia is less distinct, because motor factors have been involved and therapy has changed the original pathology. Also, with less rigorous definition or in the course of time, the number of cases of secondary microtropia may increase and may differ from one author to another. In our series we have found 123 cases of primary con-

stant microtropia in children, 128 of decompensating microtropia, and 144 which may be classified as secondary microtropia. The last were usually seen a long time after the onset of squint or after treatment, and we could not decide whether a primary decompensating microtropia had been present beforehand or not.

A study of the literature would suggest that the primary form is very rare and that the decompensating form does not exist. According to Chamberlain and Caldwell (1964) the ratio of secondary to primary microtropia is 97 to three. Bullock (1966) found in thirty cases only two in which there had not previously been a larger deviation.

### Diagnosis

The diagnosis of microtropia is made by the unilateral cover test, by the investigation of fixation, and by the examination of anomalous correspondence, which striated lenses show to be harmonious. The alternate prism-cover test shows additional heterophoria. Among other helpful tests the most important is bifoveal visuscopy whereby the angle of anomaly and the centre of anomalous correspondence can be determined with the aid of a periscope-like double mirror (Fig. 5).

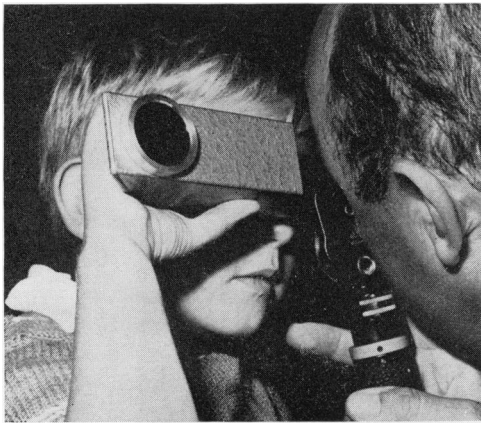
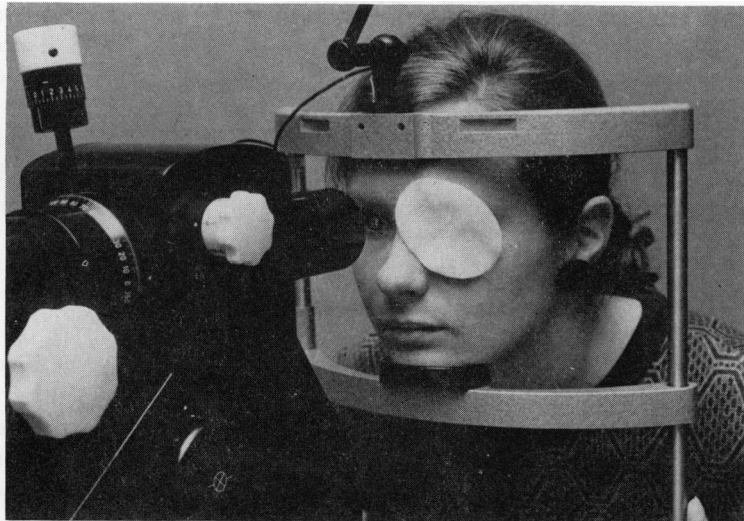


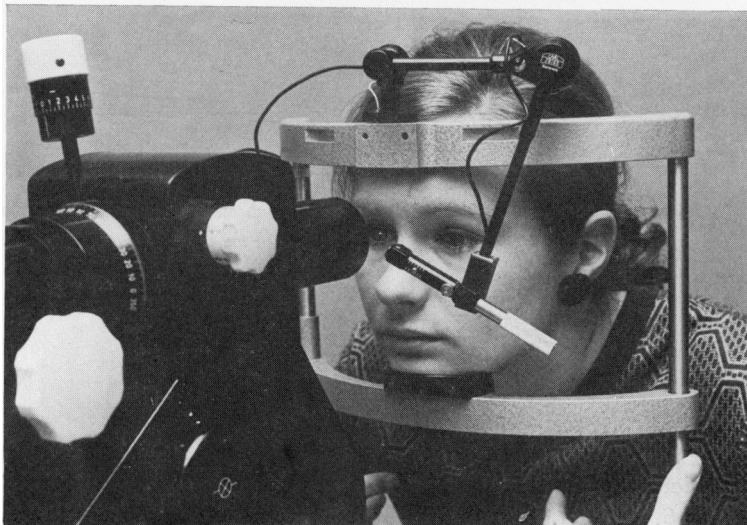
FIG. 5 *Ophthalmoscopic examination for retinal correspondence with the visuscope and double mirror*

In suitable patients (mostly adults) we have carried out fixation and correspondence photography (Fig. 6, overleaf). The non-fixing eye is dilated, the normal eye is kept closed (Fig. 6*a*), and a fixation photograph is taken in the usual way (6*a*, 7*c*). Then both eyes are kept open and the patient is asked to fix a small fixation lamp with the undilated normal eye (Fig. 6*b*). This lamp is moved so that the fundus of the dilated amblyopic eye appears in the camera. The target in the camera is now moved until it appears to be in the same direction as that the fixation lamp seen by the normal eye. At this moment the picture is taken (Fig. 7*d*). (See Fig. 7, *a-e*, overleaf).

Most cases of microtropia can be diagnosed without difficulty. Additional anisometropia and heterophoria, however, present some difficulties and sometimes in borderline cases the diagnosis must be left open. More difficult than the diagnosis of microtropia is the confusion in terminology. The English synonyms for microtropia include small angle, fixation disparity, retinal slip, flicker cases, fusion disparity, eso flick, minimal strabismus, monofixational phoria, ultras small angle, foveal slip, and monofixation syndrome. Even "small angle" means a different degree of deviation to different authors. Jampolsky (1951) used this term for a deviation up to 15<sup>Δ</sup>, Albert (1962) up to 6<sup>Δ</sup>, and Bedrossian (1968)



(a)



(b)

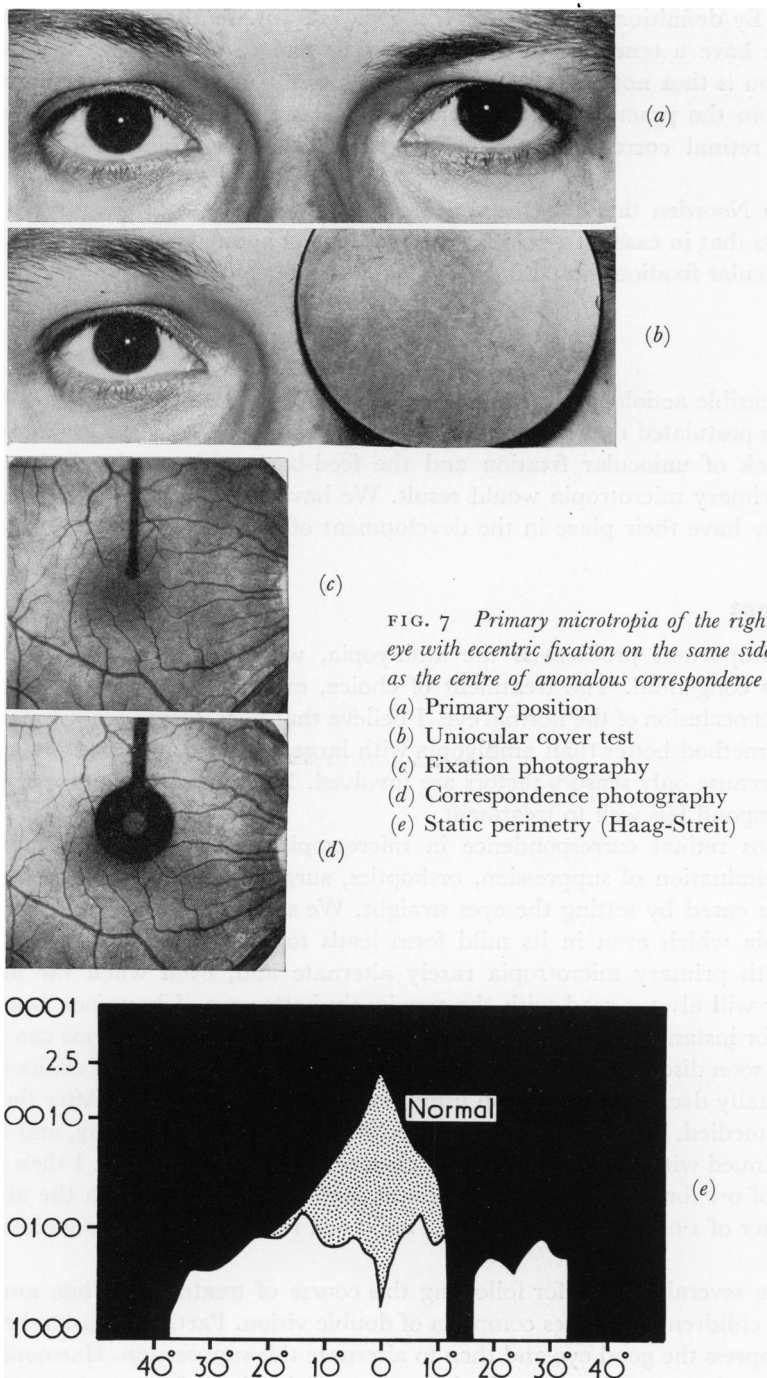
FIG. 6 *Photographic representation of fixation and retinal correspondence*

- (a) Fixation photography
- (b) Correspondence photography

from  $10$  to  $25^{\Delta}$ . It should be pointed out that Gittoes-Davis (1952) had already made a clear distinction between a small angle of about  $10^{\circ}$  and an angle of  $5^{\circ}$  and less.

### Monofixation syndrome

It seems reasonable to distinguish a microstrabismus of less than  $5^{\circ}$  from a small deviation of between  $5$  and  $12^{\circ}$ . Differentiation between microtropia and anomalies in the ortho-position gives us the opportunity to say a few words on the monofixation syndrome. Parks (1961) has rightly opposed the misuse of the term "fixation disparity", which in the sense given it by Ogle (1949) can not exceed  $20'$  of arc. He also has wisely given up the term "monofixational phoria" for a condition which essentially is not a phoria but a tropia. However, one may ask whether the new term "monofixation syndrome" is a happy choice. A careful analysis of the 100 cases published by Parks (1969) shows that the mono-



fixation syndrome includes not only microtropia but also anisometric amblyopia, stereo amblyopia, fully accommodative strabismus, surgically corrected normo-sensory convergent strabismus, operated cases of intermittent exotropia, and even an organic macular lesion. All these different conditions have just one symptom in common: the suppression

of one eye. By definition, a syndrome is a group of apparently unrelated symptoms and signs which have a tendency to appear together and to characterize a clinical picture. Monofixation is thus not a syndrome but a symptom common to many different clinical entities. From the practical point of view we prefer to distinguish microtropia with its anomalous retinal correspondence from anomalies of binocular vision in the ortho-position.

With von Noorden this disagreement on terminology seems to disappear, since he no longer insists that in cases of microtropia the centre of anomalous correspondence and the point of unocular fixation should coincide.

### **Aetiology**

The most feasible aetiology of microtropia seems to be the statistical theory of Goldmann (1967), who postulated that there must be a statistical variation in the interaction between the feed-back of unocular fixation and the feed-back of binocular fusion. From this variation primary microtropia would result. We have only to add that convergence and also heredity have their place in the development of this condition.

### **Management**

The most important problem is the amblyopia, which in the past has usually been regarded as congenital. The treatment of choice, even in cases of eccentric fixation, is simple direct occlusion of the normal eye. I believe that microtropic amblyopia responds to this simple method better than amblyopia with large manifest angles of deviation. This is probably because only sensory factors are involved. Patients with a high degree of anisometropia respond less well to treatment.

Anomalous retinal correspondence in microtropia appears to be incurable by any method—elimination of suppression, orthoptics, surgery, or prisms. Amblyopia can not therefore be cured by setting the eyes straight. We should try to prevent the recurrence of amblyopia which even in its mild form leads to an impairment of reading ability. Patients with primary microtropia rarely alternate and, even when the amblyopia is cured, they will always read with the previously better eye. I have not found part-time occlusion, for instance for television only, very effective, since no progress can be seen and the effort is soon discontinued by parents and patients. My treatment of choice is a tapering off or gradually decreasing occlusion using Bangerter's partial filters. After the amblyopia has been remedied, I continue with total occlusion alternating each day, and this alternation is continued with partial filters of less than 0.1 for about 6 months. I then go on to the next filter of 0.1 for about another 6 months, and so on until I reach the almost inconspicuous filter of 1.0 and am sure that the child can read with each eye without hesitation (Fig. 8).

There are several reasons for following this course of treatment. When amblyopia has been cured children sometimes complain of double vision. Partial occlusion helps to teach them to suppress the good eye and then to alternate this suppression. Harmonious anomalous correspondence occurs and valuable stereopsis develops. In cases in which the angle of deviation has increased during treatment for amblyopia, the angle decreases again with tapering-off occlusion. There are also psychological advantages. When the child is wearing an alternating occlusion, both his parents and himself are aware that he is still receiving treatment. Tapering-off occlusion makes the patient aware of his progress. I admit that



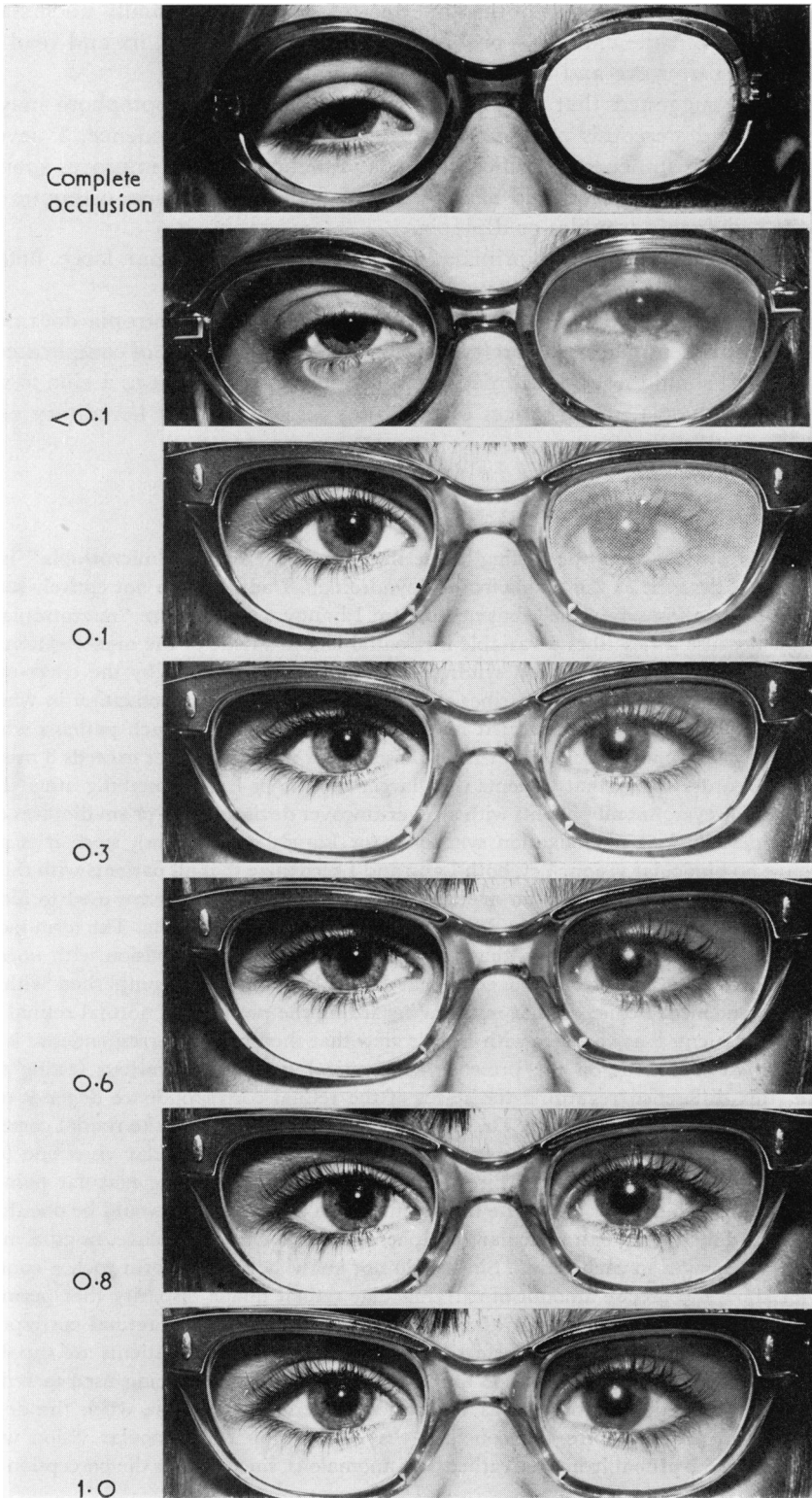


FIG. 8  
*Tapering-off  
occlusion in seven  
stages*

sometimes I make rather a cult of this tapering-off, but parents usually understand that if it is not carried out the child will probably not alternate but will fix and read only with the previously better eye and relapse again into amblyopia.

It has been suggested that orthoptic treatment with the synoptophore may improve fusion range and stereopsis on the basis of anomalous correspondence. I never do this because I feel it is unnecessary, since binocular function usually improves spontaneously during tapering-off occlusion and also because I fear that intensive orthoptic treatment may cause asthenopic troubles or diplopia.

In cases of decompensating primary microtropia or additional large heterophoria, the deviation should be treated by prisms or surgery.

In conclusion, I should like to emphasize that secondary microtropia does not arise as a consequence of treatment and that it cannot be avoided by the use of complicated methods of treatment. It should not be regarded as a tiresome symptom but as a clue to the understanding of amblyopia, disturbances of binocular vision, and the hereditary elements of strabismus.

## Discussion

**PARKS** It is obvious that the "thing" described by Dr. Lang as "microtropia" is the same condition that I describe as the "monofixation syndrome". I admit I am not entirely satisfied with the term "monofixation syndrome", but neither am I happy with the term "microtropia". I object to the latter because it describes a variable inessential characteristic of the monofixation syndrome. Some patients with the monofixation syndrome have a small deviation by the cover-uncover test while others do not. Microtropia describes only those patients with monofixation in whom a small deviation is elicited by cover-uncover; it ignores the large number of such patients who show no deviation. The cover-uncover measurement in those with a deviation never exceeds 8 prism dioptres and I would venture to say that patients with larger deviations have something other than Lang's microtropia. However, not all patients with a cover-uncover deviation of 8 prism dioptres or less have the "thing" described as monofixation syndrome (or Lang's microtropia), since it is possible for them to have no binocular vision. Yet, both Lang and I recognize that all patients with this condition have peripheral binocular vision as an essential characteristic. Both the terms used to identify these patients are weak in so far as neither describes this essential characteristic. The term monofixation syndrome leaves the possibility open that there is peripheral binocular vision with normal retinal correspondence, while microtropia suggests that binocular vision is accomplished with abnormal retinal correspondence. I concede that my view regarding the presence of normal retinal correspondence in these patients is at variance with Lang's view that their retinal correspondence is abnormal, although our views concur on the presence of identical findings by various testing techniques. Therefore, this difference regarding the status of the retinal correspondence depends on the idea of what is meant by correspondence. Dr. Lang determines the status of the retinal correspondence from a test that presents dissimilar images to the two maculae (binocular visuscope test), a test which records, supposedly, the visual directional values of corresponding macular points that are not functioning as a binocular unit. The important test in these patients would be one that assessed the binocularly functioning extramacular (peripheral) retinal correspondence, using a method that presents similar images to each retina. Since I do not know how to perform such a conclusive test and do not know the precise amount of extramacular retinal image disparity that permits normal peripheral retinal correspondence, an absolute statement regarding the retinal correspondence in these patients is not possible. However, starting from the fact that these patients are capable of both stereopsis and normal fusional vergence amplitudes (the latter often being used to reduce larger deviations to 8 prism dioptres or less, as the patient experiences diplopia when the deviation increases beyond 8 prism dioptres), I conclude that the peripheral binocular vision which these patients experience is probably normal rather than anomalous, since neither the perception of stereop-

sis nor normal fusional vergence amplitudes are characteristically encountered in patients with anomalous retinal correspondence.

The only two essentials invariably present in this group of patients are the presence of peripheral binocular vision and the absence of central binocular vision. Presuming that in normal retinal correspondence peripheral binocular vision prevails within the spectrum of manifest horizontal deviations between zero and 8 prism dioptres, all monofixation syndrome patients have identical sensory reactions despite the presence or absence of a very small deviation. It would be unfortunate further to confuse this already misunderstood group of patients by arbitrarily dividing them according to the presence or absence of a variable, minimal, and frequently questionable, deviation on the cover-uncover test, particularly when the presence of a very small deviation is not essential for the diagnosis of this syndrome. I use the term "monofixation syndrome" for lack of a better alternative and feel that it gives a more complete description of the total problem than the term "microtropia".

VON NOORDEN My concept of this situation is as follows. A large spectrum of forms of strabismus exists with inconspicuously small angles and various degrees of sensory adaptation. It is more important to analyse each case than to set up a multiplicity of confusing terms. This spectrum ranges from normal binocular vision with bifixation through fixation disparity as defined by Ogle, and the various manifestations of microtropia, to small-angle esotropia. I object to separating microtropia from small-angle esotropia taking a deviation of less than  $5^\circ$  as the only criterion. I should prefer to see microtropia separated from other kinds of small-angle squint by the criterion of the cover test. In microtropia the cover test is negative either because the fixation movement of the deviating eye is too small to be detected or because no such movement takes place, on account of identity between eccentricity of uniocular fixation and anomalous correspondence. The practical importance of microtropia lies in its recognition by the clinician. Numerous patients have come to my attention who were subjected to needless neurological surveys to establish the cause for reduced visual acuity in one eye, the doctor having failed to recognize an ultra-small deviation as the cause of the amblyopia.

I have one last point of disagreement with Dr. Lang; I cannot accept his contention that anomalous retinal correspondence is a primary factor that leads to microtropia. The observation that microtropia may disappear and correspondence become normal in certain cases after occlusion therapy does not support his theory.

PARKS I was impressed by Dr. Lang's separation of microtropia into primary and secondary. The latter includes such easily identifiable causes as anisometropia. The former is a condition which is just "there" and is undoubtedly a genetic condition like hypermetropia. In such cases the inability to use both maculae together seems to be genetically determined.

FELLS Have you any patients who have had microtropia and have later lost the good eye? If so what happened to them?

LANG We have not seen any certain case, but we suspect it in one patient who had "never squinted", but has lost one eye. Fixation in the other eye was slightly eccentric, astigmatism was present, and the visual acuity could be improved only from 0.4 to 0.7.

FELLS How many of your patients with microtropia actually complained of visual difficulties?

LANG They have no difficulty in "binocular" reading, but only in uniocular reading with the microtropic eye.

JARDINE In contrast to Dr. Lang's experience, I have found that the treatment of small-angle microtropia by occlusion of the normal eye was rarely successful.

PARKS The result of occlusion depends essentially upon the age at which it is started. Generally the results in patients below 7 years of age are good, especially if the amblyopia is slight, as it usually is. In fact, some patients have no amblyopia. However, upon cessation of occlusion, the patient tends to return to using the favoured eye exclusively, and amblyopia tends to recur making intermittent occlusion the treatment of choice until it is finally terminated at 9 years of age, after which

it appears that the amblyopia tends not to recur. A study is being carried out at present to assess the validity of this statement.

LANG I agree that the prognosis of the treatment of amblyopia in primary microtropia is very good, far better than in cases with large angles of deviation. Occlusion gives better results than pleoptics. The prognosis is less good in cases of high anisometropia. We continue tapering-off occlusion until the child can read equally well with either eye, which usually means until the age of 10 years.

VON NOORDEN Gregerson and Rindziunski (1965), who had followed these children into their teens, showed that reversion was possible beyond the age of 8 or 9, up to the age of 12 or 13 years. Partial occlusion is a method of maintaining a sensitivity gradient between the fixing and non-fixing eye and it works well; full-time occlusion is hardly ever accepted by the older child.

BILLINGHURST I support the use of part-time occlusion. Very many good results have been obtained with this technique but no upper age limit should be set. Certain patients feel that they are doing something to help their own condition and therefore react well to it.

DOBINSON I understand Dr. Lang to say that his patients with microstrabismus had well-established binocular functions and therefore did not require orthoptic treatment. I have, however, seen cases of microstrabismus with symptoms from an associated convergence insufficiency which I have successfully treated with orthoptic exercises.

LANG I agree. Of course in these special circumstances they could be treated, but it is not usually necessary.

PARKS Convergence insufficiency can be associated with practically any type of squint.

NOLAN Has Dr. Lang any explanation for the finding that patients with anisometropic amblyopia of high degree do not respond well to amblyopia treatment. I have been treating these patients with contact lenses but not so far with much success, and I do not understand why they should not improve more.

LANG They do not improve because this is a deprivation amblyopia. What is the average age of the patients treated in this way?

NOLAN I have been using contact lenses in extremely young children, less than 4 years old, without any difficulty, but I have not achieved any improvement in vision.

BARNARD Dr. Arden and I have shown that, in anisometropic amblyopia, the peripheral and central retina of the amblyopic eye appear to work in rivalry to each other. When both the periphery and the central area of the amblyopic eye are stimulated, there is suppression, but if each is stimulated separately a response can be demonstrated. This might account for some of the poor responses to occlusion of the good eye. We therefore train the children to use the anisometropic eye for small objects only, because even if they do improve with conventional occlusion for distance, they very rapidly return to using their favoured eye as soon as occlusion is stopped.

STRONG A large-angle squint in which the visual acuity of the squinting eye has been only partly improved by occlusion will become a microtropia after surgery. You seem to infer that, if one persists with occlusion, these patients will continue to improve. Most surgeons in these circumstances would consider that they had obtained a "cure" and that further persistence with occlusion would serve no useful purpose. Do you agree?

LANG Amblyopia treatment in secondary microtropia is less useful than in primary, but further occlusion after surgery is worth trying.