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Microtropia Versus Bifoveal Fixation in Anisometropic Amblyopia

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Summary

Microtropia with identity is a unique condition in which amblyopes have parafoveal eccentric fixation in the amblyopic eye in either monocular or binocular viewing, plus a macular scotoma. The condition has previously been described in anisometropic amblyopia. The records of 55 consecutively presenting anisometropic amblyopes were scrutinised and the cases divided into microtropes or non-microtropes (bifoveal fixators). The features of the two groups were then compared to identify factors associated with microtropia and to determine whether microtropes or bifoveal patients responded better to amblyopia therapy. Results show that 45% of anisometropic amblyopes have microtropia rather than bifoveal fixation. There appears to be no association between the microtropic phenomenon and age, depth of amblyopia or amount of anisometropia. Bifoveal patients may respond better to amblyopia therapy although the difference between groups was not great. The sensitive period during which amblyopia may be treated is the same for each group.

In 1967 Helveston and von Noorden described a previously uncharacterised condition found in cases of apparently non-stra bismic amblyopia.[1] The patients all showed eccentric fixation in the amblyopic eye in either binocular or monocular state when tested with visuoscopy and the area used for fixation showed totally harmonious abnormal retinal correspondence with the fovea of the non-amblyopic eye. No abnormality was detected by cover test since the position of the preferred non-foveal point in the amblyopic eye remained constant even using that eye only, as during the ocular dissociation of the cover test. The diagnosis of the entity was confirmed by the demonstration of a presumed macular scotoma in the amblyopic eye by the 4 Dioptre prism test.[2] Almost all patients had anisometropic amblyopia. All showed peripheral fusion plus some degree of stereopsis. As patients with this unique combination of properties do not have bifoveal fixation they have, in effect, a very small angle strabismus. Accordingly Helveston and von Noorden termed this condition 'microtropia': for semantic reasons (*vide infra*) this name has been expanded to 'microtropia with identity'[3] to emphasise the finding that the angle of squint, angle of anomaly and angle of eccentricity are exactly equal. In this report the shorter term microtropia is used interchangeably with the longer expression microtropia with identity.

Whatever the terminology, the condition is a remarkable sensory adaptation. It does not, however, occur in all anisometropes. Although the characteristics were well described by that first report, no comparison exists between populations of those anisometropic amblyopes with the microtropic

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phenomenon and those who show normal bifoveal fixation. Without this, we know neither why only some patients with anisometropia have microtropia, nor whether the prognosis for treatment of amblyopia is affected.

Previously we have reported the characteristics of a group of pure anisometropic amblyopes with regard to the sensitive period for the treatment of this form of amblyopia.[4] In the present account we have re-analysed this group to compare certain features of those demonstrating a positive test for microtropia with those who had bifoveal fixation. We hoped to discover whether the development of microtropia was associated with age, extent of anisometropia or amblyopia, or with degree of stereopsis, and also whether the outcome of amblyopia therapy was affected. To support our previous study we have investigated whether there is any difference in the sensitive period for treatment of anisometropic amblyopia in microtropes.

Materials and Methods

The case notes of 55 patients presenting consecutively to the University Hospital, Nottingham with a diagnosis of pure anisometropic amblyopia were scrutinised.

Inclusion criteria

These were as described previously:[4] age of above 36 months to allow linear acuity tests, amblyopia of at least two Snellen lines difference between the eyes after spectacle correction and anisometropia of at least 1.00 DS.

Exclusion criteria

These were anisometropia of more than 5.50 DS (to exclude significant aniseikonia), astigmatism of more than 1 DC, any other ocular abnormality except microtropia and heterophoria, and any structural ocular or systemic abnormality.

All children had a full ophthalmic examination, refraction under mydriasis and orthoptic assessment. After a diagnosis was made, treatment started with full spectacle correction initially plus patching of the non-amblyopic eye if acuity failed to improve.

The group of patients was subdivided into amblyopes with bifoveal fixation (BFF) or with microtropia on the basis of the 4 Dioptre prism test. Figure 1 explains the basis of this test, which shows the presence of a scotoma immediately adjacent to fixation in the amblyopic eye for microtropic amblyopes. The complete test consists of interposing the prism in front of the non-amblyopic and amblyopic eye in turn and observing the movements of the fellow eye.

Details of the case notes were analysed and the age at presentation, amount of amblyopia at presentation, initial stereopsis, initial anisometropia and final acuity attained after treatment were compared between those with bifoveal fixation and microtopes.

Results

In the group of 55 anisometropic amblyopes 30 were designated bifoveal fixators while 25 showed the macular scotoma typical of microtropia on the 4 $\triangle$ prism test. The distribution of age at presentation in each group is shown in Figure 2, the mean age being 63.9 months for the BFF group (SD 15.8) and 61.3 months for the microtropes (SD 9.47). Unpaired Student t-test shows no significant difference between the age of the two groups at presentation ($p<0.05$).

The mean amount of anisometropia at presentation for the BFF group was 2.77 D (SD 1.28) compared wtih 2.84 D (SD 0.87) for microtropes (Figure 3). The difference is not statistically significant by unpaired t-test ($p>0.05$).

Similarly there is no significant difference between the amount of amblyopia at presentation in bifoveal or microtropic amblyopes, as shown in Figure 4 which displays the frequency of initial best corrected visual acuity for each group. The mean initial acuity was just better than 6/18 in the BFF group and just better than 6/24 for the microtropes: $p>0.05$ by unpaired t-test for the difference between the two groups.

Figure 5 shows the table used for Chi^2 test of association of stereopsis in each group. This is rather a crude analysis as stereopsis is very difficult to quantify accurately in the young. Given this caveat there appears to be no statistically significant association between stereopsis and either bifoveal fixation or microtropia.

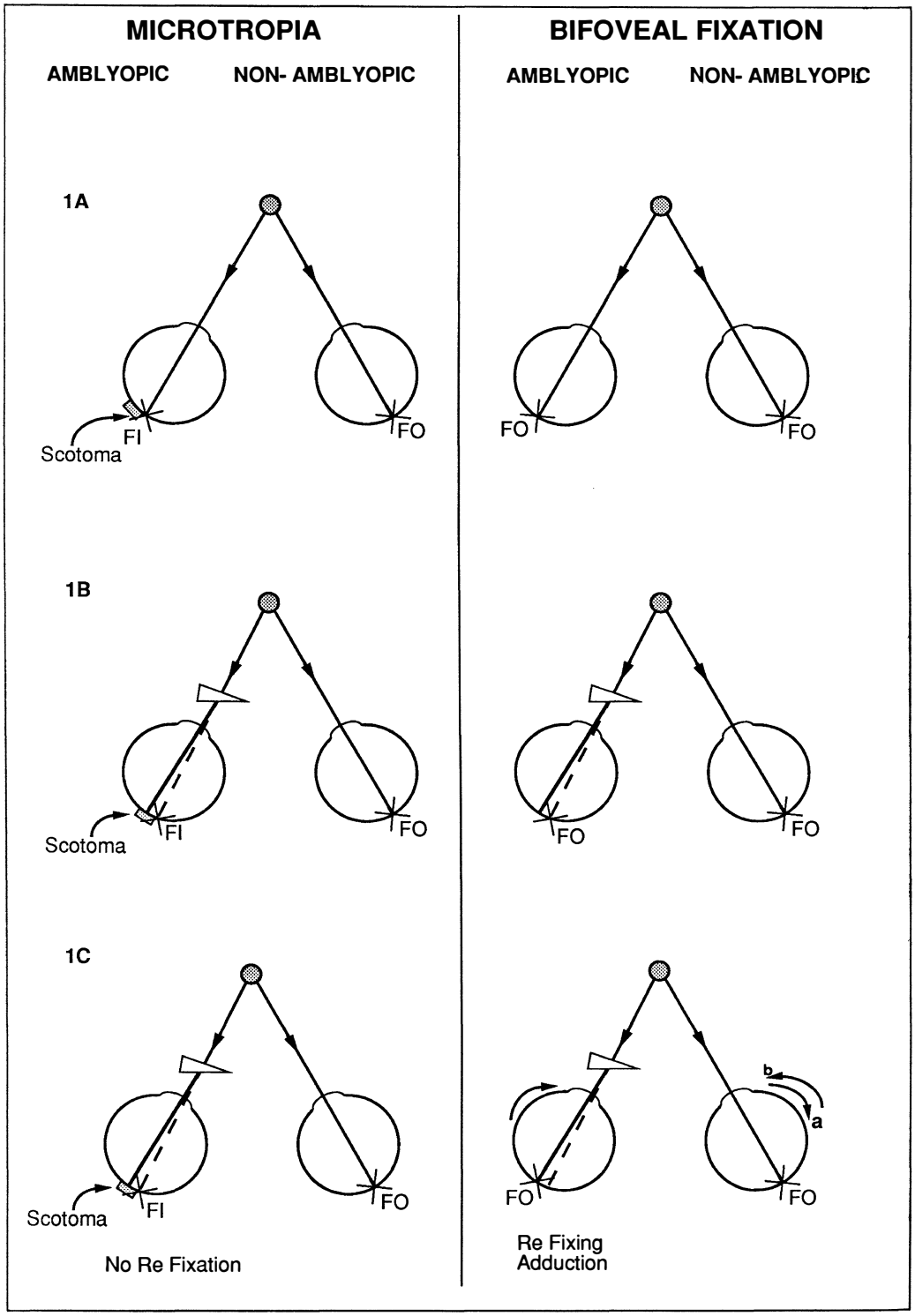
MICROTROPIA
AMBLYOPIC
NON- AMBLYOPIC
BIFOVEAL FIXATION
AMBLYOPIC
NON- AMBLYOPIC
1A
Scotoma
FI
FO
FO
FO
1B
Scotoma
FI
FO
FO
FO
1C
Scotoma
FI
FO
No Re Fixation
b
a
FO
FO
Re Fixing
Adduction

Fig. 1.

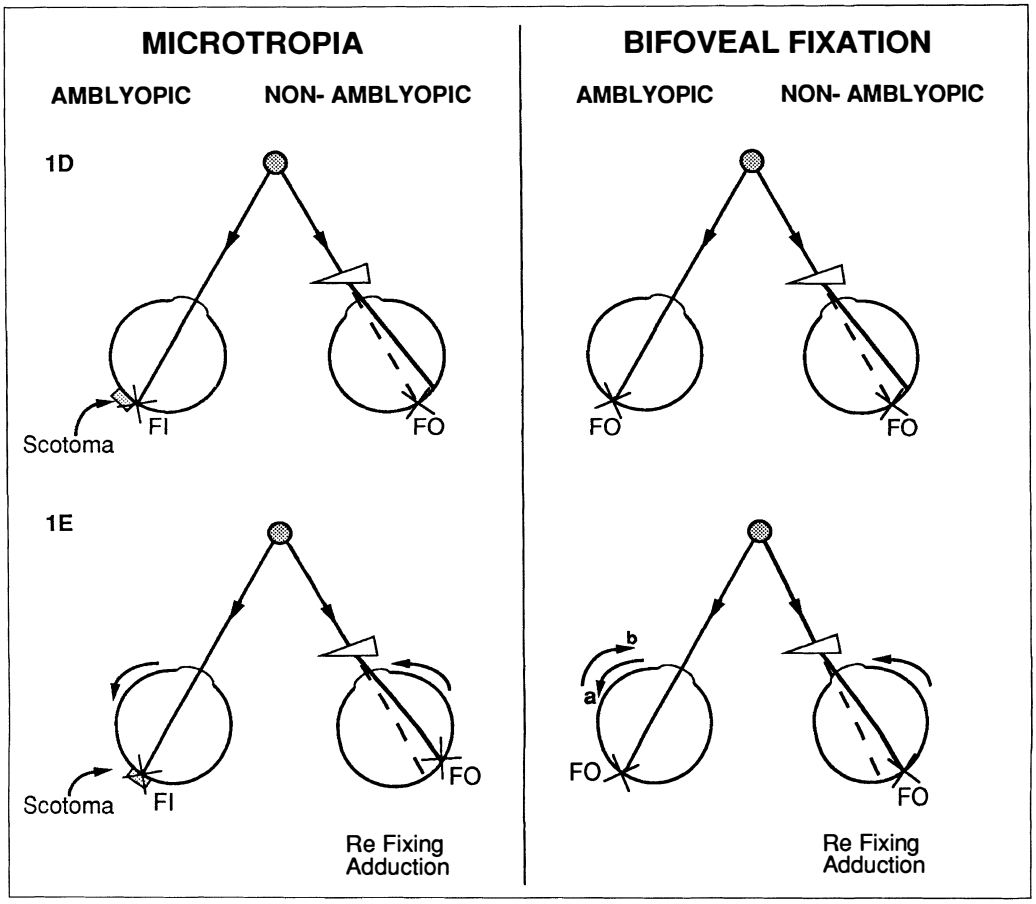

Fig. 1. *The 4 Dioptre prism test.*
1A: *Baseline*
Microtropic (MT) case. Target image falls on fovea (FO) of non-amblyopic eye and extra-foveal area (FI) of amblyopic eye Bifoveal (BFF) case. Image falls on fovea of both amblyopic and non-amblyopic eyes
1B: *4 △ prisms is interposed base out between amblyopic eye and target*
MT Case: target image is deviated laterally, into scotoma
BFF Case: target image is deviated laterally
1C: *Result*
MT case: no refixation movement is seen as the image falls into the scotoma.
BFF case: the deviated image causes an adduction movement in the amblyopic eye to realign the image and fovea (FO). The fellow eye undergoes a corresponding abduction (a) movement (by Hering's law) and an immediate following adduction (b) to regain foveal fixation.
1D: *4 △ prism is interposed base out between non-amblyopic eye and target*
BFF Case: Target image shifts laterally away from fovea
MT Case: Target image shifts laterally away from fovea
1E: *Result*
MT Case: Non-amblyopic eye has refixing adduction movement. Amblyopic eye simultaneously abducts (by Hering's law), and image then falls onto scotoma.
BFF Case: Non-amblyopic eye adducts to refixate, causing simultaneous abduction in amblyopic eye (a). This requires an immediate adduction movement to realign the fovea.

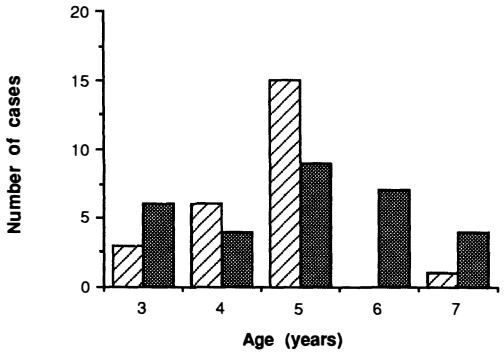

Fig. 2 *Age at presentation of microtropes (▨) and bifoveal fixators (■).*

Figure 6 demonstrates the outcome of treatment in each group by plotting the frequency of the final best corrected visual acuity attained. There is a small difference in the mean final acuity in each group, with the microtropic patients achieving a mean of just worse than 6/9 vision and bifoveal cases a mean of nearly 6/7.5. Unpaired t-test confirms that this difference is significant at the level $0.05>p<0.01$.

Figure 7 shows the improvement in acuity otained by treatment—because of the non-linearity of the Snellen chart improvement is plotted in minutes of arc of acuity. The two groups differ in the amount of improvement obtained, the mean being 2.6 minutes of arc for bifoveal cases and 3.17 minutes of arc for microtropes. This does not however reach the same level of significance ($p>0.05$ on unpaired t-test).

Figure 8 shows the relation between initial best corrected visual acuity and the age at presentation in both the BFF cases (Fig. 8a) and the microtropes (Fig. 8b). There is no apparent relationship between the two factors in either group and the Kendall rank correlation test confirms that age does not influence acuity at presentation (standardised normal deviate = 0.69 for BFF cases, 0.92 for microtropes – $p>0.05$ for either group).

Figure 9 plots the final acuity after treatment in each group against age at presentation. There is no apparent relationship between these factors by Kendall rank correlation coefficient ($p>0.05$, with standardised normal deviate = 0.83 for bifoveal cases and 0.60 for microtropes). Given that there is no relationship between initial vision and age at presentation, or between final vision and age, we can deduce that in both groups the outcome of amblyopia therapy does not depend on age at presentation, up to the limits of the ages of patients in each category in this study. Hence the sensitive period for treatment extends uniformly up to 82 months at least for microtropic anisometropic amblyopes and 91 months for bifoveal anisometropic amblyopes.

Discussion

Analysis of the literature related to the peculiar entity of 'microtropia of identity' is bedevelled by terminological ambiguity, which obscures the precise nature of the condition. Unfortunately the term 'microtropia' had already been used by Lang[5] before Helveston and von Noorden adopted it. Lang coined the word at an international strabismus symposium in 1966 to describe the condition of 'amblyopia with inconspicuous strabismus',

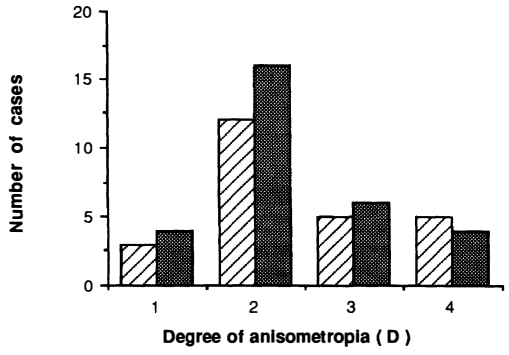

Fig. 3. *Degree of anisometropia in microtropes (▨) and bifoveal fixators (■).*

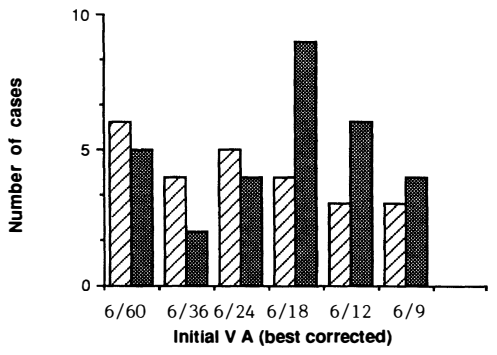

Fig. 4. *Initial best corrected visual acuity at presentation of microtropes (▨) and bifoveal fixators (■).*

Figure 5. *Stereopsis in microtopic and bifoveal fixating anisometropoic amblyopes*

	Stereopsis	*No stereopsis*	*Total*
Microtropes	18	7	25
Bifoveal Fixation	26	4	30
	44	11	55

by which was meant manifest strabismus of less than 5°, usually with harmonious anomalous retinal correspondence (ARC). He has continued to use the term in this way[6] and has classified such microtropia into primary without strabismus, primary hereditary or secondary after surgery or optical treatment. He suggests that the condition described by Helveston and von Noorden is included within his first group of primary microtropia without strabismus.

Parks also describes a condition of small angle strabismus which he originally termed monofixational phoria[7] and subsequently renamed monofixational syndrome after Lang's report emphasised that many of the patients in this group had a shift on cover test and should therefore more properly be termed tropias. Parks uses the term to include those cases of small angle strabismus (less than 8 △) where the deviation is greater on alternate cover than cover/uncover test, and where the deviation is made partially latent by suppression of the image falling on the deviated macular area.[8] He also includes the microtropic condition of Helveston and von Noorden in the 'monofixation syndrome.'

Parks seems justified in suggesting that the use of microtropia for a more precise condition than that for which it had already been coined is unwise. However, it is equally unhelpful to include the condition which Helveston and von Noorden had identified and precisely defined within the larger group of small angle squint. Furthermore, to dimiss the distinction between 'microtropia of identity' and other types of small angle strabismus as being merely a matter of semantics is inadequate.

In any consideration of strabismus both sensory and motor aspects must be specified. In conventional manifest strabismus when using both eyes the oculomotor system has a fundamental failure of alignment of the visual axes such that the target is not centred on the fovea of each eye. Unless diplopia is experienced sensory adaptation results. This may take the form of cerebral suppression of the image from one eye, or redesignation of the points on the retina of the strabismic eye to match the normal retina of the non-strabismic eye—i.e. abnormal retinal correspondence (ARC). Both these adaptations are *temporary* phenomena, being abandoned in favour of normal unsuppressed retinal values when viewing monocularly. Suppression and ARC are thus produced by visual system processing of the image from one non-aligned (strabismic) eye because of the simultaneous influence of visual input from the other eye with true alignment which thereby provides the higher cortical centres with fixed reference positional values for target objects. When using the strabismic eye alone there is no such influence, the previously strabismic eye

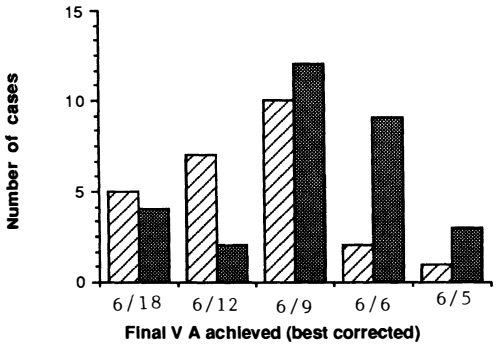

Fig. 6. *Best corrected post treatment visual acuity of microtropes (▨) or bifoveal fixators (■).*

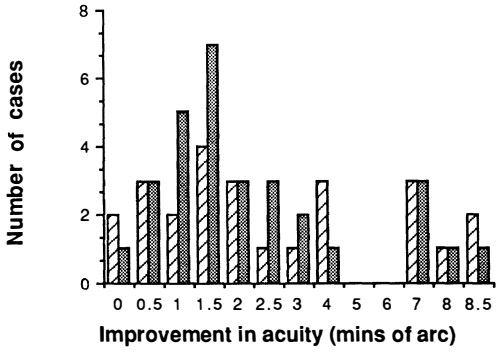

Fig. 7. *Improvement in visual acuity (in minutes of arc) after treatment of microtropes (▨) or bifoveal fixators (■).*

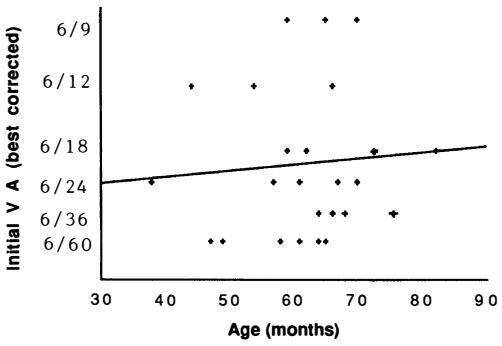

Fig. 8 (a)

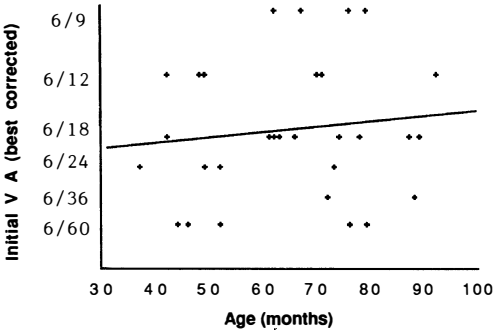

Fig 8 (b)

Fig. 8. *Initial best corrected visual acuity versus age at presentation for microtropes (8a) and bifoveal patients (8b).*

reverts to normal fovea centred retinal values, and the visual axis is redirected towards the target. This is the basis of the observed shift on the cover test, even when a strabismic eye has adopted ARC or suppression.

The condition of 'microtropia of identity' is *not* simply a strabismus too small to identify on cover test. It cannot be defined in this way because the amblyopic eye always uses the same preferred point of fixation on the retina under either monocular or binocular viewing. Consider both sensory and motor aspects of this state. There is once again a small degree of misalignment of the visual axes of the two eyes (providing the visual axis is defined as the line connecting the centre of the target to the fovea). Associated with this there is a permanent sensory change: in effect the topographical map of the outside world falling on the retina has permanently shifted so that the target is always directed to an area other than the anatomically defined area of the fovea. These changes are immutable whether reference input is provided from the other eye or not, in distinction to suppression and ARC where there is a temporary shift of the retinal values in the amblyopic eye to allow binocular viewing.

'Microtropia of identity' is the sole example of permanent change in the positional values of the retina and extra-ocular muscles to allow fair visual acuity and stereopsis in a visual system without underlying structural retinal abnormality. (A gross analogue of the condition is the eccentric fixation seen in severe macular disease.)[9] We do not know whether it is a congenital state or whether it develops after birth nor do we understand why it occurs. The theory so far advanced to explain the condition centres around the associated anisometropia: it is suggested that the macular scotoma develops in response to anisometropic amblyopia, that fixation is then assumed at the edge of this and that the extra-ocular muscles then redirect the eye such that target images fall on this new fixation spot. If

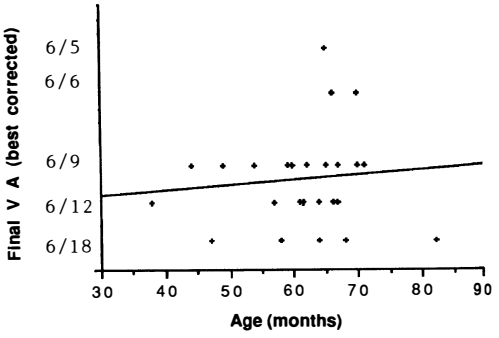

Fig. 9 (a)

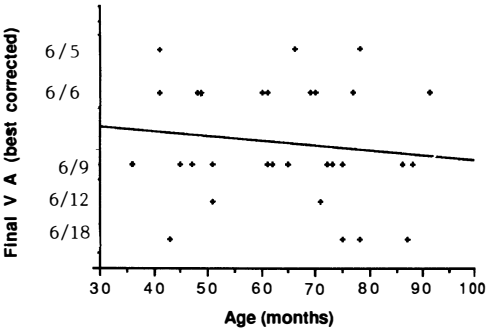

Fig. 9 (b)

Fig. 9. *Final acuity versus age at presentation for microtropes (9a) and bifoveal patients (9b).*

this is the correct explanation we must determine why a permanent scotoma develops in response to anisometropia and no other amblyogenic state. In addition, this mechanism would represent a reversal of the situation in conventional strabismus, since a sensory adaptation—i.e. a scotoma with consequent eccentric fixation—would be the primary event in the causation of the condition and not a secondary response to misaligned visual axes which we consider to be the sequence of events in most strabismus. The converse explanation for microtropia is also possible, where the initiating problem is of a minor misalignment of the visual axes and the permanent scotoma and eccentric fixation follow. This may seem unlikely, since many of these patients have stereopsis and the logical response to small angle deviation would be fusion and phoria rather than the complex process of redesignating retinal values. However, there is some evidence in animals reared with small angle prisms interposed in front of the eyes that visual cortical mapping can show a corresponding shift in the cortico-retinal projection.[10] It is possible that adaptation by fusion to correct a small angle squint or by sensory shift of cortical values may depend on the age of the individual concerned.

There is currently considerable interest in the mechanisms underlying the development of accurate and appropriate retinocortical connections in embryo. 'Microtropia with identity' provides the unique example of variation from the normal pattern of retinal map development without structural cause. In theoretical terms it is most important that it should not be regarded as a minor variant of small angle strabismus but rather, used as a model to show how the brain may redesign the retinal map of the outside world. It cannot be studied in animals as cover/uncover tests, 4 △ prism tests and visuoscopy are not possible, and so may only be investigated in the naturally occurring condition in man.[11] The precise characteristics of the human condition have been described in the original report but no study has been undertaken to identify whether this unusual state is adopted by anisometropic amblyopes only or whether it may also be found in amblyopia arising from astigmatism or occlusion. Furthermore, while all cases so far described have been anisometropic amblyopes, no attempt has previously been made to determine the frequency with which microtropia is associated with anisometropic amblyopia. Our series of patients indicates that microtropia with identity is neither an unusual nor an inevitable adaptation: 25 of the 55 consecutively recruited patients displayed the phenomenon, while 30 had bifoveal fixation. Why then do some children demonstrate microtropia and not others? It appears that age at presentation is not relevant to the development of microtropia (Fig. 2). As we believe that anisometropia is present from birth[4] it does not seem that microtropia develops in relation to the duration of amblyopia. The amount of anisometropia also does not influence whether any individual has bifoveal fixation or microtropia (Fig. 3). (In our series, as in others[1,12] microtropia always occurs in the more anisometropic, amblyopic eye.) The depth of amblyopia, represented by the initial best corrected visual acuity, is also irrelevant to determination of microtropia (Fig. 4). Two considerations arise from this. If a simple scotoma theory were adequate to explain the development of parafoveal fixation it would seem logical that microtropia would be more common in those with more dense amblyopia—our results do not show this. Also, the adoption of the parafoveal fixation of microtropia does not produce a more profound amblyopia than normal bifoveal vision.

The role of stereopsis in the development of microtropia remains speculative. Figure 5 shows the results of our analysis, but we do not feel that this is very reliable as it is known that stereoscopic testing in children is problematic,[13] and the preferred test for investigation of ARC—random dot stereograms—are particularly difficult in the young.[14]

Thus, while we have found that microtropia arises in 45% of anisometropic amblyopes we do not show a predilection for older, more anisometropic or more amblyopic individuals. What then may determine the condition? Lang[7] has speculated that there is a familial abnormal retinal correspondence, present from birth, while von Noorden[15] also suggests the presence of familial abnormal-

ities of binocular vision in microtropia. Neither explains why microtropia is unchanging in the monocular or binocular state or accounts for the so far invariable association with anisometropia. We have not investigated family members in our group and cannot therefore comment on these theories.

What is the effect of microtropia on the response to therapy of the amblyopic eye? This report shows that there is no effect on the visual acuity before treatment i.e. microtropia does not of itself cause a more profound amblyopia. Whether it may produce amblyopia which is more refractory to treatment is not entirely clear from our analysis, with Figure 6 showing slightly better acuities in the bifoveal group while Figure 7 shows no significant difference between the two groups. The Snellen chart is non linear such that the increments between lines ranges from 0.5 minutes of arc (6/6–6/9–6/12) to 1 minute of arc (6/12–6/18–6–24) to 2 minutes of arc (6/24–6/36) up to 4 minutes of arc (6/36–6/60). Using Snellen equivalents can make any statistical analysis of improvement in vision dubious, since an initial vision better than 6/60 but failing to reach 6/36 will be marked at the 6/60 level. Any subsequent improvement will then produce a spuriously large increase in acuity. This limits the value of Figure 7. Figure 6, showing the final acuity attained only, may be more helpful. In this respect we would concur with von Noorden that microtropia does not require treatment of itself, but allows conventional management of anisometropic amblyopia.

Despite our study the nature of microtropia remains unclear. We do not know why it is always associated with anisometropia. We do not understand the nature of the scotoma associated with the eccentric fixation, nor can we predict whether in time the condition may develop into full strabismus, as has been suggested.[7] While in practical terms the identification of microtropia may be of little consequence in the outcome of treatment of anisometropic amblyopia, it is vital that the 4 △ prism be performed and recorded for all cases of 'non-strabismic' amblyopia so that we may further our understanding of this unique condition.

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