Microtropia

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Abstract

Microtropia is an unilateral strabismus of less than 5°, usually with harmonious anomalous correspondence. Three forms may be distinguished: Primary constant, primary decompensating and consecutive microtropia.

In three instances microtropia is important for the ophthalmologist: In assessment of amblyopia apparently without strabismus, in evaluation of strabismus treatment results and in evaluation of hereditary factors in strabismus.

Amblyopia is more pronounced in cases with anisometropia and eccentric fixation, but usually responds well to occlusion treatment. Because of a typical ‘reading amblyopia’, treatment with alternating partial occlusion should be carried out until a child can read fluently with each eye.

It is estimated that about 1% of general population has a microstrabismus.

Primary microtropia is probably due to a primary sensorial defect, which predisposes to anomalous retinal correspondence. Primary microtropia may decompensate into a larger angle. After therapy, not a complete parallelism but a consecutive microtropia results.

Microtropia or microstrabismus may be defined as a manifest strabismus of less than five degrees, usually with harmonious anomalous retinal correspondence.

Three forms can be distinguished: Primary constant, primary decompensating and consecutive microtropia.

There are three situations in which the ophthalmologist may be confronted with microtropia: 1. in amblyopia without apparent strabismus (or so called congenital amblyopia); 2. in hereditary problems of strabismus; 3. in residual strabismus after surgery or after optical treatment. This form we have called ‘secondary’ microtropia. Since secondary has not only a temporal (after something) but also a causative (due to something) meaning, we prefer now to speak in these cases of ‘consecutive’ microtropia which means: Microtropia resulting after treatment. The term ‘secondary’ may be used for a microtropia due to some condition, e.g. secondary microtropia due to anisometropia.

Microtropia is by no means a rare condition. Out of 40,000 patients of our practice we found 1.177 microtropias. This corresponds to about 1% of microtropia in the general population.

Strabismus treatment has a history of about 150 years. But it is only in the last 30 years that interest has concentrated on very small angles. Microtropia and similar conditions were not mentioned by such well known early practitioners as Javal, Worth, Duane and Bielschowsky. Maddox in 1898 stated that very small angles were extremely rare, because
the natural tendency to fusion was much too strong as to allow small angles to exist!

Unfortunately, discussion has concentrated mainly on consecutive microtropia. Here a lot of propositions have been made on how to avoid this condition.

But instead of studying consecutive microtropias, where therapeutic measures have masked the original condition, one should concentrate on primary cases in which more insight may be gained into primary binocular pathology.

Again, primary microtropia is by no means a rare condition, when one is carefully looking for it. In a group of 805 microtropies we found 388 of the primary form (48%). Chamberlain and Caldwell found in 100 microtropia cases only 3 of the primary form, Bullock only 2 primary in 30 microtropias. This difference is probably due to the fact that these authors collected their cases mainly from an orthoptic department, whereas our cases are based on all my patients.

In primary cases a careful distinction regarding refraction should be made between children and adults. Recent studies by Lepard (2) and Lefftstra (1) and others have shown, that in unilateral strabismus there is a strong tendency for the amblyopic eye to remain hyperopic, whereas the leading eye becomes emmetropic or even myopic. Therefore anisometropia in adults may be the consequence rather than the cause of microtropia.

In the pathogenesis of large angle strabismus, primary decompensating microtropia plays an important role. Decompensation may be due to hyperopia, to a convergent position of rest, to amblyopia or to convergence excess. When, in a child with intermittent convergent squint one can show by uniaxial covering that in the apparent parallel position there is no parallelism but a microtropia, one clearly is dealing with decompensating microtropia. The same holds true, when in intermittent cases there is an eccentric fixation or when in large angles a very small angle of anomaly can be shown.

Since an anomalous correspondence pre-exists, adaptation to a new large angle is easily made. After therapy, e.g. after correction of hyperopia or surgery, the pre-existing microtropia shows up again. Therefore, consecutive microtropia cannot be blamed as being the consequence of insufficient therapy. Rather, consecutive microtropia is the best possible result in most cases of convergent strabismus (Fig. 1).

Amblyopia of course is a very important finding in microtropia. Here again, we can gain some insight into the nature of this condition. In 113 children with untreated amblyopia we found 78 patients with isometropia and 35 with anisometropia. Impairment of vision was spread over a broad range from 0,05 to 1,0. Vision was best in cases with isometropia and central fixation, the mean value here being 0,58. Vision was worst, as would be expected, in anisometropia with eccentric fixation, the mean value being 0,08. Isometric eccentric fixation and anisometropia with central fixation ranged between, 0,32 and 0,3 (Fig. 2). The prognosis of microtropic amblyopia is usually good, with the exception of cases with marked anisometropia. Simple occlusion is the best treatment.

![Fig. 1. Decompensation of primary microtropia.](image)

![Fig. 2. Amblyopia in 113 cases of untreated primary microtropia.](image)
When should treatment of amblyopia be stopped? In cured cases with occlusion abandoned before school age we found that distant and near vision with optotypes seemed to be good, whereas the reading faculty with the microtropic eye was definitely poor. This discrepancy we called 'reading amblyopia'. One has to bear in mind that microtropia cases are almost always unilateral and almost never spontaneously alternate. In using Amsler's charts we could show that reading difficulties are due to a monocular temporal scotoma which corresponds to the binocular zero point scotoma. We now therefore continue treatment with decreasing calibrated filters until the child can read equally well and fluently with each eye.

Primary microtropia can be found in a family together with other cases of large angle strabismus. Fig. 3 shows three sisters. The youngest and the eldest started to squint at the age of 3 years. The second girl never squinted, but at the age of 7 years a microtropia of the left eye was found with amblyopia of 0.2. Oggel & Rochels (3) recently could find in a family of three generations and 19 members, 10 primary microtropias and 3 large angle strabismus cases. This clearly shows the importance of microtropia in heredity strabismus. We think that no amblyopia, but the tendency to anomalous correspondence is influenced by some multifactorial hereditary way.

Diagnosis of microtropia can be made by the monolateral cover test which shows the manifest deviation and by ophthalmoscopical examination of fixation. Alternate cover may show an additional phoria. Anomalous retinal correspondence can be found ophthalmoscopically or with Bagolini lenses. We could even demonstrate the center of anomalous correspondence by a photographic method (Fig. 4).

To summarize one may say that microtropia may be regarded as a clue to the understanding of amblyopia, strabismus results and hereditary elements in strabismus.

Fig. 3. Two sisters with large angle tropia (decompensating microtropia?), one with primary constant microtropia.

Fig. 4. Primary microtropia of the right eye with eccentric fixation and anomalous correspondence. Vision 0.2. 
c) Fixation photography
d) Photography of the centre of anomalous correspondence.
e) Static perimetry of the amblyopic eye.
References


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