opinion is based upon the observation of morphological changes in the brain of cats and apes after a unilateral eyelid closure. However, after bilateral lid closure, these changes are far less marked. This leads us to suspect that monocular lid closure gives rise to suppression amblyopia rather than to deprivation amblyopia. This can be explained if one assumes that corresponding retinal points are in absolute rivalry with one another so that one eye suppresses the other, even when it is closed.

As for the treatment of ametropia, we are reluctant to prescribe correcting glasses because this places us in a dilemma: either consolidate or even increase an ametropia through spectacles, or risk a deprivation amblyopia. However, when visual acuity is defective or cannot be determined because the child is too young, the ametropia is corrected to avoid deprivation amblyopia.

Significant astigmatism is almost fully corrected and the spherical part is as much undercorrected as possible. In hypermetropia, the value found with retinoscopy without cycloplegia and the child accommodating is prescribed. The amount of hypermetropia the child can compensate during retinoscopy will not cause amblyopia. In myopia, the value found with retinoscopy with cycloplegia is reduced by two or three diopters, which gives a visual acuity that is adequate for near.

How many hours a day should the child wear the correction in order to avoid deprivation amblyopia? Nobody really knows and it is tempting to recommend a constant wear of the glasses. However, the child may become totally dependent on its correction. Orthophorization upon the glasses occurs and there is a risk of producing asthenopic complaints or even a squint on their removal. Furthermore, it is often observed that the correction needs to be strengthened regularly as if a latent hypermetropia is made manifest by the positive glasses or as if a spasm of accommodation is reinforced by negative glasses.

We prescribe a daily wearing of the correction during three hours or more, depending on the degree of ametropia and visual acuity. Throughout this period, alternate occlusion is carried out; one day the right eye and the other day the left eye. Once visual acuity can be measured and is normalized, occlusion is reduced to one hour a day.

4. Inappropriate prescription of prism glasses for obligate fixation disparity measured with the Pola test

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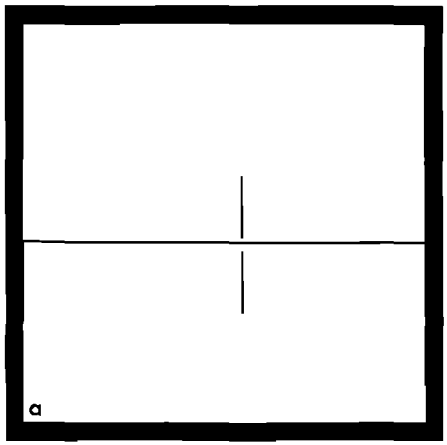
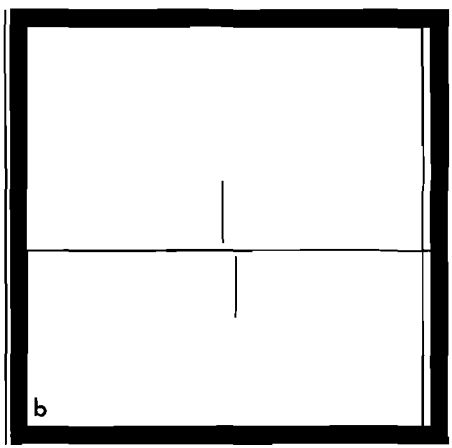
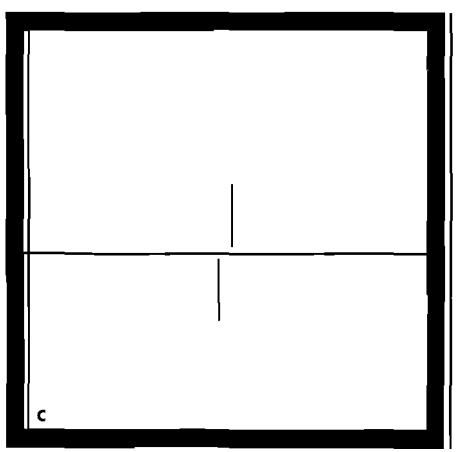
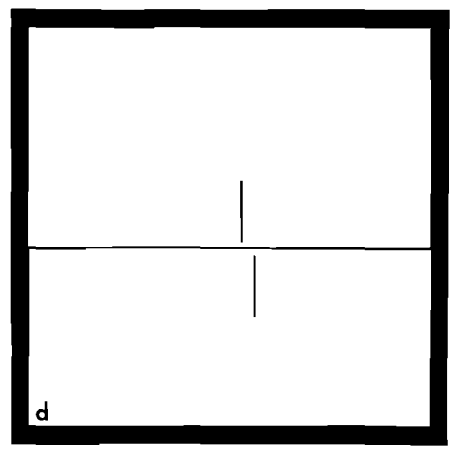
In the subjective measurement of fixation disparity (FD), the subject sees and fuses contours presented in the peripheral macular areas of both eyes ('fusion lock', a square for instance). The pointing direction of the foveolae of both eyes relative to each other is determined by means of two haploscopically seen objects presented in the central visual field (Fig. 1a). These are, for instance, two vertical nonius lines, one above the other, separated by a binocularly seen horizontal









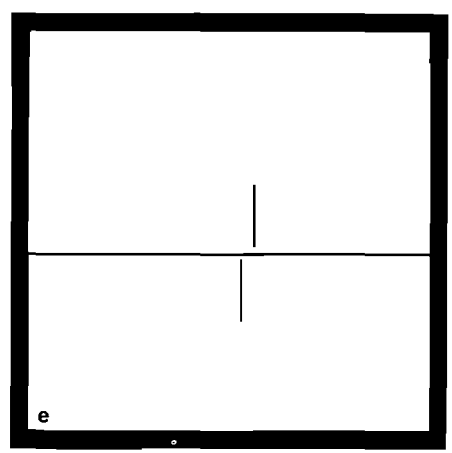


Fig. 1. (a) A schematic view of the pattern seen by the subject during the subjective measurement of FD. The large square is seen binocularly and serves as a fusion lock. The horizontal line is also seen binocularly, but does not induce fusion. The right eye sees the top vertical nonius line and the left eye the bottom vertical nonius line. The subject is instructed to shift the top nonius line horizontally until the two appear aligned. (b) In reality, the two may well be shifted, for instance, when base-in or (c) strong base-out prisms are given. Although most of the fusion is accomplished by vergence, part of it is sensory, *i.e.*, Panum's fusional area is utilized in the macular periphery to overcome a minimal diplopia, represented in this schematic diagram by doubling of the edges of the fusion lock. (d–e) In pathological (obligate or facultative) FD, the nonius lines are set shifted by the subject without fusional effort evoked by prisms.

line. One of the nonius lines can be moved horizontally by the subject, and this is done until the two nonius lines seem aligned to the patient (in the Pola test, a vertical line is seen by one, and a horizontal line is seen by the other eye, and the object is to see a cross with equal sides). For the haploscopically seeing subject the two nonius lines appear aligned, whereas in reality they may well be shifted. If they are shifted, it means that the foveolae of both eyes are not directed towards the same point on the screen, and FD is said to be present. FD is usually not larger than a few minarc. FD can be either physiological or pathological. Physiological FD occurs in normal subjects during fusional vergence evoked, for instance, by diverging or by converging prisms. The reason for the occurrence of physiological FD is, most likely, that during an excessive fusional effort, Panum's area in the peripheral retina is used to fuse the fusion lock, *i.e.*, a small portion of the fusion is not accomplished by a vergence eye movement but by sensory means (Figs. 1b–c). It has been found that this form of FD can change significantly if prisms are worn over longer periods. Pathological (obligate or facultative) FD occurs spontaneously, without fusional effort evoked by prisms, *i.e.*, although the subject fuses the fusion lock perfectly, the foveolae are not pointed towards the same point on the screen: A slight incongruence exists between the retinal correspondence in the peripheral macular areas and the retinal correspondence in the foveolae (Fig. 1d–e). In other words, perfect fusion of the fusion lock is obtained at another angle of vergence than perfect fusion of objects in the central visual field.

Some optometrists and ophthalmologists prescribe prisms to patients with heterophoria and asthenopia, guided by the FD measurement with the Pola test, and increase the strength of the prisms to the point that the measured FD becomes zero. As will be clear from the above, at that point the pathological FD is compensated by physiological FD. They report that, after the patient has been wearing the prisms for some time, usually a recurrence of FD is found, and stronger prisms have to be prescribed, sometimes several times, to reduce the FD to zero, in our opinion due to the fact that physiological FD is highly adaptable.

In the past three years, we were confronted with ten such patients, five of whom we summarize below. A 40-year-old female suffered from headaches after reading. From age 20 onwards, she had consulted an optician from time to time for complaints of asthenopia. He had given her weak prism glasses. Being discontent that the complaints could not be relieved completely she changed optician. The new optician used the Pola test, found fixation disparity and gave prism glasses up to 32 PD base temporal. She was referred to an ophthalmologist favoring the Pola test who performed a 5mm recession of the right medial rectus and a 5mm resection of the lateral rectus. Postoperatively, the optician found more fixation disparity and again increased prisms up to 26 PD base temporal. We found emmetropia, TNO stereo disparity 60″, and straight eyes on unilateral cover test after leaving the glasses off for two weeks. A second patient, a 28-year-old female, complained of headache and a central scotoma. She consulted an optician who gave, guided by the Pola test, prism glasses up to 39 PD base temporal; a referral for surgery was planned. We found myopia (S-3.50), TNO stereopsis of 120″ and an esophoria of 4 PD. Two other females, age 23 and 25, both with headaches, had a similar story with prisms up to 16 and 15 PD base temporal, respectively. A 36-year-old male with intermittent diplopia had ten pairs of prism glasses prescribed by an optician employing the Pola test in one year. We found straight eyes

on alternating cover, even after patching one eye for a weekend. Although he had TNO stereopsis of 60″, he had weak fusion and obligate fixation disparity. In the four cases where referral for surgery had been planned by the optician, the prism glasses did not have to be paid for by the patient, as these were supposed to be booked later on the bill for surgery as preoperative measure. After consulting us, these patients were confronted with a huge bill for prism glasses.

There has been speculation that obligate FD is causally related to heterophoria and asthenopia, and this may well be true, as perfect fusion is never possible in these patients: At one angle of vergence, fusion is perfect in the peripheral macular area, but at another angle fusion is perfect in the foveola. We have suggested previously that this may well be caused by slight growth asymmetries of the two eyes: If, for instance, after the age of ten, the foveola of one eye would shift during growth by only 0.01 mm relative to the peripheral macular area, pathological FD will inevitably be found. It will be clear from the above that only a transient relief of asthenopic complaints caused by obligate FD is possible by prescribing prisms guided by the Pola test.