Viewpoint • On the Etiology of Eccentric Fixation

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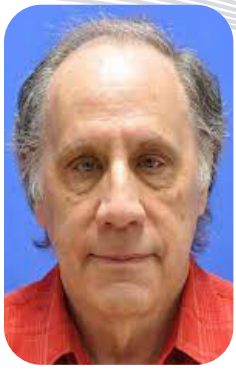

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ABSTRACT

Amblyopia is a complex visual condition consisting of abnormal and interactive sensory, motor, and perceptual aspects. One such abnormality is eccentric fixation (EF). A description of EF, along with its proposed etiology, will be considered.

Introduction

The condition of human amblyopia is a major public health concern, as it affects at least 2% of the U.S. population.[1] It is a complex phenomenon, with a range of abnormal and interactive sensory, motor, and perceptual components (Table 1).[2]

One of the most interesting, and important, visual abnormalities is eccentric fixation (EF), not to be confused with eccentric viewing (EV).[2] EF is an anomaly of monocular vision in which the time-average position of the fovea is off the object of regard.[2] That is, with the fellow eye fully occluded with a black eye patch, the amblyopic eye fixates, and localizes, a target as "straight-ahead" using a non-foveal point (i.e., abnormal oculocentric localization). EF is found in approximately 80% of amblyopic eyes[3] and is therefore a major factor to consider in its diagnosis and remediation. However, the question remains: "What is the etiology of EF?"

This important question has had a long history. One of the most interesting and logical proposals was expounded by Worth in 1943.[4] He believed that EF resulted from binocular suppression. Worth conceptualized the central, retino-cortical pathway as having a foveal "invagination" of sensory depression, with the immediately surrounding region exhibiting relatively higher visual acuity (Figure 1). Thus, one eccentrically fixated to maximize the residual visual acuity. This seemed like a plausible explanation. However, if true, then the EF point could logically occur anywhere along this rim of equi-acuity, with equal probability, and moreover vary its position over time (e.g., nasal retina one moment and temporal retina the next moment). However, EF is typically located at one relatively fixed retinal position (e.g., nasal 0.5 degrees), with some variability centered around this point.

In a careful experiment performed in 1978,[5] Worth's idea was disproven. Essentially, the researchers trained the amblyopic eye of adults to fixate centrally and steadily over a 5-10 hour pre-test period. Then, visual acuity was assessed across the horizontal retinal

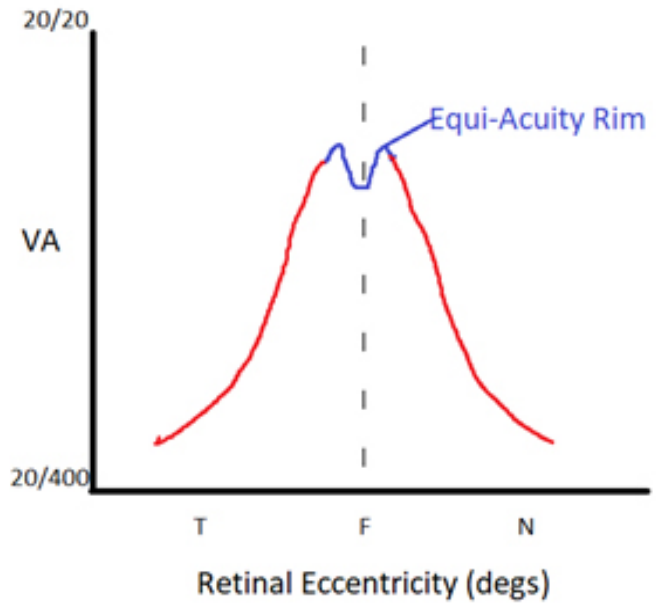

Figure 1. Retinal profile reflecting Worth's concept of binocular suppression effect and resultant central depression of visual acuity as a function of retinal eccentricity. Equi-visual acuity rim depicted by arrow. Symbols: F=fovea, T=temporal retina, and N=nasal retina.

Table 1. Major Sensory, Motor, and Perceptual Abnormalities in the Amblyopic Eye

• Reduced visual acuity
• Eccentric fixation
• Reduced stereoacuity
• Monocular spatial distortion
• Abnormal eye movements
• Reduced accommodation
• Impaired threshold and suprathreshold contrast perception
• Abnormal pupillary responses
• Abnormal visually evoked responses
• Abnormal visual-motor control

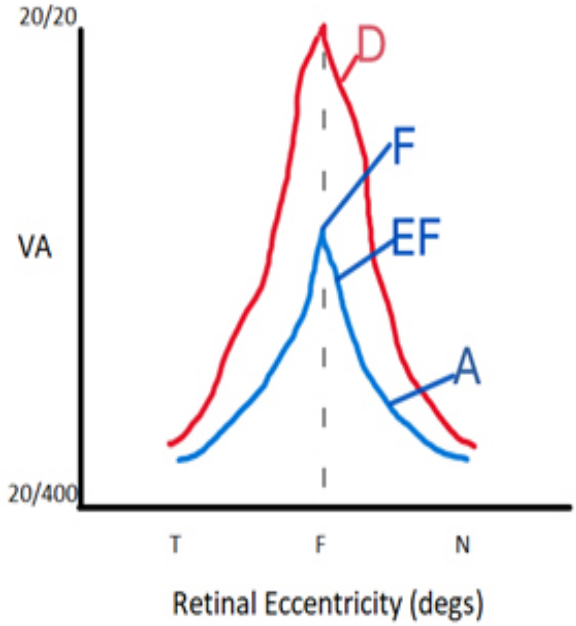

Figure 2. Results showing schematic representation of dissociation between reduced visual acuity and eccentric fixation in the strabismic amblyopic eye. Symbols: D=dominant/ normal eye, A=amblyopic eye, F=fovea, EF=eccentric fixation point, T=temporal retina, and N=nasal retina.

meridian at several retinal loci. They found visual acuity to be highest at the fovea, and NOT the EF point, with visual acuity overall depressed in the amblyopic eye as compared to the fellow eye (Figure2).

Given the above important finding, what might be the etiology, or underlying mechanism(s), of EF? The following is a proposed scenario. In strabismic amblyopia, where EF is typically the largest, there is also the presence of monocular spatial distortion (MSD) out to approximately 15 degrees of retinal eccentricity in all directions with respect to the fovea (Figure 3).[6] That is, the visual directional values are not organized in an orderly, sequential manner across the central retinal region (i.e., abnormal Lotze's "local signs"),[2] as found in normal eyes. In the dominant eye, the spatial pattern resembles a "bulls-eye" target of concentric circles, demonstrating a uniform and orderly spatial arrangement/oculocentric localization throughout the region. The fovea retains its zero, sensorimotor value of "straight-ahead" (i.e., normal oculocentric localization). In contrast, in the fellow strabismic amblyopic eye, there is directional distortion, with this being maximal in the central retinal region. Now the EF point, and NOT the fovea, acquires the zero, sensorimotor value with straight-ahead localization. This abnormality may reflect the underlying MSD. Such marked, central, directional distortion would shift the spatial "centroid," or zero point, from the fovea to the EF point. Thus, its proposed etiology: a central directional spatial shift,

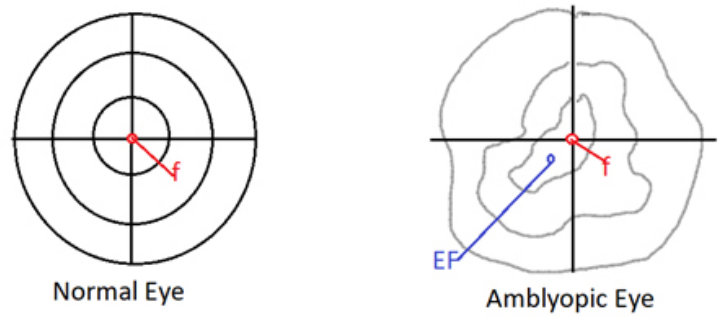

Figure 3. Results of experiment (schematic representation) showing orderly array of oculocentric spatial values in the normal eye, and abnormal distorted disarray of oculocentric values in the strabismic amblyopic eye. Symbols: f=fovea, EF=eccentric fixation point.

and a relatively simple scenario.

Lastly, how could this proposed mechanism be tested? One could assess visual acuity, EF, and MSD before and after "successful" treatment in strabismic amblyopes. If the MSD normalized, and fixation centralized, this finding would support the proposed idea. However, if not, then other factors/mechanisms would be involved. For example, the MSD might shift directionally and independently of the EF. Or the EF might become central in the absence of any change, or just partial change, in MSD. Other factors might be involved, such as contrast perception and eye movement patterns. Thus, experiments should be performed in the future to answer this most important clinical and basic science question.

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