

Monocular fixation with the optic nerve head: a case report

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Abstract

Purpose: To document and discuss the case of a patient with left esotropia (ET) who uses the left optic nerve head (ONH) for monocular 'fixation'.

Case report: The patient was an 80-year-old male with left ET from early childhood. Retinal tracking monocular fixation measurements with a Nidek MP-1 revealed stable fixation within the left ONH area. In an attempt to challenge the initial observation, further assessments of fixation were performed with a smaller target size and requiring various gaze positions. MP-1 fixation data showed remarkably stable monocular fixation ($\pm 1^\circ$ over 30 s) mostly within the left ONH for all the target sizes and positions of gaze tested. Additional clinical binocular evaluations showed concomitant left ET $\sim 28\Delta$, no movement with cover test regardless of fixation target and no significant monocular motility restrictions. Visuoscopy also revealed fixation at the left ONH. There was a strong family history of ET, but none of the other affected descendants tested ($n = 3$) demonstrated the same behaviour.

Conclusions: This is the first report documenting an abnormally developed monocular ocular motor system, with principal visual direction and zero retinomotor value shifted from the fovea to the ONH. We do not believe that there is any direct visual input from the ONH. The patient may use visual information obtained by glancing with peri-papillary areas to determine the target position (although this was largely ruled out), or obtain position information from the average luminance produced by scattered light around the ONH margin. The abnormal oculocentric direction might then be combined with extraretinal information (efferent copy or extraocular muscle proprioception) of the eye location in the orbit to stabilize the fixation. This patient does not have the blind spot syndrome (Swan, 1948). We propose the use of a retinal perimeter for documentation of eccentric fixation in strabismus.

Keywords: blind spot, eccentric fixation, esotropia, optic nerve head, principal visual direction, retinal perimeter, retinomotor value

Background

In early acquired strabismus, the visual system typically develops sensory adaptations to avoid the symptoms of diplopia and visual confusion (Pickwell, 1984), often reported as double vision. If these adaptations did not develop, the patient would experience diplopia, as the image of an object seen with the fovea of the

non-deviated eye would also be seen with a peripheral retinal area of the deviated eye (retinal images would not fall on corresponding points); and visual confusion, as the fovea of the deviated eye would receive an image different to that falling on the fovea of the non-deviated eye (two corresponding points would receive different images which would appear superimposed in the same direction). These phenomena are usually reported, when they occur in central retina, by patients who develop strabismus as adults (at an age when adaptations do not usually develop). While diplopia and visual confusion occur only under binocular viewing conditions (as a consequence of strabismus), the resultant adapting mechanisms may function under either binocular viewing conditions or may persist under monocular conditions.

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The sensory adaptive changes that may occur during binocular vision are suppression and abnormal retinal correspondence (ARC) (Von Noorden, 1969; Pickwell, 1984). A suppression scotoma may appear in the central retina of the deviated eye to avoid visual confusion and may extend [nasally in esotropia (ET)] to avoid diplopia (Pickwell, 1984). ARC is a binocular adaptation of the visual direction system, measurable only under binocular conditions, where objects are perceived as localized in the same direction in spite of their images falling on normally non-corresponding retinal locations (Pickwell, 1984).

These adaptation mechanisms may persist monocularly in the form of amblyopia or eccentric fixation. Amblyopia is the developmental impaired vision (with no organic cause) typified by reduced visual acuity (VA), distorted space perception and incorrect localization of objects in space (Bedell *et al.*, 1985; Skottun *et al.*, 1986; Fronius *et al.*, 2004). It may be a consequence of long-term binocular suppression that remains under monocular conditions – although some advocate that it may precede the strabismus (Schor, 1978). Strabismic amblyopia is most commonly associated with constant ET (Quah *et al.*, 1991). Eccentric fixation refers to abnormal monocular visual fixation with an area other than the fovea (Von Noorden, 1970; Kirschen and Flom, 1978; Pickwell, 1984). The eccentric fixation locus typically occurs within 5° ($\sim 10\Delta$) of the fovea (Flom *et al.*, 1980; Pickwell, 1981; Bedell *et al.*, 1990; Matilla *et al.*, 1995; Cleary and Thompson, 2001) and it may be stable or unstable.

The perceived direction of an object in space is determined by the retinal location it stimulates in conjunction with extraretinal information (e.g. eye position in orbit). Each retinal locus has a retinal visual direction associated with it. In a normal eye, the fovea has the principal visual direction (PVD) that is associated with the straight-ahead egocentric visual direction when the eye is in the primary position of gaze. Likewise, each retinal locus has a motor value, which means that it induces a fixed magnitude eye movement (saccadic) during the fixation reflex that brings the fovea to the direction of the object that stimulated that retinal location. The zero retinomotor value (ZRMV) (normally at the fovea) is the retinal point to which the eye makes a reflex saccadic re-fixation movement when presented with a peripheral stimulus (Ciuffreda, 1991; Steinman *et al.*, 2000). Usually, the new monocular fixation locus in eccentric fixation becomes associated with the PVD and the ZRMV (Von Noorden, 1970; Ciuffreda *et al.*, 1979; Bedell and Flom, 1981), although some authors argue that the PVD may remain unchanged (Pickwell, 1984).

Esotropia with an angle of deviation of about 15° ($\sim 28\Delta$) is not rare (Swan, 1948; De Muelenaere and

Hambresin, 1956; Guzzinati, 1956; Prizner, 1975; Olivier and Von Noorden, 1981; Birch *et al.*, 2004). In these cases, the fovea of the deviated eye is directed at an object whose image falls within the physiological blind spot of the non-deviated eye and vice versa. It has been suggested that this phenomenon may be a mechanism to avoid diplopia (Swan, 1947) and visual confusion. Swan (1948) defined the *blind spot syndrome* as a type of adaptation to ET with angle of deviation of about 15° , with occasional diplopia, normal retinal correspondence and normal VA. Others have reported cases of blind spot syndrome (De Muelenaere and Hambresin, 1956; Guzzinati, 1956; Uemura, 1964; McKenzie *et al.*, 1970; Prizner, 1975; Harrer, 1984). However, controversy surrounds the nature of these cases. The case we report here is not a case of blind spot syndrome (see Discussion section).

We have documented, using an objective method of assessing visual direction, a case of left ET wherein (under left monocular viewing) the left PVD and ZRMV are located within the left physiological blind spot.

Case report

Patient

The patient was an 80-year-old male with left ET and amblyopia documented to exist at least since age 3 (*Figure 1*) and with no reported change in VA or angle of deviation over time. He did not receive surgical, refractive or visual training treatment. No other health problems or illnesses occurring during birth or childhood were reported.

His right eye has reduced VA because of a non-arteritic anterior ischaemic optic neuropathy (NAION) diagnosed 4 years before our evaluation. His VA was normal, recorded Snellen 20/20 (6/6), in this eye prior to the NAION acute event.

Procedures

The patient came to our research laboratory to participate in a low vision research project. During the routine visual exam conducted prior to the experimental procedures, we conducted retinal fixation measurements with a Nidek MP-1 retinal perimeter (Nidek Technologies, Vigonza, Italy; software version available in 2004: MP1 SW1.4.1. SP1). Measurements were made monocularly, with the other eye occluded with an eye patch. During the retinal fixation exam, the patient was asked to look at a fixation target (a white cross) for 30 s. An auto-tracking system recorded the patient's retinal movements, frame by frame, using a reference retinal feature chosen by the examiner on the infrared (IR) retinal image. Thus, the fixation locus and its variability over

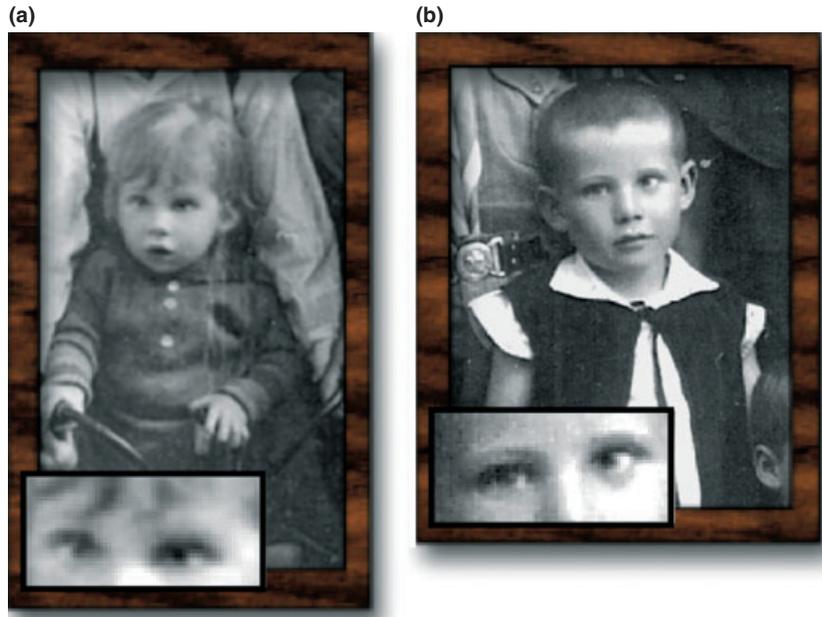


Figure 1. Patient's childhood pictures. (a) At 3 years of age and [b] at 5 years of age, both showing left ET.

time were evaluated. During 30 s net of retinal fixation recordings, 750 retinal fixation points were recorded (rate 25 Hz), skipping images lost as a result of blinking or other causes. The system allowed placement of the fixation cross into any desired position within the (45°) screen seen by the patient, enabling evaluation of fixation at different gaze positions. A colour retinal photograph was obtained and registered with the IR image so that the results were displayed on the colour image.

The initial fixation target used was a white cross of $2^\circ \times 2^\circ$ positioned centrally hence requiring straight ahead gaze position. Fixation data showed that the right eye fixated with an area slightly superior to the fovea (consistent with central field loss because of NAION, Figure 2a) and the left eye fixated within the optic nerve head (ONH) (Figure 2b).

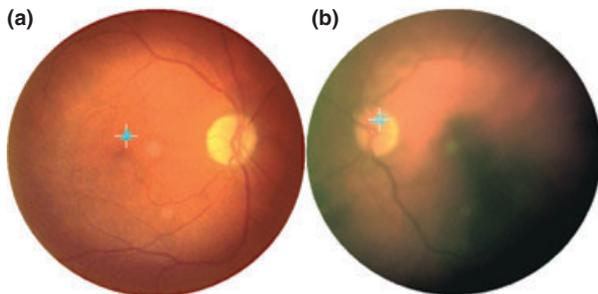


Figure 2. Fundus photographs and fixation data taken with the Nidek MP-1 during the routine exam [(a) right eye; (b) left eye] using a large fixation cross target ($2^\circ \times 2^\circ$). Measurements were performed monocularly. The patient was instructed to 'look at the centre of the cross'. The patient reported 'I do not see that it is a cross but I know where it is'. The left eye image quality is reduced because of the cataract.

To challenge this surprising observation, additional measurements were taken on the left eye. It was possible that the patient was not looking at the cross as instructed, but rather 'holding' (Steinman *et al.*, 1969) his eye in that straight ahead gaze position. The patient might have been using the peripherally-visible edge of the screen and the observation tubes as visual cues to help him direct his ONH to the centre of the screen (Sansbury *et al.*, 1973). It was also possible that, in order to fixate he was obtaining visual information from an area near the ONH. As the initial cross was large ($2^\circ \times 2^\circ$), the ends of two of the arms of the cross might have been visible in peripapillary retina. Therefore, additional measurements of fixation with the left eye were taken using a smaller fixation cross ($1^\circ \times 1^\circ$) which was positioned at various locations within the screen seen by the patient, requiring different gaze positions.

Findings

Fixation measurements with Nidek MP-1. The patient followed the target (white cross on a black background) easily into all positions of gaze with his left eye and at each position demonstrated a remarkably stable monocular fixation during the exam [the duration of the total fixation time required to record 30 s of valid data varied between the seven positions (55 ± 12 s)]. For most gaze positions, the fixation was stable (based on the instrument's manual definition – more than 75% of the fixation points are within a 2° circle) (Nidek MP-1 User Manual v1.5.0). On average, the gaze was within a 2° diameter circle for $73 \pm 11\%$ of the testing time (30 s) and within 4° for $97 \pm 3\%$ of the time.

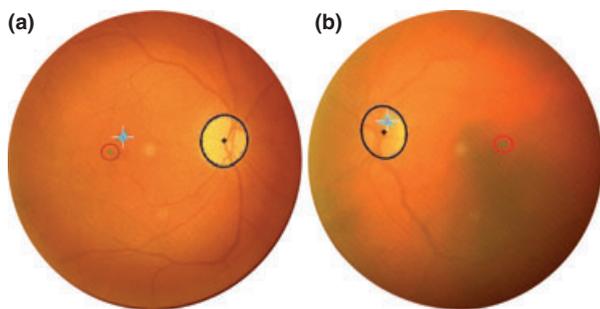


Figure 3. Fundus photographs and fixation data taken with the Nidek MP-1 and processed to calculate the distance from the centre of the disc to the position of the estimated fovea (green dot) and 95% confidence limits (red circle).

Custom software was used to calculate fixation eccentricity (*Figure 3*). The rim of the ONH (blue crosses) is manually marked (variability $\leq 0.13^\circ$) (Woods *et al.*, 2007) and the program fits an ellipse (black continuous line). The centre of the ellipse is considered as the centre of the ONH (black cross). The location of the fovea is calculated from the centre of the ONH (green dot). The red circle represents the 95% confidence limits. The white cross and the cluster of cyan dots represent the area used for fixation. The estimated fovea position is based on an average from studies with normally-sighted subjects (Hu *et al.*, 1994; Rohrschneider, 2004; Rohrschneider *et al.*, 2005; Timberlake *et al.*, 2005): 15.3° temporal and 1.5° inferior to the centre of the ONH (*Figure 3*).

With the $2^\circ \times 2^\circ$ target size, the right eye showed pericentral fixation, with the fixation locus situated 13.6° temporal and 0.3° superior to the centre of the ONH (*Figure 3a*), near but not at the fovea for that eye. Under the same conditions, the left eye used the superior-temporal part of the ONH (0.6° temporal and 1.5° superior to the centre of the ONH) as its fixation locus (*Figure 3b*).

With the smaller target size (white cross, $1^\circ \times 1^\circ$), the fixation cross was located within the ONH margins in the left eye for most gaze positions (*Figure 4*), except for the extreme left gaze positions when it lay outside. There was a small variation in the exact location of the left monocular fixation at various gaze positions, with a fixation location of $0.2^\circ \pm 1.5^\circ$ (mean ± 1 S.D.) temporally and $1.4^\circ \pm 0.5^\circ$ (mean ± 1 S.D.) superiorly from the centre of the ONH. In most gaze positions, the whole fixation cross was within the ONH during the entire recording time.

Potential spatial misalignment and head rotation have to be considered in the calculation of the fixation location. Since, unlike the original scanning laser ophthalmoscope (SLO), the Nidek MP-1 uses a different light source (IR) to track the fundus image than to present the stimuli, there is potential for misalignment



Figure 4. Fundus photographs and fixation data taken with the Nidek MP-1 on the left eye using a smaller fixation target ($1^\circ \times 1^\circ$) placed at various locations. Measurements were performed monocularly. The patient was instructed to 'look at the centre of the cross'. The patient reported 'I do not see that it is a cross but I know where it is'.

between these two systems (Woods *et al.*, 2007). When applying the correction factor proposed by Woods *et al.* (2007) for the misalignment found in the MP-1 used in this study, the fixation location for the left eye would be shifted further into the centre of the ONH (approximately 0.2° vertically and 0.7° horizontally). Therefore, with this systematic error correction, the whole fixation cross would be located within the ONH in all gaze positions.

Additional optometric examinations. External observation revealed no facial or postural anomalies. Visual acuity (single letter, Test Chart PRO2000; Thomson Software Solutions, Herts, UK; <http://www.thomson-software-solutions.com>) for the right eye was 6/30 (20/100) and for the left eye 6/480 (20/1600). Visual acuity for his left eye's fovea (measured by asking him to fixate at a target located at 15° to the left of the VA target position) was 3/387 (20/2580).

No movement was found in the cover/uncover or alternating cover tests regardless of the fixation target used (e.g. large letter, light source). Hirschberg and Krimsky tests were performed at various gaze positions and results showed left concomitant ET of $\sim 28\Delta$ (*Figure 5*). Ocular motility tests showed unrestricted monocular motility in each eye and reduced abduction in the left eye during binocular testing. Visuoscopy was carried out with a special large fixation target inside the ophthalmoscope and showed fixation of the right eye with an area near the fovea and at the ONH in the left eye. Finger-pointing showed the same (accurate)



Figure 5. Patient's eyes at time of evaluation (age 80 years) showing the left eye deviation when looking straight ahead at distance.

response when finger-pointing with the left eye (performed first) or the right eye. He also accurately pointed under a table at the directions of objects placed on the table while he could not see his hand, suggesting that egocentric mislocalizations are not currently present (Von Noorden *et al.*, 1970). His ability to navigate in a cluttered room with his right eye covered was unimpaired.

Other family members. An unusually strong family history of ET was observed in the patient's family

(Figure 6), with both his sons and one grandson having partially accommodative ET. Therefore, other family members were tested looking for a repetition of the phenomenon; however, none had an ET of about 15° (Table 1).

Discussion

We have documented, for the first time, a case of an abnormal monocular visual system adaptation to ET wherein its fixation locus, PVD and ZRMV have shifted and are now located within the ONH, a visually blind area. Indirect evidence that the patient is using the ONH for left eye fixation is the poor VA (20/1600) found. The size of the smallest letter seen by the patient corresponds to a letter that extends just beyond the limits of the physiological blind spot area (Figure 7). This supports the hypothesis that the physiological blind spot has been placed in the centre of the letter when asked to identify it. The abnormal oculocentric direction is subsequently combined with extraretinal information of the eye

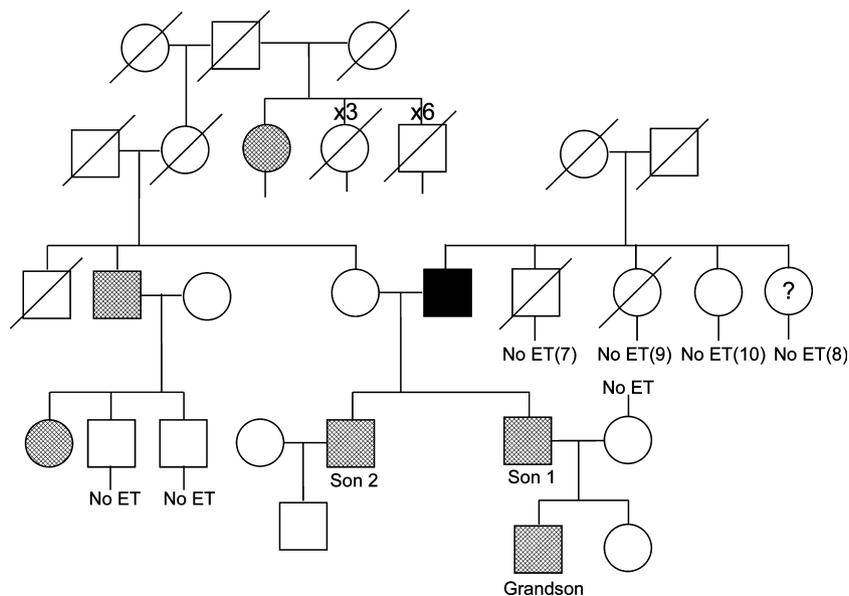


Figure 6. Familial genetic tree of the patient's descendants showing the patient (blacked filled square) and other members affected with left ET (shaded symbols). Males are represented by squares and females by circles, diagonal lines indicate a deceased family member. Some individuals not affected with ET were removed for simplification; the number of such individuals (descendants of the patients' siblings) is given in brackets. Descendants named as Son 1, Son 2 and Grandson are those family members tested in our lab (Table 1).

Table 1. Information about the three family members tested

Family member	Age (years)	Distance cover test (Δ)	Type of deviation	VA (single letter) of affected eye	ET treatment
Patient	80	No movement	Left ET	6/480 (20/1600)	None
Son 1	55	6 Δ (~3°)	Left ET	6/7.5 (20/25)	Refractive error correction single vision lenses, patching and visual training
Son 2	53	6 Δ (~3°)	Left ET	6/12 (20/40)	None
Grandson	24	15 Δ (~8°)	Left ET	6/6 (20/20)	Refractive error correction, including bifocal lenses

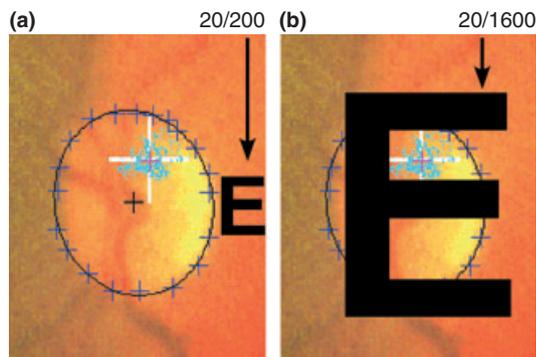


Figure 7. (a) Illustration of the retinal size for a 20/200 letter which represents the normal VA expected at the edges of the ONH. (b) Illustration of the retinal letter size corresponding to the patient's left eye VA (20/1600) which is just slightly larger than the ONH.

location in the orbit to give visual direction and egocentric position (Weir *et al.*, 2000).

We are not proposing that there is direct visual input from the ONH. We believe that the patient obtains visual input from areas surrounding the ONH to acquire information on the position of the stimulus, by using scattered light information (Faubert *et al.*, 1999), and then placing the physiological blind spot in the direction of the stimulus. Alternatively, it is possible to 'hold' the eye in a particular gaze direction for short periods of time, as shown in normally-sighted subjects following specific instructions (Steinman *et al.*, 1969). When doing so, the visual system does not concentrate on details and saccades are not elicited. We eliminated this possibility by evaluating his monocular fixation while we located the target at various gaze positions; results showed that the patient was moving his eye following the target and maintaining the ONH as his monocular fixation point. It seems that a complete reorientation of his left eye directionality mapping was developed resulting in the ONH now carrying the straight ahead value. It is, nevertheless, not obvious how the visual system could consistently, accurately and steadily direct the eye to a non-visual area.

We consider two possible aetiologies of the eccentric fixation:

(1) the patient may have simply developed 'by chance' 15° of eccentric fixation which corresponds to his 15° of ET and hence fixation is located at the ONH.

(2) The 15° angle of ET with the accompanying fixation and primary directionality within the ONH may have developed as an adapting mechanism to avoid visual confusion and diplopia. If a deviation had occurred (primary or as a consequence of reduced VA), and the adaptation mechanism of suppression (or ARC, but not likely in our case given the large deviation) had not been sufficiently deep to avoid diplopia and confusion, a different adaptation mechanism would have been

needed. The initial angle of the deviation may have been adjusted, perhaps because it was an accommodative ET with variable angle (at least two of the descendants are known to have had accommodative ET), so that the physiological blind spot was used as a central scotoma to avoid visual diplopia and visual confusion – at least centrally.

We believe that the changes in fixation and PVD occurring in this case did result from the need for a complete suppression scotoma in central retina to avoid diplopia and confusion at some point during development. This may be similar to cases of microtropia with identity which demonstrates eccentric fixation of equal magnitude to the angle of the strabismus (Von Noorden, 1969).

The use of the physiological blind spot as a mechanism to avoid diplopia and visual confusion was suggested by Swan (1947). Swan (1947) studied 296 patients with concomitant ET, 80 of whom had a deviation that would overlap the fovea of one eye with the physiological blind spot of the other eye in binocular conditions (he defined this as $ET = 12^\circ\text{--}17^\circ$). Of these 80 cases, seven cases seemed to use the physiological blind spot as the only mechanism to relieve diplopia because there was neither a suppression scotoma nor ARC. He identified some common characteristics of these cases including concomitant $ET = 12^\circ\text{--}17^\circ$ ($\sim 25\text{--}35\Delta$) at distance and near, occasional diplopia, normal VA in each eye (20/20), normal retinal correspondence and good fusion, and named this as the *blind spot syndrome*. None of these cases continued to fixate with the ONH under monocular conditions.

In 1948, Swan reported a larger series of these cases (102) that fall into the definition of blind spot syndrome, 75 of those reported occasional diplopia when asked (seldom volunteered). All showed normal retinal correspondence measured with after-images and the synoptophore, and had potentially good fusional capabilities.

Others have reported cases of blind spot syndrome as it was defined by Swan (Guzzinati, 1956; McKenzie *et al.*, 1970; Harrer, 1984). However, its existence has been disputed, particularly the absence of sensory adaptations as described by Swan (Olivier and Von Noorden, 1981). Oliver and von Noorden argued that Swan found normal retinal correspondence because the techniques he utilized were not sensitive enough, and that if Bagolini lenses had been used, ARC would have been found in all cases.

We do not claim to have presented here a case of blind spot syndrome and this case does not fulfil the definition of blind spot syndrome, primarily because the shift in fixation holds under monocular conditions, the patient has reduced VA and he has never reported diplopia.

In this case, we documented the retinal area used as the fixation locus and PVD in a patient with eccentric fixation. Retinal perimeters such as the Nidek MP-1 or SLOs are currently used to determine the preferred retinal locus for fixation in patients with macular disease. We propose the assessment of eccentric fixation in strabismus as an additional use of retinal perimeters with real-time eye tracking.

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References

- Bedell, H. E. and Flom, M. C. (1981) Monocular spatial distortion in strabismic amblyopia. *Invest. Ophthalmol. Vis. Sci.* **20**, 263.
- Bedell, H. E., Flom, M. C. and Barbeito, R. (1985) Spatial aberrations and acuity in strabismus and amblyopia. *Invest. Ophthalmol. Vis. Sci.* **26**, 909–916.
- Bedell, H. E., Yap, Y. L. and Flom, M. C. (1990) Fixational drift and nasal-temporal pursuit asymmetries in strabismic amblyopes. *Invest. Ophthalmol. Vis. Sci.* **31**, 968–976.
- Birch, E. E., Felius, J., Stager, D. R., Sr, Weakley, D. R., Jr and Bosworth, R. G. (2004) Pre-operative stability of infantile esotropia and post-operative outcome. *Am. J. Ophthalmol.* **138**, 1003–1009.
- Ciuffreda, K. J. (1991) *Amblyopia: Basic and Clinical Aspects*. Butterworth-Heinemann, Boston.
- Ciuffreda, K. J., Kenyon, R. V. and Stark, L. (1979) Fixational eye movements in amblyopia and strabismus. *J. Am. Optom. Assoc.* **50**, 1251–1258.
- Cleary, M. and Thompson, C. M. (2001) Diagnosis of eccentric fixation using a calibrated ophthalmoscope: defining clinically significant limits. *Ophthalmic Physiol. Opt.* **21**, 461–469.
- De Muelenaere, H. and Hambresin, L. (1956) [Influence of the blind spot on strabismic deviation]. *Bull. Soc. Belge Ophthalmol.* **112**, 174–178.
- Faubert, J., Diaconu, V., Ptito, M. and Ptito, A. (1999) Residual vision in the blind field of hemidecorticated humans predicted by a diffusion scatter model and selective spectral absorption of the human eye. *Vision Res.* **39**, 149–157.
- Flom, M. C., Kirschen, D. G. and Bedell, H. E. (1980) Control of unsteady, eccentric fixation in amblyopic eyes by auditory feedback of eye position. *Invest. Ophthalmol. Vis. Sci.* **19**, 1371–1381.
- Fronius, M., Sireteanu, R. and Zubcov, A. (2004) Deficits of spatial localization in children with strabismic amblyopia. *Graefes Arch. Clin. Exp. Ophthalmol.* **242**, 827–839.
- Guzzinati, G. C. (1956) [Blind spot syndrome]. *Ann. Ottalmol. Clin. Ocul.* **82**, 263–274.
- Harrer, S. (1984) [Blind spot syndrome in convergent strabismus of the horror-fusionis type]. *Klin. Monatsbl. Augenheilkd.* **185**, 126–127.
- Hu, S. Y., Schuchard, R. A., Fletcher, D. C. and Sabates, F. N. (1994) Physiological blind spot characteristics and position relative to retinal locus for fixation SLO testing. *Invest. Ophthalmol. Vis. Sci.* **35**, 00.
- Kirschen, D. G. and Flom, M. C. (1978) Visual acuity at different retinal loci of eccentrically fixating functional amblyopes. *Am. J. Optom. Physiol. Opt.* **55**, 144–150.
- Matilla, M. T., Pickwell, D. and Gilchrist, J. (1995) The effect of red and neutral density filters on the degree of eccentric fixation. *Ophthalmic Physiol. Opt.* **15**, 223–226.
- McKenzie, K., Iacobucci, I. and Armitage, M. D. (1970) The importance of the clinical investigation of blind-spot syndrome in adults. *Am. Orthopt. J.* **20**, 96–99.
- Olivier, P. and Von Noorden, G. K. (1981) The blind spot syndrome: does it exist? *J. Pediatr. Ophthalmol. Strabismus* **18**, 20–22.
- Pickwell, L. D. (1981) A suggestion for the origin of eccentric fixation. *Ophthalmic Physiol. Opt.* **1**, 55–57.
- Pickwell, D. (1984) *Binocular Vision Anomalies*. Butterworths, London.
- Prizner, S. (1975) Blind spot syndrome vs blind spot mechanism. *Am. Orthopt. J.* **25**, 76–78.
- Quah, B. L., Tay, M. T., Chew, S. J. and Lee, L. K. (1991) A study of amblyopia in 18–19 year old males. *Singapore Med. J.* **32**, 126–129.
- Rohrschneider, K. (2004) Determination of the location of the fovea on the fundus. *Invest. Ophthalmol. Vis. Sci.* **45**, 3257–3258.
- Rohrschneider, K., Springer, C., Bultmann, S. and Volcker, H. E. (2005) Microperimetry – comparison between the micro perimeter 1 and scanning laser ophthalmoscope – fundus perimetry. *Am. J. Ophthalmol.* **139**, 125–134.
- Sansbury, R. V., Skavenski, A. A., Haddad, G. M. and Steinman, R. M. (1973) Normal fixation of eccentric targets. *J. Opt. Soc. Am.* **63**, 612–614.
- Schor, C. (1978) A motor theory for monocular eccentric fixation of amblyopic eyes. *Am. J. Optom. Physiol. Opt.* **55**, 183–186.
- Skottun, B. C., Bradley, A. and Freeman, R. D. (1986) Orientation discrimination in amblyopia. *Invest. Ophthalmol. Vis. Sci.* **27**, 532–537.
- Steinman, R. M., Skavenski, A. A. and Sansbury, R. V. (1969) Effect of lens accommodation on holding the eye in place without saccades. *Vision Res.* **9**, 629–631.
- Steinman, S. B., Steinman, B. A. and Garzia, R. P. (2000) *Foundations of Binocular Vision: A Clinical Perspective*. McGraw-Hill Professional, New York.
- Swan, K. C. (1947) A squint syndrome. *Arch. Ophthalmol.* **37**, 149–154.
- Swan, K. C. (1948) The blind spot syndrome. *Arch. Ophthalmol.* **40**, 371–388.
- Timberlake, G. T., Sharma, M. K., Grose, S. A., Gobert, D. V., Gauch, J. M. and Maino, J. H. (2005) Retinal location of the preferred retinal locus (PRL) relative to the fovea in scanning laser ophthalmoscope (SLO) images. *Optom. Vis. Sci.* **82**, 177–185.
- Uemura, Y. (1964) [Blind spot esotropia]. *Nippon Ganka Gakkai Zasshi* **68**, 277–283.

- Von Noorden, G. K. (1969) The etiology and pathogenesis of fixation anomalies in strabismus. *Trans. Am. Ophthalmol. Soc.* **67**, 698–751.
- Von Noorden, G. K. (1970) Etiology and pathogenesis of fixation anomalies in strabismus. I. Relationship between eccentric fixation and anomalous retinal correspondence. *Am. J. Ophthalmol.* **69**, 210–222.
- Von Noorden, G. K., Awaya, S. and Romano, P. E. (1970) Past-pointing in paralytic strabismus. *Trans. Am. Ophthalmol. Soc.* **68**, 72–85.
- Weir, C. R., Cleary, M., Parks, S. and Dutton, G. N. (2000) Spatial localization in esotropia: does extraretinal eye position information change? *Invest. Ophthalmol. Vis. Sci.* **41**, 3782–3786.
- Woods, R. L., Vera-Diaz, F. A., Lichtenstein, L. and Peli, E. (2007) Spatial alignment of microperimeters (abstract). *Invest. Ophthalmol. Vis. Sci.* **48**, 144.