CASE REPORT

Clinical Management of Monofixation Syndrome due to Microtropia

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ABSTRACT

BACKGROUND

Monofixation syndrome is a binocular vision disorder characterized by peripheral fusion with a central suppression of one eye due to small-angle strabismus (microtropia) or anisometropic amblyopia. Reduced stereopsis and acuity are noted in the non-fixating eye. Although often left untreated due to good cosmetic appearance, central suppression and reduced stereopsis can affect visual performance, comfort, and depth perception.

CASE REPORT

EJ, a 17-year-old Caucasian female was previously treated for amblyopia of the left eye (OS) with glasses and patching since age 8 with no improvement to vision. EJ experienced the following symptoms: blurred vision at near when reading, blurred vision at far distances, eye pain and fatigue, difficulty copying from the board, difficulty seeing at night while driving, and poor depth perception. Entering uncorrected visual acuity was $20/20^{-2}$ OD and $20/50^{-2}$ OS with Snellen Letter Chart. Near uncorrected acuity was 20/20 OD and 20/200 OS with a reduced Snellen chart at 40 cm. Microtropia of 2 to 12 prism diopters of esotropia, eccentric fixation on visuoscopy, and central suppression on various tests were measured. EJ was diagnosed with monofixation syndrome, monocular esotropia of the left eye, strabismic amblyopia of the left eye, suppression of binocular vision (OS), and fusion with defective stereopsis. The patient completed 19 sessions of in-office vision therapy with the practice of home reinforcement activities between therapy sessions. Visual performance was reassessed after 10 and 19 sessions of vision training with improvements noted in acuity, accommodation, oculomotor accuracy and speed, eye alignment, fusional vergence skills, and stereopsis.

CONCLUSION

Optometric vision therapy can decrease suppression, improve central fusional ability, visual acuity, accommodative accuracy, and stereopsis in a patient with monofixation syndrome.

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INTRODUCTION

Monofixation syndrome is a disorder of normal binocular vision development in which the patient fixates centrally with one eye (monofixates) and suppresses the central vision of the other eye. Although cosmetic alignment is good, amblyopia develops due to the presence of a small angle strabismus of less than 10 prism diopters (Δ) or monocular retinal image blur due to anisometropia or opacity. Monofixation is classified by central foveal suppression in the strabismic eye of 5 degrees or less with retained peripheral binocular fusion. The central suppression scotoma prevents bifixation and reduces stereopsis.[1]

CASE REPORT

Initial Visit:

Patient EJ, a 17-year-old Caucasian female in the 11th grade presented on February 14, 2018, for a chief complaint of "lazy eye". She had the following symptoms: blurred vision at near when reading, blurred vision at far distances, eye pain and fatigue, difficulty copying from the board, difficulty seeing at night while driving, and poor depth perception. Her family history was positive for amblyopia (unknown type) in her father and paternal uncle. She was diagnosed with amblyopia of the left eye at age 8 and prescribed glasses and patching. She was told to discontinue glasses wear and patching when progress evaluations showed no improvement in clarity of vision of the left eye over the following two years.

EJ did not report any medical conditions, illnesses, surgeries, or injuries. She had no known allergies and was not taking any medications. The patient's hobbies included playing the piano and harp. She was an excellent student and described by her mother as focused, detailed, and organized. She reported no tobacco, alcohol, or drug use. The patient was awake and alert and oriented to time, place, and person. Pupils were equal, round, and reactive to light without an APD. Color vision was normal per Ishihara testing. Confrontation visual fields were full to finger count. The patient was symptomatic when completing the near point of convergence test. The measured break was 7.5 cm and recovery of 13 cm with tearing and reported discomfort. Slit-lamp examination with funduscopy and direct ophthalmoscopy showed clear and quiet structures with no structural abnormalities, inflammation, pigmentation, or hyperemia. Clinical examination findings are reported in Table 1.

Northeastern State University College of Optometry (NSUCO) protocols and scoring were used to assess pursuit and saccadic eye movement performance.[2,3] Even though published norms extend to age 14, standardized testing allows for baseline and progress assessment. Pursuit testing showed 3 fixation losses, intermittent slight head movements, and the ability to complete only 3 rotations. Saccade testing showed completion of 4 cycles and constant small undershoots. The Developmental Eye Movement (DEM) was completed for an objective measure of oculomotor ability and speed.[4]

The cycloplegic examination was deferred due to the need to return to school the same day. The patient returned 5 weeks later for follow-up. IOP with iCare tonometer was 12 mmHg OD and OS. Due to the patient's homework demand, the patient was dilated with 1% Tropicamide in place of the planned 1% Cyclopentolate. Testing did not show a significant latent hypermetropia, with +0.75 the maximum plus accepted OD and OS by subjective refraction and retinoscopy.[5] EJ had normal fundus health on Binocular Indirect Ophthalmoscopy with 20 D lens. There were no signs of macular elevation or scarring, optic nerve pallor or edema, C/D ratios of 0.25/0.25 OD and OS, normal vasculature, and no peripheral retina pigmentations, tears, or elevations.

Differential Diagnosis:

1. **Microtropia**[6]
   - A. Consecutive microtropia is a type of secondary esotropia resulting from prior surgical intervention. Patients who were over-corrected during surgery for exotropia or who have a history of strabismus surgery that only partially aligned congenital esotropia are the most common to develop monofixation syndrome.[7] EJ had no prior strabismus surgery ruling out post-surgical microtropia.
   - B. Primary microtropia is a deviation of less than 10Δ and occurs under 3 years of age. This type of esotropia is constant and unilateral. Testing and review of history were consistent with findings of a primary microesotropia.

Table 1: Examination Findings

Test	Initial Evaluation	Progress Evaluation #1 After 10 VT Sessions	Progress Evaluation #2 After 19 VT Sessions
Distance Snellen Acuity (20 feet) **Reduced Snellen Acuity (40 cm)** **Retinoscopy**	OD $20/20^{-2}$ OS $20/50^{-2}$	OD $20/20^{+2}$ OS $20/30^{-1}$	OD 20/20 OS 20/30+2
	OD 20/20 OS 20/200	OD 20/20 OS 20/25	OD 20/20 OS 20/25
	OD +0.50 (Distance) OS +0.50	OD +1.00 (MEM) OS +1.25	OD Plano-0.50x180 (D) OS +0.25-0.50x030
Subjective Refraction **Habitual Focal Distance** **Contour and Random Dot Stereopsis**	OD -0.50 DS 20/20 OS +0.50 DS $20/50^{+2}$	Not completed	OD Plano 20/20 OS Plano $20/30^{+2}$
	19" in primary gaze No head tilt, tip, or turn	16"	16"
	OS suppression Randot Wirt circles 200" No RDS was perceived on Randot RDS, Lang I, or Lang II.	Randot Wirt circles 50", Correct identification of all 500" RDS targets and 1/3 250" RDS	Randot Wirt circles 40", Correct identification of all 500" RDS targets and 1/3 250" RDS
Worth 4 Dot	Fusion response at 33 cm and left eye suppression at distance viewing at 6 M (light & dim)	Fusion response at 33 cm, left eye suppression at 2 M (light & dim)	Fusion from 33 cm to 6 M with Luster (light & dim)
Near Point of Convergence **NSUCO Pursuits**	7.5/13 cm	5/7.5, 2.5/5, 2.5/5 cm	To the nose (TTN) x 3
	17/20	20/20	20/20
NSUCO Saccades	17/20	20/20	20/20
Developmental Eye Movement Test (DEM)	Vertical: 26 seconds Horizontal: 28 seconds Ratio: 1.08 Errors: 0	Vertical: 30 seconds Horizontal: 30 seconds Ratio: 1.07 Errors: 0	Vertical: 28 seconds Horizontal: 27 seconds Ratio: 0.96 Errors: 0
+/-2.00 Facility **Suppression control: Polaroid bars and lenses**	OD 8 cpm OS 0 cpm OU suppression OS	OD 16 cpm OS 11 cpm OU: suppression OS	OD 22 cpm OS 18 cpm OU 11 cpm
13 BO/3 BI Fusion Facility (Polaroid suppression control)	Suppression OS	6 cpm	13 cpm
Computer Vergence Range (Vision Builder)	Suppression OS	158 BO/29 BI	190 BO/78 BI
Distance Cover Test	Recorded as 'no movement' due to the central suppression finding on the Worth 4 Dot test	Orthophoria	Orthophoria
Distance Phoria	Variable: 2Δ eso to 3Δ Exo/ Isophoria (Iso)	Ortho/Iso	1.5Δ Eso/Iso
Distance Fusional Vergence Range	Suppression OS, diplopia reported when out of the suppression zone at 10Δ BO and 8Δ BI	BO x/16/14 BI x/9/4	BO 19/26/17 BI x/14/7
Near Cover Test (40 cm)	LET: Cover test showed an increasing angle of deviation from 2Δ to 12Δ	2Δ esophoria	2Δ esophoria
Near Phoria	2 eso/iso on near Modified Thorington test. Suppression OS on von Graefe	2Δ esophoria/iso	2Δ eso/iso
Near Fusional Vergence Range PRA	BO break of 28 BI break of 14	BO x/29/22 BI x/14/9	BO x/21/16 BI x/12/10
	OD only: -1.50	-1.50 (doubles)/-1.25 recovery clear and single	-2.50 blur/-2.00 clear
NRA **Fused Cross Cylinder**	OD only +1.75	+2.75/+2.50	+2.75 blur/+2.50 clear
	OD only +1.25	+1.25	
Fixation Accuracy	Visuoscopy: OD: central and steady. OS: unstable, nasal eccentric fixation between 1-1.5Δ	Visuoscopy: OD: central and steady. OS: unsteady nasal eccentric fixation 1Δ After-image flash: Sees a cross offset left of center. MIT Haidinger's Brush: central and steady but OS image appears smaller.	Visuoscopy: Fixation was central, steady, maintained, and equal OD and OS MITT Haidinger's Brush: OD and OS able to align on dot targets

2. **Amblyopia**[8,9]

A. Refractive:

Monocular refractive amblyopia most commonly results from anisometropia in astigmatism (>1.50 D), hypermetropia (>1.00 D), or myopia (>3.50 D).[8] EJ did not have an amblyopiogenic amount of anisometropic refractive error.

B. Deprivation:

A blurred macular image from media opacity may cause a central suppression scotoma and mild to moderate amblyopia. EJ did not have media opacity (cataract, corneal opacity, ptosis, hyphema, vitreous opacification) on examination or per history.

C. Strabismic:

Strabismic amblyopia is the result of a constant unilateral strabismus present before 8 years of age that disrupts bifoveal fixation. Overlapping images of different objects in the same visual space cause visual confusion and the appearance of the same object in two separate lines of sight cause diplopia. The visual system adapts by active inhibition of the image from the deviated eye (suppression) and/or eccentric fixation (the eye is aligned to a non-foveal point). EJ showed shallow amblyopia, nasal eccentric fixation, and central suppression of the deviated left eye consistent with strabismic amblyopia.

3. **Macular abnormality**

Patients with a macular lesion will experience a central scotoma that is present under both binocular and monocular testing. In pediatric patients, causes of macular lesions could include solar retinopathy, laser pointer damage to the eye or macular hole with eccentric viewing. The most common cause of macular scars in pediatric patients is infection. Inflammation due to active infection of toxoplasma gondii, Histoplasma capsulatum, Toxocara Canis, rubella, cytomegalovirus, and herpes simplex virus may lead to pigmented macular scars.[10] The patient's history and retinal examination results ruled out macular pathology. The use of Ocular Coherence Tomography (OCT) could further aid in the identification of the presence or absence of macular structural abnormalities.

Assessment:

1. H50.42 Monofixation syndrome
2. H50.012 Monocular esotropia, left eye
3. H53.032 Strabismic amblyopia, left eye
4. H53.34 Suppression of Binocular Vision, OS
5. H53.32 Fusion with defective Stereopsis

Treatment options of lenses, prism, occlusion, and surgical management were considered. The purpose of a compensatory prism is to move an image to where the eye is pointing to allow sensory fusion and eliminate diplopia. Due to the suppression of the vision in the left eye, prism was not prescribed at this visit. Due to the small angle of esotropia and the presence of peripheral fusion, EJ was not referred for surgical consultation. Occlusion of the better-seeing eye is used to allow the amblyopic eye the opportunity to improve function and decrease active suppression. EJ had completed occlusion therapy as previously prescribed with no improvement per history.

Based on the symptoms and clinical findings, EJ was prescribed a program of optometric vision therapy designed to improve central fixation, accurate accommodation, eye movement efficiency, and binocular function. Anticipated treatment length was 30 +/-5 hours of in-office therapy with home reinforcement activities to be practiced 20 minutes per day. Vision therapy was scheduled weekly for 45-minute sessions. The prognosis was improved by the patient's developmental level, anticipated level of compliance, moderate amblyopia, and the presence of peripheral fusion.

Vision Therapy programming was based on the College of Optometrists in Vision Development (COVD) prescribed treatment regimen[11] and by following the management guidelines according to the American Optometric Association.[6,8] Therapy sessions included procedures sequenced as follows:

1. Development of adequate fusional vergence ranges, flexibility, and stability
2. Enhanced accommodative/convergence relationships
3. Integrated binocular function and information processing
4. Integrated binocular skills and accurate motor responses
5. Sensory integration
6. Increased visual accuracy and stamina

In-office optometric vision therapy and home reinforcement activities were prescribed in four general areas: binocular fusion, accommodation, tracking (fixation, pursuits, and saccades), and perceptual-motor integration.

Table 2: Optometric Vision Therapy Procedures

	Phase 1: Equating Monocular Skills & Fusion Range Development (Sessions 1-10)	**Phase 2: Fusion Range Extension (Sessions 11-19)**
Binocular	Red/Green (R/G) Tranaglyph R/G hidden pictures Red/Red rock and Franzblau +/-1.00 Vectograms: Quoits, Figure 8, Gem Mirror convergence/divergence Vivid Vision: Pepper Picker Optics Trainer VR: Fruit Ninja Red pegboard Brock String Vision Builder: Binocular Reading, Tennis VTS-4: Road Race Physiological diplopia Mirror superimposition Cooks Rings Sherman R/G Cards Cheiroscopic Tracing	Bernelloscope with Visicare fusion Red/Red rock +/-2.50 Aperture Ruler Cheiroscopic tracing: mazes Tranaglyph BI/BO/BI Prism flipper 4 BO/BI Vectogram: Clown, Spirangle, Double Quoits Rotoscope BO/BI Ranges Lifesaver Card VTS-4: Road Race BC Tranaglyph with R/G flipper Magic Eye Book Eccentric Circles Trellis Card Vision Builder: Jump Ductions
Accommodation	Pull-Aways Loose lens rock: -3.00D, -4.00D Jensen accommodative rock Biocular Ball Rock -4.00 D lens +/-1.50 Flipper with Word Search +/-2.00 Flipper with reading Near-Far Focus Charts Biocular Split Spirangle Focus Bull's Eye Rock	MFBF Focus Flexibility +/-2.50 Flipper Mental Minus -6.00D Near/Far Hart Chart Near/Far Tricky Finger Patterns Near/Far Anti-suppression chart BIM/BOP: +/-1.75 and 6 BO/BI
Tracking (Fixations, Pursuits, Saccades)	Eye Stretches Clock Fixations Optics Trainer: Asteroids Marsden Ball tracking Kirschner rotations Alphabet tracking Optics Trainer Saccades Saccadic Fixator (R/G Glasses) Space Fixator After-image flash Macular Integrity Tester and Trainer (MITT): mazes and dots Block Fixations Sanet Vision Integrator (SVI): Proactive and Reactive Saccades Wayne Directional Sequencer	VTS-4 Pursuits Red ink Groffman Tracking Marsden ball taps Kirschner rotations: Arrows Brock String rotations Red/Green Lite Track MITT: mazes Computerized Perceptual Therapy (CPT): Scan Accuvision SVI
Perceptual-Motor Integration	Striped pegboard with alternate cover Yoked prism bean bag toss (10 Δ) Lumosity Bird Watching Ballistic pointing	Parquetry Blink: shape/color/number match

The patient's visual performance goals included:

1. Stop losing place while reading or copying
2. Get schoolwork done with less effort
3. See more clearly
4. Copy school work more accurately

In addition, EJ wanted to feel prepared to take the Scholastic Aptitude Test (SAT). The doctor's goals included fixation accuracy OS, improved visual acuity OS, improved accommodative amplitude, accuracy, and facility, and development of sensory fusion and accurate motor alignment. Optometric Vision Therapy procedures completed are summarized in Table 2.

Progress Evaluation #1

The first progress evaluation was completed after 10 sessions over 4 months. EJ reported that she had more stamina and that school work was easier. Clinical findings are reported in Table 1. The patient was advised to continue optometric vision therapy.

Progress Evaluation #2

The second progress evaluation was completed after 19 sessions and 7 months. EJ's vision therapist stated "EJ no longer complained about losing her place when reading or copying. Focus flexibility, tracking speed and accuracy, and random dot stereopsis on the VTS-4 3D computer program were improved. EJ sustained good posture, accuracy, and speed when reading. All initial symptoms had been resolved, with therapy activities performed successfully and with comfort. EJ mastered free-space fusion techniques and saw magic eye images requiring divergence. She had started the 12th grade and had no difficulties in the classroom." EJ reported greatly improved clarity of vision, ability to focus, improved comfort, and improved depth perception. Clinical findings are reported in Table 1.

Maintenance therapy was prescribed and included the Lifesaver Card and Eccentric Circles. EJ returned to her primary care optometrist for routine annual eye examinations. Follow-up communications at 1 year, 20 months, and 2.5 years post-therapy reported that EJ maintained acuity and stereopsis gains, excelled in high school and college academically, and attributed her improved visual performance to vision therapy.

DISCUSSION

Variations from expected relating to the patient's presentation were inconclusive test results on the 4 BO prism and Bagolini striated lens tests. At EJ's initial evaluation, she was only able to note diplopia findings when the image shift created by prism was outside her central suppression zone. This required a minimum of 10Δ BO and 8Δ BI. Clinically version and vergence movements were not observed with 4 BO prism testing over either eye. The Bagolini test was attempted with patient EJ at her initial evaluation, but she was uncertain and unable to describe what she perceived.

Monofixation Syndrome

Monofixation syndrome is defined by a facultative scotoma. The central suppression scotoma is only present under binocular conditions. Active inhibition of the central 5+/- degrees of vision occurs only where and when binocular disparity exists. To diagnose monofixation syndrome, a binocular disparity must be present that is amblyogenic for central fusion but not to a degree that causes suppression of the entire eye. One of the following etiologies is required for diagnosis: small-angle constant strabismus of less than 10Δ, anisometropia, unilateral astigmatism, or partial media opacity such as a cataract.

The term monofixation syndrome was described by Parks in 1969, after first calling the syndrome "monofixational phoria."[12] Parks' theory was that peripheral fusion enabled a lower magnitude of misalignment on the unilateral cover test and when this fusion lock was broken with an alternate cover test, the esodeviation increased in amplitude.

The prevalence of monofixation syndrome in the United States is 1%. In families with a history of congenital esotropia, the prevalence increases to 9%.[7] The presence of a small angle micro-esotropia deviation is the most common cause of monofixation syndrome. Other potential causes include anisometropic hypermetropia, meridional anisometropia from unilateral astigmatism, or unilateral partial media opacity such as infantile cataract lens opacity. The patient will typically have fusional vergence eye movements, but a small angle strabismus or underlying phoria will manifest during cover testing.

Monofixation should be considered if visual acuity is reduced but in the range of 20/100 or better, stereopsis is reduced, and strabismus amplitude is small.[1,13] Clinical findings of monofixation syndrome should identify central foveal suppression under binocular conditions with intact peripheral fusion and reduced or absent random dot stereopsis. One such test is the 4-prism diopter base-out test. When a base-out prism is introduced in front of one eye, the image shift causes the eyes to make a version in the direction of the prism apex, followed by a convergence movement. If the patient has monofixation syndrome, the versional movement will occur, but no vergence will be made due to central suppression in the affected eye. This testing is clinically useful when the suppression zone is 4Δ or smaller.

Symptoms reported in patients with Monofixation syndrome are listed in Table 3.

Table 3: Symptoms of Monofixation Syndrome

1. Reduced efficiency, accuracy, and consistency of work
2. Diminished performance with time on task
3. Inconsistent depth judgment
4. Eye pain
5. Headaches
6. Difficulty sustaining near visual function
7. Avoidance of visually demanding tasks
8. Inaccurate eye-hand coordination
9. Transient blur
10. Transpositions when copying
11. Avoidance of eye contact
12. Diplopia or a tendency to cover an eye to prevent diplopia
13. Abnormal posture
14. Spatial disorientation
15. Photophobia
16. Reduced visual attention
17. Fatigue
18. Dizziness
19. Motion sickness
20. Incoordination
21. Asthenopia

Decision-making

Ocular pathology must be first be ruled out by assessment of the retina and a review of the patient's history. The examination would need to specifically exclude a structural issue such as a macular scar, retinal dystrophy, and central serous maculopathy. Refraction and binocular vision testing ascertains amblyogenic factors. Visuoscopy or Macula Integrity Tester (MIT) testing specifies the direction and amplitude of eccentric fixation (14). Sensory fusion and oculomotor alignment testing then allows the clinician to diagnose monofixation syndrome.[8,15] This testing could be completed with a Synoptophore or by completing diagnostic testing as described in Figure 1.

1. Cover Testing

Cover testing often shows very small angle strabismus, requiring careful observation on the unilateral cover test. The alternating cover test may not elicit an alignment deviation in up to one-third of patients. When alternate cover testing does break the peripheral fusion lock, the angle of deviation becomes more apparent in two-thirds of patients showing a 2-3 times greater amplitude of heterophoria.[4]

2. Worth 4-dot

The Worth 4 dot flashlight with 6 mm dots spaced within a 34 mm diameter circle measures 6 degrees apart when viewed at 33 cm.[12] The common suppression scotoma found in monofixation syndrome is less than 5 degrees, allowing a fusion response at standard near testing distance. The suppression scotoma becomes apparent as the Worth 4-dot target is moved to a greater viewing distance and the

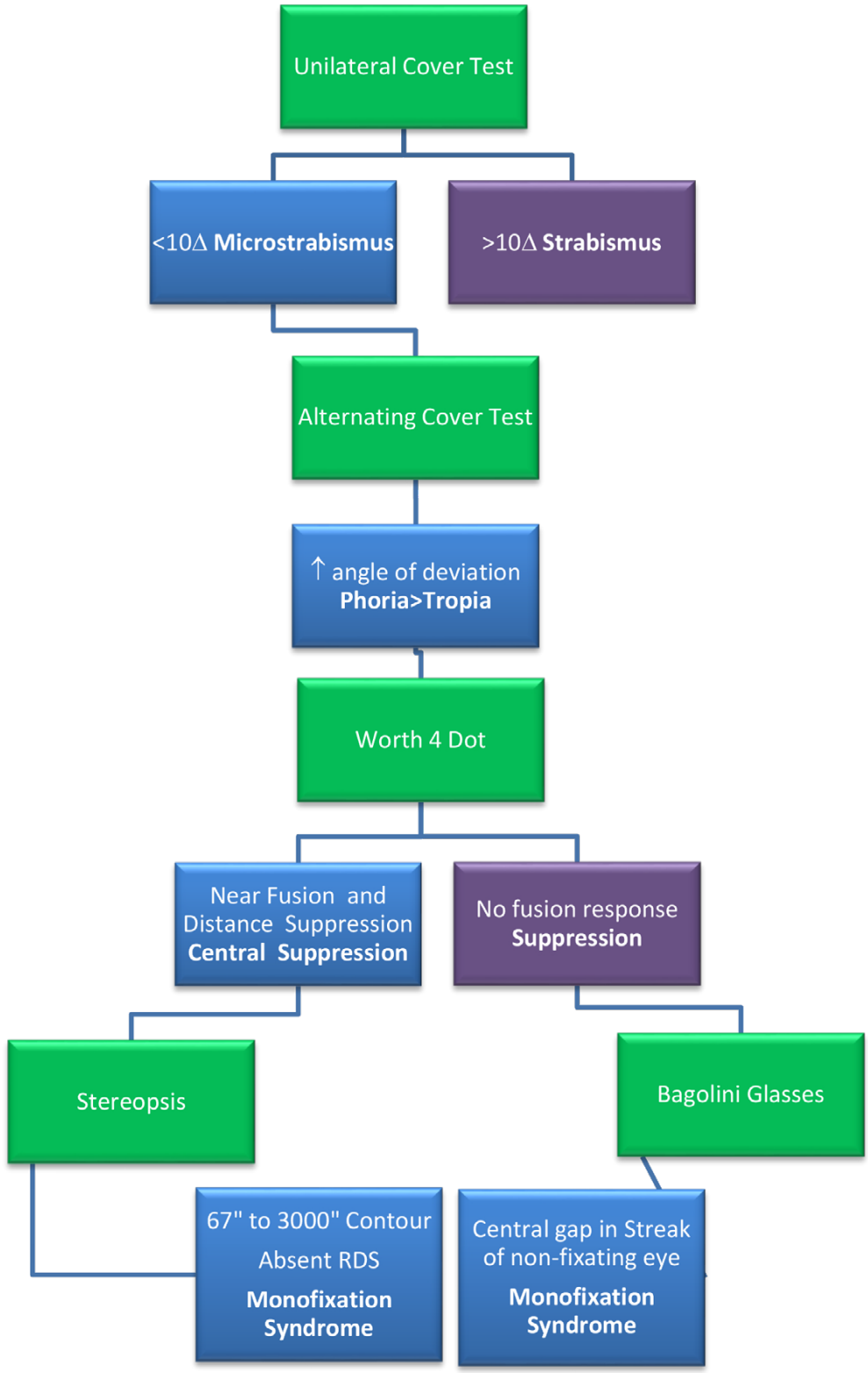


Figure 1. Monofixation Diagnosis Flow Chart[12]

angular size of the target becomes smaller. At 0.5 M, the target size is 4 degrees, at 1.0 M, the target size is 2 degrees, and at 2 M, the target size is 1 degree. Testing using a target that can be varied in size can also be used to identify a central suppression scotoma.[15] Using an iPad target with a central 0.6 degree (4 mm at 40 cm), 2-degree target (16 mm at 40 cm), and 5-degree target (35 mm at 40 cm) can also reliably identify central suppression.[16]

3. Bagolini Striated Lens Testing

Bagolini striated lenses are used in the evaluation of fusion, suppression, diplopia, and

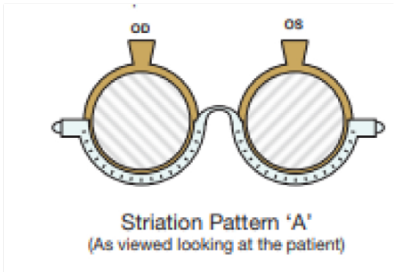
retinal correspondence. A missing diagonal streak indicates the suppression of an eye. The presence of a gap in the light streak centrally indicates binocular central suppression. Central suppression with the retained peripheral fusion of the affected eye is expected in cases of monofixation syndrome as viewed in Figure 2.[26]

4. Stereopsis Testing

Central suppression scotoma may only manifest during binocular testing and measure between 2-5 degrees in size. When measured monocularly, the patient may no longer suppress this central area as active suppression occurs when binocular disparity is present. Random dot stereopsis may be absent because the receptive fields of the macula are small and the disparity of image clarity or retinal image position can lead to monocular central suppression of the affected eye. Contour stereoacuity typically measures between 67-3000 arc seconds.[17,18,27] The receptive fields of the peripheral retina are larger and require less accuracy to stimulate corresponding fields to create binocular fusion. The suppression scotoma is proportional to the degree of image disparity. For larger angle strabismus, the peripheral fields also show disparity and the patient is unable to fuse the images leading to suppression of input from the entire eye and amblyopia.

Treatment Protocol

Treatment for monofixation syndrome includes addressing the underlying cause of central suppression. This includes addressing the refractive error with glasses or contact lenses



Patient points to Figure:	Patient is wearing Striation Pattern A
1	Fusion
2	Suppression OS
3	Suppression OD
4	Fusion w/ Central suppression OS
5	Fusion w/ Central suppression OD
6	Uncrossed diplopia
7	Crossed diplopia

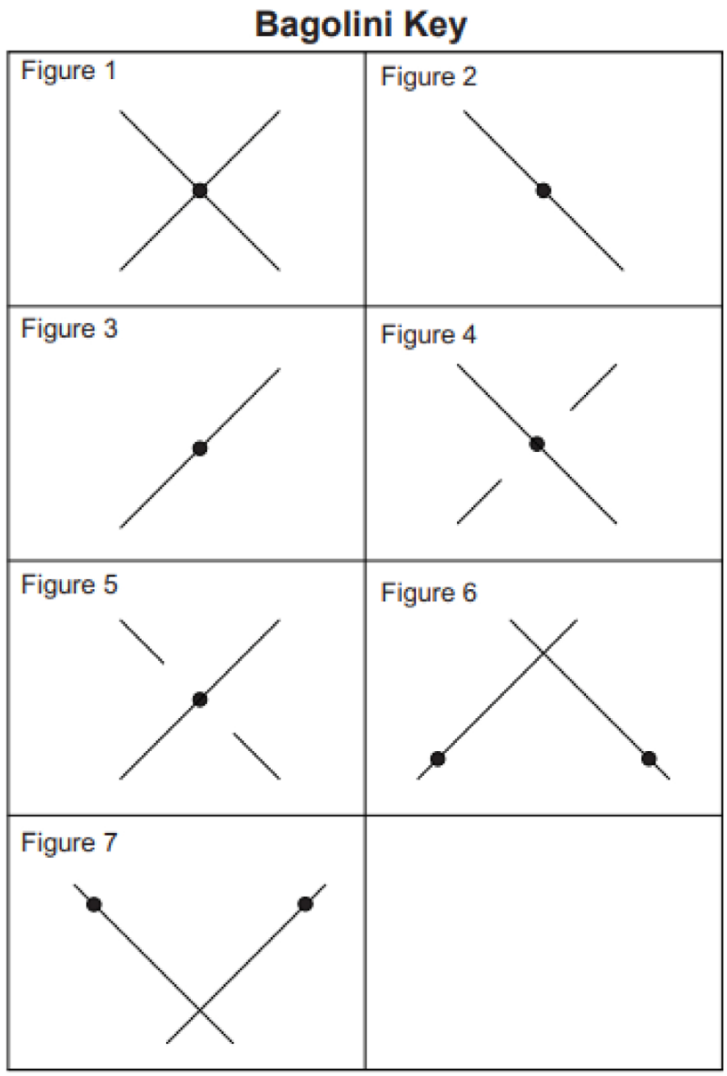


Figure 2. Bagolini Key[13,26] *Image credit: Good-Lite Company*

and surgical treatment of media opacities if indicated. Often patching or penalization therapy is prescribed to treat monocular amblyopia.[19,20,21] Patching to treat amblyopia often improves acuity, but is unlikely to decrease the central scotoma present in the non-fixating eye under binocular conditions.[22] Visual penalization is an alternate therapy that utilizes atropine sulfate to cycloplege the accommodative system in the nonaffected eye. This decreases the clarity of the normally fixating eye when looking at near distances, but has little effect on the disparity of the visual input with distance viewing.

Common clinical management is withholding treatment of this condition based on the assumption that foveal suppression under binocular conditions will limit the success of occlusion or penalization therapy.[17] Monofixation syndrome typically remains stable. A very small percentage of patients achieve improved stereoacuity over time or experience decompensation of peripheral fusion leading toward an increased angle of strabismus.[7,19,23]

Vision therapy as a treatment for small-angle esotropia is described as a treatment option in the AOA clinical management guideline for strabismus.[6] Optometric vision therapy requires multiple office visits, home practice, and costs of equipment and services. Vision therapy utilizes biofeedback such as the Haidinger's brush and after-image flash to train accurate fixation of a small deviation. Multiple strategies using red/green glasses, red/blue glasses, Polarized glasses, liquid crystal glasses, virtual reality, and stereoscopic training are aimed at decreasing suppression, building sensory fusion, and increasing fusional vergence skills to improve depth perception and binocular performance.[25]

Monofixation syndrome caused by microtropia is successfully treated by vision therapy as described in case reports.[13,24,25] Optometric vision therapy remediates symptoms of reading difficulties and blurred vision associated with central suppression.[20] Treating monofixation syndrome is a more extensive process than solely addressing a monocular reduction in visual acuity; it is a visual impairment that includes binocular vision dysfunction, fixation instability, and poor oculomotor control.[21] Vision therapy for monofixation syndrome should be clinically recommended for patients as functional improvements are not dependent on age. Patching or penalization therapy alone is insufficient for improving binocular function.

CONCLUSION

Monofixation syndrome is assessed by ruling out macular pathology and identifying the presence of an amblyogenic factor that affects bifoveal fixation while permitting peripheral fusion. Stereopsis and visual acuity can be improved in patients with monofixation syndrome by treating the underlying binocular sensorimotor dysfunction, central suppression, inaccurate fixation, and amblyopia. Vision therapy procedures designed to decrease suppression, improve sensory fusion, build accommodative facility, and increase fusional vergence ranges eliminated symptoms and improved both visual performance and comfort.

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