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Original Article

Evaluation of fixation in amblyopic patients with myopic anisometropia

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ABSTRACT

Objectives: This study aimed to evaluate amblyopic participants with myopic anisometropia who, despite not being initially diagnosed with strabismus, failed to improve their visual acuity (VA) after treatment. The hypothesis is that these individuals present with microtropia (MT) and sensory adaptations. Since they appear orthotropic on the unilateral cover test, the hypothesis was tested through an objective evaluation of fixation, the presence of eccentric fixation (EF) would indicate that these eyes are monocularly deviated.

Methods: Fourteen participants (16.07 ± 10.56 years) were recruited from the Vision Clinic at the University of Murcia. They were myopic, amblyopic, anisometropic, and initially classified as non-strabismic, with no improvement following treatment. They underwent refractive, motor, sensory, eye health and fixation assessments using visuoscopy.

Results: A slight tendency towards esophoria was observed at distance (1.07Δ ± 2.67Δ) and near (2.50Δ ± 6.26Δ). Refraction in amblyopic eyes was 3D more myopic than in non-amblyopic eyes, showing a strong correlation ($r = 0.91$, $p < 0.0001$). A non-significant relationship was found between the degree of anisometropia and VA in the amblyopic eye ($r = -0.27$, $p = 0.3944$), as well as between anisometropia and stereopsis (Spearman $r_s = 0.22$, $p = 0.439$). Of the 14 amblyopic eyes evaluated, 10 presented with EF (indicating strabismus), 2 had unstable fixation, 1 could not be evaluated, and 1 presented central fixation.

Conclusion: The majority of the participants evaluated (71.4%) exhibited MT and EF. While this association is well-documented in hyperopic anisometropia, it has not been previously characterized in myopic anisometropia.

Introduction

Functional amblyopia is a neural disorder in which visual function is reduced, usually in only one eye, affecting up to 3% of the population.[1,2] It can be classified as strabismic, anisometropic, or isometropic amblyopia, with the anisometropic type being considered the most frequent.[3] Strabismic and anisometropic amblyopias are often comorbid; a common case is partially accommodative esotropia (PAET), where anisometropia is typically present, leaving a variable residual angle of deviation after correcting the patients' total hyperopia. This is also the case with microtropia (MT).[4,5]

Functional recovery[6] in these patients is complex because they often exhibit sensory adaptations such as central suppression and Anomalous Sensory Correspondence (ASC), where the fovea of the non-amblyopic eye corresponds to a parafoveal area of the amblyopic eye.[7] They also commonly present Eccentric Fixation (EF), where the amblyopic eye fixates with a parafoveal area when the non-amblyopic eye is occluded. Furthermore, these patients typically have impaired stereopsis.[8]

In patients presenting with both strabismus and anisometropia, there is uncertainty regarding which condition is primary: did strabismus cause anisometropia, or was anisometropia the cause of the visual axis deviation? Alternatively, may this phenomenon occur in both directions? Prospective studies in animal models have shown that anisometropia can precede amblyopia and vice versa. Moreover, in these animal models, monocular deprivation has been demonstrated to affect eye growth and synaptic circuits in the visual cortex, leading to both amblyopia and anisometropia.[9]

In humans, due to the difficulty of performing studies in very young children, little is known about the origin of this phenomenon. Previous studies have shown that anisometropia often appears in the first months of life and typically normalizes around the first year.[10] Other longitudinal studies in children suggest that in the presence of early esotropia (ET), strabismus causes amblyopia, which in turn affects the emmetropization process, leading to anisometropia.[11]

In clinical practice, amblyopic subjects with myopic anisometropia are sometimes encountered who, despite showing no strabismus on the unilateral Cover Test (UCT) and having healthy eyes, do not improve their VA after appropriate treatment, including optimal optical correction, occlusion therapy, and Active Vision Therapy (VT).[12] In these cases, it can be conjectured that an unnoticed subclinical phenomenon,

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such as MT with identity (where no manifest movement occurs on the UCT) and EF, may have caused amblyopia and affected the emmetropization process.[13]

Accordingly, the primary objective of this study was to evaluate a group of amblyopic participants with myopic anisometropia who did not improve their VA after standard treatment (refraction, occlusion, and VT), by determining, among other factors, their fixation pattern.[14] The hypothesis is that these individuals, initially diagnosed solely with myopic anisometropic amblyopia, also present with MT and EF. Secondary objectives included exploring the relationship between the degree of anisometropia and both the VA of the amblyopic eye and stereopsis levels.

Material and methods

Participant selection

A retrospective search was performed in the database of the University Clinic of Vision (CUVI) at the University of Murcia. We analyzed patients from the binocular vision and pediatric sections, looking for participants who were amblyopic, initially diagnosed as non-strabismic, and whose eye with the lower VA was at least 1D myopic, with one or more lines of VA difference compared to the better eye, and anisometropia of $\geq$ 0.75D.[15] All included participants had undergone amblyopia treatment (refraction, occlusion, VT) without achieving expected VA improvement. We selected a total of 14 participants (0.21% of the total CUVI population), mean age 16.07 $\pm$ 10.56 years (8 women and 6 men), who were recalled for evaluation at CUVI.

Informed consent was obtained from all participants (or legal guardians). This research was approved by the Ethics Committee of the University of Murcia and conducted in accordance with the Declaration of Helsinki.

Procedure and data collection

Tests were performed by two optometrists. One performed refraction and visuoscopy, while the other performed the remaining tests, which included:

- Monocular distance VA (Topcon ACP.8®), with habitual optical correction (decimal scale).
- Objective refraction with automatic autorefractor (L79-ARK® Visionix®) and distance retinoscopy (Welch Allyn®).
- Subjective refraction, based on retinoscopy and seeking the most positive lens for best VA. If there was a change from their spectacle correction, subsequent tests were performed with trial frames using the new subjective refraction.
- Ocular alignment using distance and near Cover Test (CT), assessed with a prism bar (positive values for esodeviation, negative values for exodeviation).
- Distance and near fusion tests: Worth 4-Dot Test, Bagolini Striated Lenses, penlight and red/green (R/G) glasses.
- Stereopsis (TNO test) assessed in seconds of arc.

Following these tests, cycloplegic drops (Colircusí Cycloplegic® ophthalmic solution) were instilled in both eyes two times with a 15-minute interval. Thirty minutes later, Optical Coherence Tomography (OCT) and retinography (Topcon D OCT-1000®) to assess retinal structure, and automatic autorefractometry (L79-ARK® Visionix®) were performed. Finally, visuoscopy was performed to assess fixation using a Welch Allyn® LED 3.5v coaxial ophthalmoscope. In a darkened room, the examiner focused on the participant's retina (matching the examiner's eye to the participant's). After occluding the non-evaluated eye, the participant was asked to fixate on the projected visuoscope star. The fixation pattern was recorded after allowing several seconds for stabilization.

Data were analyzed using GraphPad Prism. The Shapiro-Wilk test was used to assess normality. For normally distributed variables (refraction and VA), Pearson correlation and linear regression with 95% confidence bands were used. For non-normal data (stereopsis), Spearman's rank correlation was applied. For refraction, all values were collected as spherical equivalent (SE). A paired *t*-test compared SE between eyes. A multiple linear regression model explored the influence of age, anisometropia, and fixation on VA. Significance was set at $p < 0.05$.

Results

The mean age of participants was 16.07 $\pm$ 10.56 years (range 5−44). All had previously undergone an amblyopia treatment program during childhood, consisting mainly of refraction, occlusion, and active VT (except participant 10, aged 44, who did not perform VT).

Mean subjective SE results were −6.69D $\pm$ 4.10D right eye (RE) (ranging from +0.50D to −14.25D) and −5.62D $\pm$ 4.28D left eye (LE) (ranging from −1.00D to −15.25D). Mean automatic refractometry SE after cycloplegia was −6.65D $\pm$ 4.18D RE and −5.58D $\pm$ 3.83D LE, results very similar to the subjective refraction. The amblyopic eyes were 3.00D more myopic than the non-amblyopic eyes: −7.68D $\pm$ 3.81D versus −4.63D $\pm$ 4.03D (paired *t*-test: $t(13) = 6.90$, $p < 0.0001$), with a strong correlation: $r = 0.91$ (95% CI for slope: 0.62 to 1.10, $p < 0.0001$) (Fig. 1). Mean decimal VA was 0.66 $\pm$ 0.15 (amblyopic), and 1.07 $\pm$ 0.21 (non-amblyopic) (Table 1). A weak, non-significant

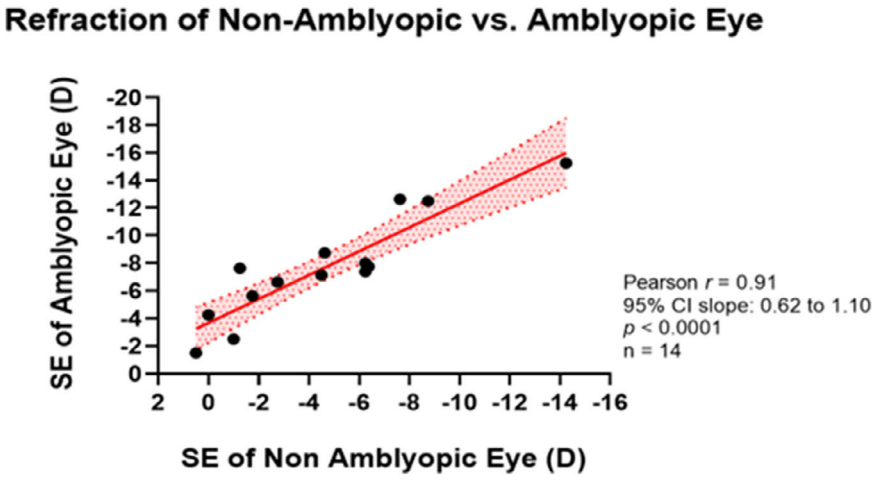

Fig. 1. Relationship between the SE refraction of the non-amblyopic and amblyopic eye. Solid line indicates linear regression; shaded area indicates 95% confidence bands ($r = 0.91$, $p < 0.0001$).

Table 1
SE and VA of each participant's amblyopic and non-amblyopic eyes, after subjective refraction.

PARTICIPANT	AGE	SE AMBLYOPIC EYE (D)	AMBLYOPIC VA (decimal)	SE NON AMBLYOPIC EYE (D)	NON-AMBLYOPIC VA (decimal)
1	12	−7.38	0.58	−6.25	0.8
2	9	−1.50	0.8	0.50	1.2
3	30	−12.50	0.32	−8.75	0.64
4	14	−4.25	0.62	0.00	1.2
5	7	−7.13	0.68	−4.50	1.2
6	9	−15.25	0.66	−14.25	0.76
7	6	−5.63	0.8	−1.75	1.2
8	18	−8.75	0.72	−4.63	1.2
9	18	−8.00	0.78	−6.25	1.12
10	44	−2.50	0.52	−1.00	1.2
11	20	−7.75	0.9	−6.38	1.2
12	5	−7.63	0.5	−1.25	0.84
13	20	−12.63	0.78	−7.63	1.2
14	13	−6.63	0.58	−2.75	1.2
MEAN	**16.07**	**−7.68**	**0.66**	**−4.63**	**1.07**
SD	**10.56**	**3.81**	**0.15**	**4.03**	**0.21**

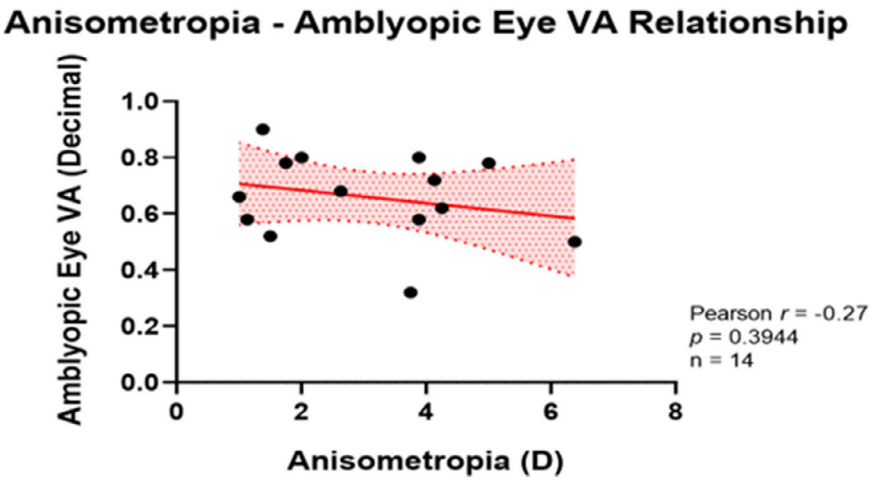

Fig. 2. Relationship between anisometropia and amblyopic eye VA. The relationship was not significant ($p = 0.3944$). Shaded area shows 95% confidence bands.

correlation was found between anisometropia (difference in SE refraction between the amblyopic and non-amblyopic eye) and the VA of the amblyopic eyes: $r = -0.27, p = 0.3944$ (Fig. 2).

Regarding alignment (CT), the mean phoria at distance was 1.07Δ ± 2.67Δ (6 esophoric, 7 orthophoric, and 1 exophoric) and at near was 2.50Δ ± 6.26Δ (8 esophoric, 3 orthophoric, and 3 exophoric) (Table 2).

About the sensory evaluation conducted through three subjective tests, Bagolini striated lenses, Worth 4-Dot Test, and penlight and red/green (R/G) glasses, responses were variable, challenging to quantify, and occasionally paradoxical (Table 2). For near vision, most participants reported fusion across all three tests, with the exception of participant 14, who reported diplopia during the R/G test, a finding consistent

Table 2
Amblyopic eye fixation, VA, distance and near fusion tests (BO: Base Out, F: Fusion, NA: not assessable), stereopsis, and distance and near CT (Positive Value: Eso).

PARTICIPANT	FIXATION TYPE	VA	WORTH		BAGOLINI		R/G		STEREOPSIS (`` of arc)	CT (Δ)	
			DISTANCE	NEAR	DISTANCE	NEAR	DISTANCE	NEAR		DISTANCE	NEAR
1	Nasal	0.58	F	F	8BO	F	F	F	240	8	4
2	Superior Unstable	0.8	F	F	F	F	F	F	240	1	0
3	Not evaluable (NE)	0.32	NA	F	NA	F	NA	F	240	0	−7
4	Nasal-Inferior	0.62	F	F	F	F	F	F	120	−4	−2
5	Nasal	0.68	F	F	F	F	F	F	1200	0	0
6	Central Unstable	0.66	F	F	F	F	F	F	240	0	18
7	Superior	0.8	F	F	F	F	F	F	480	0	2
8	Nasal-Inferior	0.72	F	F	6BO	F	F	F	480	2	4
9	Central Unstable	0.78	F	F	F	F	F	F	480	0	−6
10	Nasal-Inferior	0.52	F	F	F	F	F	F	240	2	8
11	Central	0.9	F	F	F	F	4BO	F	240	4	8
12	Nasal-Inferior	0.5	F	F	F	F	F	F	480	0	2
13	Eccentric Unstable	0.78	F	F	F	F	F	F	240	0	0
14	Nasal	0.58	2BO	F	F	F	8BO	4BO	480	2	4
MEAN		**0.66**							**385.71**	**1.07**	**2.50**
SD		**0.15**							**267.00**	**2.67**	**6.26**

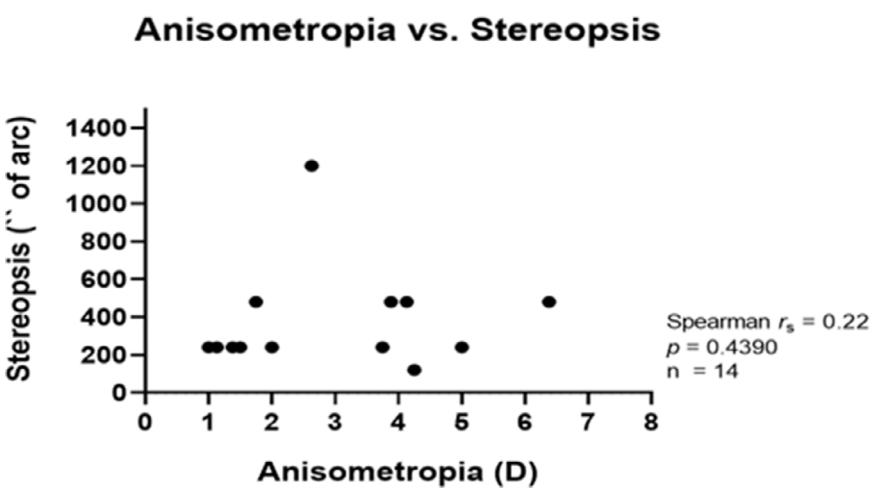

Fig. 3. Relationship between anisometropia and stereopsis. Spearman correlation was used due to non-normal distribution ($r_s = 0.22, p = 0.439$).

with the CT value. For distance vision, sensory function for participant 3 could not be assessed due to the poor VA of the amblyopic eye (0.32). Participant 1 reported uncrossed diplopia in the Bagolini test, consistent with the CT findings; participant 8 also reported diplopia during the Bagolini test at a value exceeding the CT measurement; participant 11 presented diplopia in the R/G test with a value equal to CT; while participant 14 reported diplopia values identical to the CT in the Worth 4-Dot Test and higher than the CT in R/G test.

After pupillary dilation, retinal assessment was performed for all participants using OCT and retinography, followed by an evaluation of amblyopic eye fixation via visuoscopy. Retinal health was normal for all participants except for participant 3, who presented with incipient myopic retinopathy in both eyes. Of the 14 amblyopic eyes evaluated, 10 presented with EF, 2 demonstrated unstable central fixation, and 1 exhibited stable central fixation (corresponding to the participant with the highest VA), one participant could not be evaluated. The 10 cases of EF were categorized as follows: 7 nasal or combined nasal-vertical (consistent with ET), 2 vertical, and 1 exhibiting highly unstable EF.

The mean stereopsis across all participants was 385.71″ ± 267.00″, with a peak value of 120″ and participant 5 demonstrating null stereopsis[16] (Table 2). The correlation between anisometropia and stereopsis was $r_s = 0.22, p = 0.439$. After excluding participant 5 (null stereopsis), the relationship remained non-significant ($r_s = 0.27, p = 0.368$) (Fig. 3). Finally, a multiple linear regression analysis, incorporating age, anisometropia, and fixation type, indicated that these variables did not significantly predict the VA of the amblyopic eyes ($F(3, 9) = 0.578, p = 0.643, R^2 = 0.16$).

Discussion

In the absence of pathology, functional amblyopia is associated with anisometropia, strabismus, and isometropia.[3] The rationale for treating anisometropic amblyopia follows the same protocol as for isometropic cases: prescribing the optimal refractive correction, followed by occlusion and active VT if VA does not improve.[17–19] If no underlying pathology exists, participants should recover visual function through this standard protocol, although marked anisometropia may require contact lenses to manage the aniseikonia caused by the refractive difference.[20]

It is common to observe anisometropic amblyopia associated with strabismus, as seen in PAET. These cases typically involve aniso-hyperopia, frequently bilateral astigmatism, amblyopia of the strabismic eye (which is usually the most ametropic) with EF, and sensory adaptations such as central suppression and ASC. Functional recovery in these strabismus cases is complex, a residual angle remains after correcting the ametropia and improving fusional reserves, frequently necessitating surgical intervention.[21] Conversely, MTs exhibit characteristics very similar to those mentioned above: amblyopia and sensory adaptations are typical, although the esotropic angle is difficult to detect due to its small magnitude, often coinciding with EF and ASC.[22,23] In such cases, there is no manifest movement on the unilateral CT, making the evaluation of fixation essential to confirm the diagnosis. As previously stated, this is known as MT with identity.[13]

In daily optometric practice, however, we occasionally encounter amblyopic participants with myopic anisometropia who are classified as non-strabismic. While these participants should ideally achieve a decimal VA of 1.0 in the affected eye with appropriate treatment, this outcome is not always achieved. This clinical observation raises the question of the underlying etiology. Since these eyes are otherwise healthy, a possible explanation is the presence of subclinical strabismus, specifically MT with identity, which shows no movement on the unilateral CT. In our retrospective search of 2099 participants at CUVI, only 14 (0.67%) were initially diagnosed with these characteristics: myopic anisometropia with non-strabismic amblyopia. This small sample size reflects the rarity of this clinical profile, providing a specific cohort of treatment-resistant cases. Since none of the selected participants showed tropia on the unilateral CT, despite a clear, albeit small, trend toward esophoria (Table 2), the only definitive method to determine if these phoric values represented MT with identity was through the evaluation of fixation.

The most common clinical method to evaluate fixation is visuoscopy.[24] Of the 14 myopic and amblyopic eyes evaluated by visuoscopy in our study, 10 presented with EF (71.40%), 2 demonstrated unstable central fixation (and were therefore inconclusive), 1 could not be evaluated, and only 1 exhibited stable central fixation. This indicates that at least 10 participants were strabismic, suggesting that EF, rather than anisometropia, was the primary cause of the amblyopia, as illustrated in Fig. 2. Participant 4 is noteworthy for exhibiting an "exo" deviation despite presenting nasal (typical of ET) and inferior EF. This phenomenon is occasionally observed if the examiner does not maintain the visuoscope focus on the retina long enough for fixation to stabilize or if the participant is uncooperative.[25] Furthermore, the most significant eccentricity in this participant was vertical.

EF is a highly amblyogenic condition, as the participant fixates with a parafoveal area where VA is dramatically reduced due to a lower density of cones.[26] Previous research has indicated that in participants with ASC, the distinguishing factor between those with good VA in the amblyopic eye and those with deeper amblyopia is fixation, which is eccentric in the latter group. (Martínez García A. Evaluation of visual function in the amblyopic eye in subjects with anomalous sensory correspondence. [Final Degree Project]. University of Murcia; 2018)

As stated, anisometropic amblyopia associated with MT with identity is typically linked to hyperopia and has been extensively studied.[27,24,28,11] However, to our knowledge, this study demonstrates

for the first time that the majority of participants with myopic anisometropia who do not improve distance VA after treatment present with EF. Only one prior study exclusively evaluated VA improvement in myopic anisometropic amblyopes after refractive treatment and patching, that study found that VA improvement was inversely proportional to the presence of EF.[29]

Assessing sensory correspondence in these participants is complex due to the small angles involved and the subjectivity of the tests, making the results highly debatable. Nevertheless, in the three fusion tests performed at distance and near, most participants reported fusion, suggesting the presence of harmonious ASC, as they achieve fusion despite a manifest deviation.[30] At distance, only participant 1 (Bagolini), participant 11 (R/G), and participant 14 (Worth 4-Dot Test) demonstrated Normal Sensory Correspondence (NSC), as the objective CT value coincided with the subjective test results. Conversely, participants 8 (Bagolini) and 14 (R/G) showed paradoxical responses. For near vision, only participant 14 (R/G) exhibited NSC. These results are consistent with Bagolini's 1967 study, which found that in strabismus of <10Δ, >90% of participants presented harmonious ASC when assessed with a low-dissociating test such as striated lenses.[31]

Consequently, at least 10 out of the 14 myopic, anisometropic, and amblyopic participants presented with microtropia with EF and likely harmonious ASC. The stereopsis results support this diagnosis,[32] as stereopsis was poor or null for nearly all participants, except for participant 4 (exophoric with mostly vertical EF), whose stereopsis was acceptable (120″ with TNO test). Additionally, although a slight trend toward poorer stereopsis with increasing anisometropia was observed, it failed to reach statistical significance, indicating high variability among the participants (Fig. 3).

There is a rare form of non-accommodative restrictive ET associated with myopia, accounting for approximately 4% of non-accommodative cases, which occur in high myopes where both eyes can become extremely adducted.[33] As this does not reflect the clinical profile of the participants in our study, it is necessary to propose hypotheses regarding the etiology of this myopic anisometropia.

The first hypothesis is that these participants presented with MT during early childhood, which led to amblyopia and subsequently disrupted the emmetropization process,[34] resulting in anisometropia. This hypothesis is consistent with studies by Abrahamsson and Sjöstrand, which suggest that while anisometropia is common in the first months of life and typically disappears through emmetropization, this process does not occur in strabismic and amblyopic children.[35] It is probable that in their early years, these children were less hypermetropic than average (or even slightly myopic), the myopia may have been detected late, exacerbating the condition, as it is well known that retinal blur induces myopia.[36]

A second hypothesis would be the opposite process: binocularly normal participants developing myopia and anisometropia. The uncorrected anisometropic eye would experience blurred vision and passive central suppression,[2] eventually leading to unstable fixation and the subsequent development of amblyopia and MT.

Finally, this study has several fundamental limitations. The first is the small sample size ($n = 14$), and the second is the lack of quantitative measurement for the magnitude of the EF. Furthermore, an additional limitation involves the absence of a control group consisting of non-amblyopic myopic anisometropes. Regarding the first limitation, the small cohort size is a challenge to address due to the highly specific clinical characteristics required for inclusion, this limits the generalizability of our findings. However, we believe these results provide a reasonable suspicion regarding the lack of visual improvement in this type of amblyopia and can serve as a foundation for future studies with larger populations. Regarding the second limitation, evaluating fixation is complex with visuoscopy due to the required measurement conditions and high level of participant cooperation; therefore, microperimetry may offer a superior alternative for future research. With respect to the final limitation, while the participants' non-amblyopic eyes provided a baseline for comparison, the inclusion of a separate control group would be methodologically superior in future investigations.

Conclusion

Out of 14 participants initially diagnosed with myopic anisometropic amblyopia without strabismus, who did not improve their VA despite various treatment attempts during childhood, 10 presented with EF, likely representing MT with identity and ASC. Notwithstanding its limitations, our findings have clinical implications, emphasizing the importance of evaluating fixation in these participants. There is a high probability that functional amblyopias appearing to be purely anisometropic but failing to improve with standard treatment are actually associated with subclinical strabismus. This association was known for MT with identity and anisohyperopia, but not as much for myopic anisometropia.

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Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the authors used Gemini (Google) in order to assist with translation and improve language readability. After using this tool/service, the authors reviewed and edited the content as needed and takes full responsibility for the content of the publication.

Declaration of competing interest

Declarations of interest: none.

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References

1. Hess RF, Mansouri B, Thompson B. Restoration of binocular vision in amblyopia. Edited by: Jampolsky A, Hiatt RL, eds. In: *Strabismus*. 2nd ed. Taylor & Francis; 2011:110–118. https://doi.org/10.3109/09273972.2011.600418.
2. Levi DM. Rethinking amblyopia 2020. *Vis Res*. 2020;176:118–129. https://doi.org/10.1016/j.visres.2020.07.014.
3. Ciuffreda KJ, Levi DM, Selenow A. *Amblyopia: Basic and Clinical Aspects*. Butterworth-Heinemann; 1991.
4. Von Noorden GK. The etiology and pathogenesis of fixation anomalies in strabismus. *Trans Am Ophthalmol Soc*. 1969;67:698–751.
5. Tomaç S, Ener EC, Sanaç A. Clinical and sensorial characteristics of microtropia. *J Pediatr Ophthalmol Strabismus*. 2002;39(3):139–143.
6. Flom MC. The prognosis in strabismus. *Am J Optom Arch Am Acad Optom*. 1964;41(9):503–514.
7. Jennings JAM. Anomalous retinal correspondence: a review. *Ophthalmic Physiol Opt*. 1985;5(4):357–368.
8. Ghasia F, Wang J. Amblyopia and fixation eye movements. *J Neurol Sci*. 2022;441:120373. https://doi.org/10.1016/j.jns.2022.120373.
9. Mitchell D, Sengpiel F. Animal models of amblyopia. *Vis Neurosci*. 2018;35:E017. https://doi.org/10.1017/S0952523817000244.
10. Abrahamsson M, Fabian G, Sjöstrand J. A longitudinal study of a population based sample of astigmatic children: II. The changeability of anisometropia. *Acta Ophthalmol (Copenh)*. 1990;68:435–440. https://doi.org/10.1111/j.1755-3768.1990.tb01672.x.
11. Barrett BT, Bradley A, Candy TR. The relationship between anisometropia and amblyopia. *Prog Retin Eye Res*. 2013;36:120–158. https://doi.org/10.1016/j.preteyeres.2013.05.001.

12. Suttle CM. Active treatments for amblyopia: a review of the methods and evidence base. *Clin Exp Optom*. 2010;93(5):287–299. https://doi.org/10.1111/j.1444-0938.2010.00486.x.
13. Houston CA, Cleary M, Dutton GN, McFadzean RM. Clinical characteristics of microtropia-is microtropia a fixed phenomenon? *Br J Ophthalmol*. 2002;86(1):15–18.
14. Milla M, Piñero DP. Characterization, passive and active treatment in strabismic amblyopia: a narrative review. *Int J Ophthalmol*. 2020;13(7):1132–1147. https://doi.org/10.18240/ijo.2020.07.18.
15. Cass K, Tromans C. A biometric investigation of ocular components in amblyopia. *Ophthalmic Physiol Opt*. 2008;28(5):429–440. https://doi.org/10.1111/j.1475-1313.2008.00585.x.
16. Chopin A, Bavelier D, Levi DM. The prevalence and diagnosis of 'stereoblindness' in adults less than 60 years of age: a best evidence synthesis. *Ophthalmic Physiol Opt*. 2019;39(1):66–85. https://doi.org/10.1111/opo.12607.
17. Fern KD. Visual acuity outcome in isometropic hyperopia. *Optom Vis Sci*. 1989;66(10):649–658.
18. Cotter SA. Treatment of anisometropic amblyopia in children with refractive correction. *Ophthalmology*. 2006;113(6):895–903. https://doi.org/10.1016/j.ophtha.2006.01.068.
19. Milla M, Molina-Martín A, Piñero DP. Long-term efficacy of the combination of active vision therapy and occlusion in children with strabismic and anisometropic amblyopia. *Child (Basel)*. 2022;9(7):1012. https://doi.org/10.3390/children9071012.
20. Evans BJW. Orthoptic indications for contact lens wear. *Cont Lens Anterior Eye*. 2006;29(4):175–181. https://doi.org/10.1016/j.clae.2006.03.007.
21. Lembo A, Serafino M, Dello Strologo M, Saunders RA, Trivedi RH, Villani E, et al. Accommodative esotropia: the state of the art. *Int Ophthalmol*. 2019;39(3):497–505. https://doi.org/10.1007/s10792-018-0821-6.
22. Lang J. Management of microtropia. *Br J Ophthalmol*. 1974;58(3):281–288.
23. Lea SJH, Snead MP, Loades J, Rubinstein MP. Microtropia versus bifoveal fixation in anisometropic amblyopia. *J Pediatr Ophthalmol Strabismus*. 1991;28(1):51–53.
24. Cooper J, Gelfond I, Carlson PE, Campolattaro B, Wang F. Comparison of eccentric fixation measurements using the streak target of an ophthalmoscope and a traditional visuoscopy target. *Optom Vis Sci*. 2004;81(10):785–792.
25. Kalnica-Dorosenko K, Kalnupa M, Svede A, Valeina S. Eccentric fixation measurements using visuoscopy and macula integrity tester (MIT) in children with amblyopia. *SPIE-Intl Soc Opt Eng*. 2021:37. https://doi.org/10.1117/12.2597226.
26. Hess RF. On the relationship between strabismic amblyopia and eccentric fixation. *Am J Optom Physiol Opt*. 1977;54(7):448–456.
27. Birch EE, Subramanian V, Weakley DR. Fixation instability in anisometropic children with reduced stereopsis. *J AAPOS*. 2013;17(3):287–290. https://doi.org/10.1016/j.jaapos.2013.03.011.
28. Lysons D, Tapley J. Is microtropia a reliable indicator of the presence of amblyopia in anisometropic patients? *Strabismus*. 2018;26(3):118–121. https://doi.org/10.1080/09273972.2018.1503308.
29. Pang Y, Allison C, Frantz KA, Block S, Goodfellow GW. A prospective pilot study of treatment outcomes for amblyopia associated with myopic anisometropia. *Arch Ophthalmol*. 2012;130(5):579–584. https://doi.org/10.1001/archopthalmol.2011.1203.
30. Sireteanu R, Fronius M. Different patterns of retinal correspondence in the central and peripheral visual field of strabismics. *Dev Brain Res*. 1989;48(1):159–162.
31. Bagolini B. Anomalous correspondence: definition and diagnostic methods. *Doc Ophthalmol*. 1967;23:346–387.
32. Rutstein RP, Eskridge JB. Stereopsis in small-angle strabismus. *Am J Optom Physiol Opt*. 1984;61(8):491–498.
33. Ranka MP, Steele MA. Esotropia associated with high myopia. *Curr Opin Ophthalmol*. 2015;26(5):362–365. https://doi.org/10.1097/ICU.0000000000000180.
34. Schaeffel F, Swiatczak B. Mechanisms of emmetropization and what might go wrong in myopia. *Vis Res*. 2024;220:108402. https://doi.org/10.1016/j.visres.2024.108402.
35. Abrahamsson M, Sjostrand J. Natural history of infantile anisometropia. *Invest Ophthalmol Vis Sci*. 1996;37(4):S570.
36. Biswas S, El Kareh A, Qureshi M, Lee DMX, Sun CH, Lam JSH, et al. The influence of the environment and lifestyle on myopia. *J Physiol Anthr*. 2024;43(1):33. https://doi.org/10.1186/s40101-024-00354-7.