ORIGINAL ARTICLE

Impact of Mimicked Anisometropia on Visual Functions and Aniseikonia in Adults

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ABSTRACT

Introduction: Anisometropia is a condition in which refractive error between two eyes is differs in ≥ 1Diopter (D) and potentially disrupt the visual function. This study aimed to investigate the effects of mimicked anisometropia on stereoacuity, visual acuity (VA), contrast sensitivity (CS) and aniseikonia among different magnitude of anisometropia groups. **Materials and methods:** This cross-sectional study was conducted on 20 healthy emmetropic adults. Soft contact lenses from 1Diopter Sphere (DS) to 4DS were fitted to induce unilateral myopia, hyperopia and astigmatism. VA, stereoacuity, CS and aniseikonia were measured at baseline and at each level of defocus. **Results:** VA degraded in all groups at all magnitudes of anisometropia induced as compared to baseline ($p<0.05$). No significant changes was observed on stereoacuity and CS in hyperopic (stereoacuity:0.08±0.21; CS: 1.71±0.25) and astigmatic (stereoacuity:0.03±0.35; CS: 1.78±0.17) groups at 1D anisometropia induced compared to baseline (stereoacuity: -0.11±0.20; CS: 1.91±0.11). In comparison with baseline, significant percentage of horizontal aniseikonia was observed at magnitude of 3D (myopic: $p<0.01$; hyperopic: $p=0.04$; astigmatic: $p=0.03$) and 4D (myopic: $p<0.01$; hyperopic and astigmatic: $p=0.02$) for all groups. Meanwhile, for vertical anisometropia, significant percentage was presented at 3D ($p=0.04$) and 4D ($p<0.01$) in myopic group, 4D ($p=0.02$) in hyperopic group and non-significant increment was observed in vertical aniseikonia of astigmatic anisometropia group. **Conclusion:** Maximum effects on visual functions were observed among mimicked myopic anisometropia, followed by hyperopic and astigmatic anisometropia. Out of all visual functions assessed, the most pronounce change was observed in stereoacuity.
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Keywords: Mimicked anisometropia, Visual acuity, Stereoacuity, Contrast sensitivity, Aniseikonia

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INTRODUCTION

Anisometropia is presented in both adult and paediatric populations, affecting individuals with varying age groups. In adult population, anisometropia usually caused by the conditions such as cataract, post-refractive surgeries and injuries or trauma affecting one eye (1, 2). As for paediatric population, anisometropia may occur due to interocular imbalance of refractive components such as axial length, cornea curvature and lens shape (1, 3).

The prevalence of anisometropia based on studies conducted in Malaysia was between 0.7% to 7.7% (4–6). Besides, it was also reported that anisometropia is the leading factor that contribute to the neurodevelopmental disorder of visual system known as amblyopia (7, 8). Late detection and delay intervention in children with anisometropia are among the causes of anisometropic amblyopia which resulted in poor stimulation and development of visual pathway (9). This highlights the significance of earlier intervention among anisometropic children to avoid the suppression of the weaker eye which may lead to amblyopia.

The severity of amblyopia varies among individuals, depending on the associated factors such as genetic, environmental and developmental factors. Recent study suggested the main risk factor for the increment of anisometropia among school children is due to lifestyle such as less time spent outdoors and increased in near work (10). The severity of anisometropia is related to visual impairment, in which mild anisometropia may not significantly affect visual functions, but bigger interocular differences in severe and moderate anisometropia may associate with higher severity of amblyopia and worse visual functions (11, 12). Related to this, this study aimed to evaluate the outcome of mimicked anisometropia on visual functions among different magnitudes of anisometropia groups.

MATERIALS AND METHODS

Study population

The sample size was calculated using PS- Power and sample size calculation, based on previous study by Jethani et al. (2015). With the value of mean difference, 39.00 and standard deviation of ±36.90, the sample size calculated was 15 participants. However, 30% of participants was added to the total sample size to allow for possible dropouts among the participants. Thus, the total sample size was 20 participants for each magnitude of anisometropia for every group.

This cross-sectional study recruited 20 emmetropic adults, aged between 18 to 40 years old with spherical equivalence of refractive error ranged between -0.25DS to +0.50DS, VA of 6/6 or better on both eyes and stereoacuity of 60" of arc or better measured using TNO. The participants were excluded from this study if they have uncorrected refractive error, reduced stereopsis or having binocular vision problems. The study procedures adhered to the tenets of the Declaration of Helsinki and study protocols were approved by the International Islamic University Malaysia (IIUM) Research Ethics Committee (IREC; IIUM/504/14/11/2/IREC 2018-227). Before data collection, all participants were briefed on the study steps and written consent was obtained. Prior to data collection, an optometric evaluation was conducted in order to assess the ocular health of the participants and ensure that the inclusion and exclusion criteria were met.

Mimicked anisometropia

Anisometropia may disrupt binocularity, leading to deficits in visual functions (14, 15). It was suggested that the use of contact lenses may minimise the difference of retinal image sizes among anisometropic eyes (16), thus the deterioration of visual functions is expected to be lessen as compared to spectacles. Related to this, in this study, the anisometropia was induced with the use of contact lenses.

Conventional Aire CD 38 and Aire Toric 38 soft contact lenses (Apple Vision Sdn. Bhd., Malaysia) with diameter of 14.20 millimeters (mm) and base curve of 8.60 (17) were fitted on dominant eye of all emmetropic participants to induce anisometropic condition. Plus (+) soft contact lens mimicked myopic anisometropia, minus (-) lens wear mimicked hyperopic anisometropia and against-the-rule cylindrical contact lens simulated with-the-rule astigmatism. Soft contact lens was fitted on participants' dominant eyes to mimic different levels of anisometropia, with magnitude of ±1DS, ±2DS, ±3DS and ±4DS. Similarly, for astigmatism, the same magnitudes were mimicked with 90° axis.

In order to ensure good contact lens-cornea relationship, 5 to 15 minutes (18) of adaptation period was given for each participant before the assessment of visual functions. For Aire Toric 38 soft contact lens, anisometropia was mimicked at 90-axis meridian (evoking 'with the rule astigmatism') to induced maximum potential astigmatic blur degradation of visual and binocular functions (19). For each visit, the participant was fitted with only four different contact lenses to minimize the learning effect and fatigue.

Visual functions and aniseikonia measurement

To eliminate the accommodation effect, one drop of Cyclopentolate Hydrochloride 1% (Alcon Laboratories Inc, Fort Worth, TX, USA) was instilled in both eyes of the participant. After 15 minutes, pupillary response to light and pupil diameter is observed; no light reflexes and pupil size ≥ 6 mm are considered adequate cycloplegia (20–22). Baseline measurement of VA (monocular and binocular) was conducted using Standard logMAR chart under well-lit room condition. The VA measurement was conducted at four meters, in photopic vision. All participants were instructed to read the smallest letter that they could, and if they couldn't read even the largest letter on the chart, they might have to move one metre closer to the chart till the largest letters could be seen. Visual acuity was recorded when three letters on a line have been missed.

After VA measurement, stereoacuity measurement was conducted using TNO stereo test. This test was conducted in a well-illuminated room, with the TNO book presented at 40cm from the participants' eye. Prior to testing, all participants need to wear a red-green goggle in front of their eyes. They were asked to identify which of the targets appear closest or seems to be floating above the page. The stereoacuity score was noted when two consecutive targets were missed. For analysis, stereoacuity threshold is converted to logarithm of Minimum Angle of Resolution (logMAR) using the following equation:

$$Stereoacuity\ (logMAR) = log\ \frac{min\ of\ arc}{60}$$

Aniseikonia was measured using a validated application, Smart Optometry Apps Version 3.3.1 (Smart-Optometry Ltd., Slovenia) (18). To measure the aniseikonia, participants need to wear a red-green google in front of their eyes. Participants were instructed to compare the sizes of the red and green semicircular brackets displayed on the phone screen while maintaining their gaze on the white dot located at the center of the brackets. (Figure 1). The goal was to equalize the size of both brackets and the difference in size indicates the percentage of aniseikonia. The aniseikonia measurement was taken as an average of three measurements to eliminate any response bias.

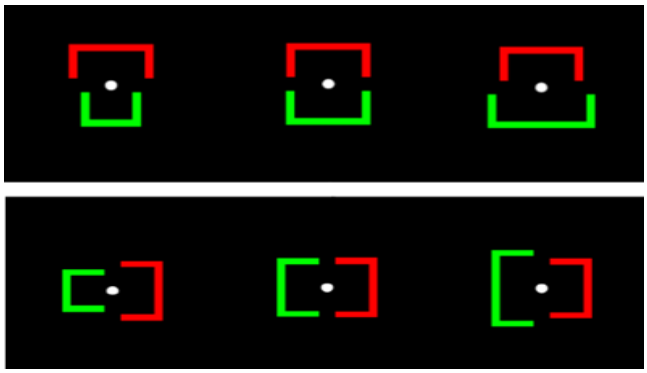

Figure 1: Aniseikonia feature along horizontal (top) and vertical (bottom) meridian in Smart Optometry™ application.

Lastly, the participants were required to read the optotype-contrast sensitivity chart (Pelli-Robson contrast sensitivity chart) monocularly and binocularly to examine the baseline for CS. To perform this test, participants were seated in a well illuminated room, at one meter distance from the Pelli-Robson contrast sensitivity chart with the middle of the chart was at the participants' eye level. The participants were asked to read the smallest letters they could discern from the Pelli-Robson chart with the tested eye while the fellow eye was occluded. Each correctly identified letter was assigned with a score of 0.05 log. The measurement was then repeated under binocular conditions.

After completing all the baseline parameters, soft contact lens was fitted on the participant's dominant eye. Ten minutes after contact lens insertion, Park 1 Auto-refractometer (OCULUS Inc., USA) was used to confirm the refractive error of the participant with contact lens. Then, VA measurement of the tested eye (dominant eye) was conducted. Afterwards, the same procedure of assessing stereoacuity, CS and aniseikonia were repeated while being fitted with contact lens. Five minutes interval was given before each assessment to avoid fatigue and wash out period of 5 minutes was allowed before the next soft contact lens fitting (23). The same procedures were repeated using other dioptric power of soft contact lens.

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 20 (IBM Corp., Armonk, N.Y., USA). The normality of data distribution was assessed using the skewness and kurtosis of the distribution (24). To compare the VA, stereoacuity, CS and aniseikonia at different magnitudes of anisometropia in each myopic, hyperopic and astigmatic anisometropia, Repeated Measures One-way Analysis of Variance (RM ANOVA) was conducted. This method was chosen to account for the within-subject design, where each participant experienced all magnitudes of induced anisometropia and the measurements for all parameters of interest were taken at each magnitude. Statistical significance was set at a p-value <0.05.

RESULTS

Visual Acuity, Stereoacuity, Contrast Sensitivity and Aniseikonia at Different Magnitudes of Anisometropia

Mean baseline of participants' VA, stereoacuity, CS and aniseikonia was 0.00 logMAR, 1.91 ± 0.11 log unit, -0.11 ± 0.20 logMAR and 1.22 ± 1.41 for horizontal aniseikonia and 1.11 ± 0.79 for vertical aniseikonia, respectively. RM ANOVA demonstrated a significant effect of myopic anisometropia increment on VA; $F[(2.36,44.86)=357.43]$, $p<0.01$. Subsequent Bonferroni post-hoc pairwise comparisons revealed a significant deterioration in VA across all magnitudes of myopic anisometropia (1.00D to 4.00D) when compared with baseline. Similarly, in hyperopic anisometropia, significant dropped in VA were identified across all magnitudes; $F[(2.47,47.01)=224.03]$, $p<0.01$. The same pattern was observed in astigmatic anisometropic group, in which significant reduction of VA was noted with increment of astigmatism magnitude; $F[(3.96,75.29)=118.61]$, $p<0.01$, specifically across all magnitudes of astigmatic anisometropia.

RM ANOVA analysis also exhibited significant reduction of stereoacuity in all magnitudes, with increasing myopic anisometropia magnitude; $F[(3.64,69.22)=90.04]$, $p<0.01$. Bonferroni analysis revealed that stereoacuity statistically impaired at all magnitudes (1.00D to 4.00D) of myopic anisometropia. Likewise, RM ANOVA revealed significant effects of hyperopic and astigmatic anisometropia increment on stereoacuity; $F[(3.48,66.48)=64.10]$, $p<0.01$ in hyperopic anisometropia group and $F[(3.84,72.97)=48.12]$, $p<0.01$ in astigmatic anisometropia group. However, in contrast to myopic anisometropia group, stereoacuity began to deteriorate noticeably at 2.00D and beyond in both hyperopic and astigmatic anisometropia groups when compared to baseline and 1.00D.

RM ANOVA showed analogous findings of CS with VA and stereoacuity, in which significant reduction of CS were observed in all anisometropia groups with the increment of anisometropia magnitudes; myopic anisometropia: $F[(2.24,42.59)=111.30]$, $p<0.01$, hyperopic anisometropia: $F[(2.68,50.88)=85.11]$, $p<0.01$ and astigmatic anisometropia: $F[(2.71,51.42)=75.76]$, $p<0.01$. Meanwhile, Bonferroni post-hoc test revealed significant reduction of CS in all magnitudes of myopic anisometropia as compared to CS value at baseline. For the other two groups, CS began worsening at 2.00D and greater when compared to baseline and 1.00D. With maximum magnitude of anisometropia (±4.00D), both myopic and hyperopic induced anisometropia degraded to 1.0 log unit, which was outside normal limit. Interestingly, in astigmatic anisometropia, although the CS reduced with the increment of anisometropia magnitudes, the measurement was still within normal range. The results revealed that stereoacuity was the

most affected by the higher magnitude of anisometropia, while the least affected visual function was CS.

As for horizontal aniseikonia, RM ANOVA revealed high percentage of aniseikonia in each anisometropia groups; myopia: F[(2.59,49.22)=16.81], p<0.05; hyperopia: F[(2.18,41.36)=6.01], p<0.05 and astigmatism: F[(2.55,48.43)=5.98], p<0.05. A significantly higher percentage of aniseikonia was demonstrated in all anisometropia groups at magnitude of ≥ 3D along horizontal meridian. Meanwhile for vertical aniseikonia, a highly significant percentage of aniseikonia was observed at ≥ 3D of myopic anisometropia, 4D of hyperopic anisometropia and no significant difference in astigmatic anisometropia group {myopia: F[(2.68, 50.69)=9.33], p<0.01; hyperopia: F[(4, 76)=5.14], p<0.01; astigmatism F[(4,76)=1.66], p:0.19}. Table I shows the reduction of visual functions and higher percentage of aniseikonia with the increment of induced anisometropia in myopic, hyperopic and astigmatic anisometropia groups.

Table I: RM-ANOVA and mean of visual functions and aniseikonia in myopic, hyperopic and astigmatic anisometropia groups

Anisometropia group	Parameters		0.00D (Mean ± SD)	1.00D (Mean ± SD)	2.00D (Mean ± SD)	3.00D (Mean ± SD)	4.00D (Mean ± SD)	*p* (RM Anova)
Myopic	VA (logMAR)		0.00 ± 0.00	0.11 ± 0.14	0.53 ± 0.21	0.85 ± 0.13	1.00 ± 0.08	**<0.01**
	Stereoacuity (logMAR)		-0.11 ± 0.20	0.08 ± 0.19	0.45 ± 0.37	0.77 ± 0.40	1.30 ± 0.35	**<0.01**
	CS (log)		1.91 ± 0.11	1.75 ± 0.16	1.47 ± 0.14	1.28 ± 0.16	1.00 ± 0.30	**<0.01**
	Aniseikonia (%)	Horizontal	1.22 ± 1.41	1.31 ± 0.86	1.98 ± 1.55	2.99 ± 1.79	4.37 ± 2.97	**<0.01**
		Vertical	1.11 ± 0.79	1.69 ± 1.22	2.00 ± 1.89	2.89 ± 2.49	4.31 ± 2.87	**<0.01**
Hyperopic	VA (logMAR)		0.00 ± 0.00	0.17 ± 0.16	0.35 ± 0.15	0.79 ± 0.15	1.05 ± 0.10	**<0.01**
	Stereoacuity (logMAR)		-0.11 ± 0.20	0.08 ± 0.21	0.20 ± 0.31	0.57 ± 0.44	1.23 ± 0.43	**<0.01**
	CS (log)		1.91 ± 0.11	1.71 ± 0.25	1.58 ± 0.19	1.42 ± 0.16	1.06 ± 0.26	**<0.01**
	Aniseikonia (%)	Horizontal	1.22 ± 1.41	1.85 ± 1.57	1.93 ± 2.23	2.74 ± 2.07	3.40 ± 2.14	**<0.01**
		Vertical	1.11 ± 0.79	1.66 ± 1.31	1.70 ± 2.01	2.36 ± 1.81	3.2 ± 2.43	**<0.01**
Astigmatic	VA (logMAR)		0.00 ± 0.00	0.15 ± 0.04	0.41 ± 0.05	0.61 ± 0.04	0.68 ± 0.04	**<0.01**
	Stereoacuity (logMAR)		-0.11 ± 0.20	0.03 ± 0.35	0.29 ± 0.48	0.66 ± 0.51	1.08 ± 0.53	**<0.01**
	CS (log)		1.91 ± 0.11	1.78 ± 0.17	1.64 ± 0.16	1.52 ± 0.15	1.44 ± 0.11	**<0.01**
	Aniseikonia (%)	Horizontal	1.22 ± 1.41	2.27 ± 1.62	2.82 ± 2.09	3.65 ± 2.55	3.86 ± 3.17	**<0.01**
		Vertical	1.11 ± 0.79	1.25 ± 1.45	1.54 ± 1.35	1.61 ± 1.49	2.04 ± 1.88	0.19

VA, visual acuity; CS, contrast sensitivity; D, diopter; SD, standard deviation; RM, repeated measure

Comparison of Visual Acuity, Stereoacuity, Contrast Sensitivity and Aniseikonia in Myopic, Hyperopic and Astigmatic Anisometropia

Steeper slope value (+0.27) of VA (Figure 2) was observed in myopic anisometropia as compared to the other groups, indicating that VA of myopic anisometropia rapidly degrades with the increment of myopic anisometropia magnitude. A similar pattern was also observed in stereoacuity (Figure 3) as it worsens with an increase in anisometropia magnitude, particularly in myopic anisometropia (+0.35) compared to hyperopic (+0.32) and astigmatic anisometropia (+0.30). Higher anisometropia magnitude also reduced the CS (Figure 4) in myopic anisometropia (-0.23) followed by hyperopic (-0.20) and astigmatic anisometropia (-0.13). For horizontal aniseikonia (Figure 5; top), the highest slope value (+0.88) was demonstrated by myopic group, followed by astigmatic (+0.72) and hyperopic anisometropia (+0.59). Meanwhile for vertical aniseikonia (Figure 5; bottom), astigmatic group showed the lowest slope value (+0.19), as there was insignificant increment in aniseikonia percentage with the increment of anisometropia magnitudes. Table II shows the slope value for each visual function and aniseikonia in each anisometropia group.

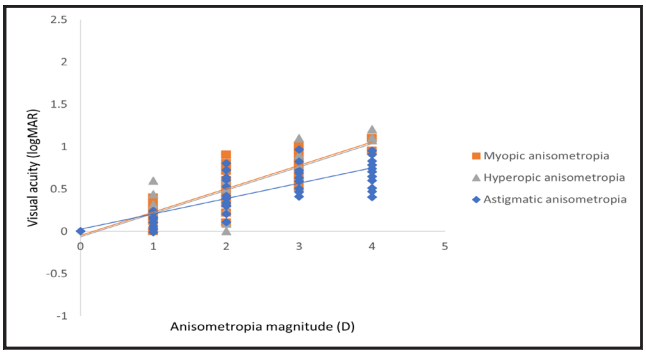

Figure 2: Line graph corresponds to visual acuity versus anisometropia magnitude.

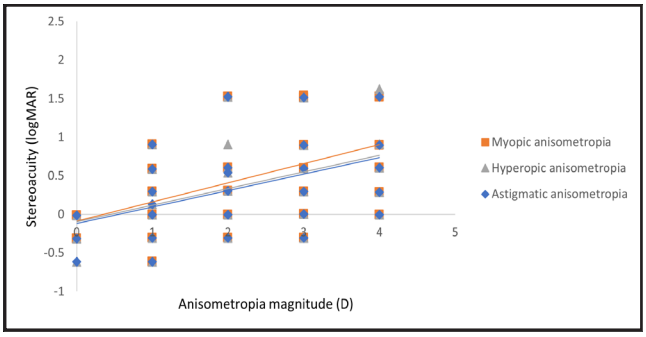

Figure 3: Line graph corresponds to stereoacuity versus anisometropia magnitude.

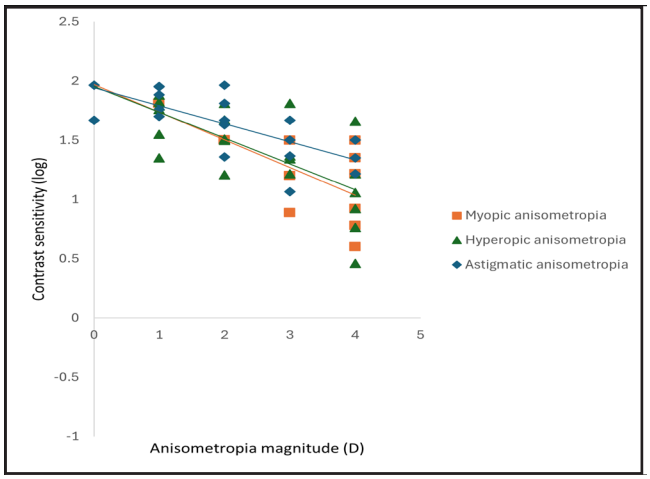

Figure 4: Line graph corresponds to contrast sensitivity versus anisometropia magnitude.

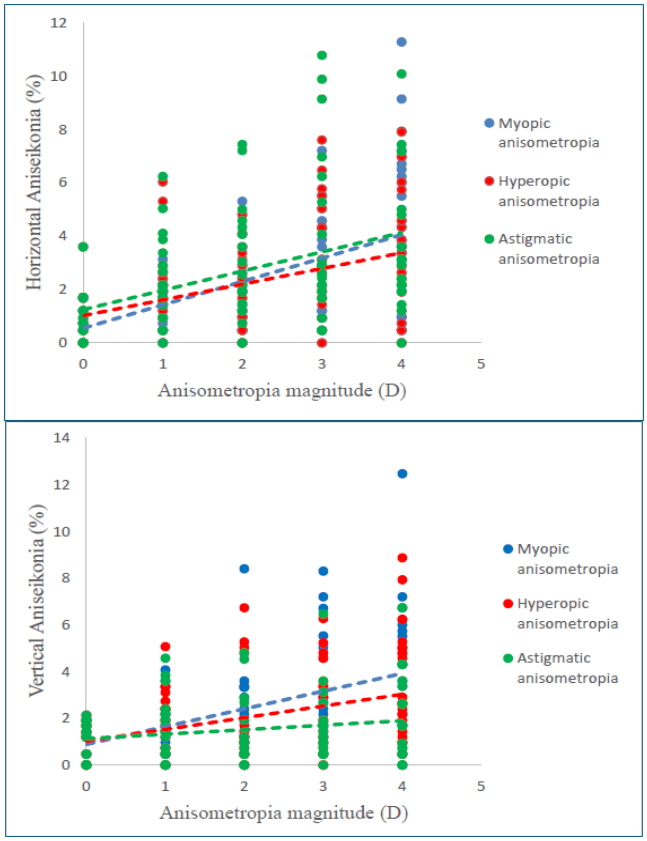

Figure 5: Aniseikonia on horizontal meridian (top) and vertical meridian (bottom) versus anisometropia magnitude.

Table II: Slope value of all parameters measured in induced myopic, hyperopic and astigmatic anisometropia

Anisometropia group	Slope value				
	Visual acuity	Stereoacuity	Contrast sensitivity	Aniseikonia	
				Horizontal	Vertical
Myopic	+0.27	+0.35	-0.23	+0.88	+0.76
Hyperopic	+0.27	+0.32	-0.20	+0.59	+0.50
Astigmatic	+0.18	+0.30	-0.13	+0.72	+0.19

DISCUSSION

This study aimed to determine the impact of induced anisometropia on visual functions (VA, stereoacuity and CS) and aniseikonia among myopic, hyperopic and astigmatic anisometropia groups. Overall, increasing the magnitude of induced myopic, hyperopic and astigmatic anisometropia resulted in substantial decreased in all three parameters of visual functions. Besides, increment in the percentage of aniseikonia in both horizontal and vertical meridians were also observed with the increment of anisometropia magnitude of all the anisometropia groups.

Myopic anisometropia

Increasing myopic anisometropia magnitudes resulted in loss of visual functions and significant aniseikonia. With maximum magnitude of anisometropia induced (4D), VA in myopic anisometropia degraded to +1.00logMAR. This finding was in line with the result demonstrated by Singh et al. (2015) which showed that VA reduced to +0.94logMAR on inducing myopia at the highest magnitude (+3D blur). Meanwhile, stereoacuity was found to degraded more than 10 lines (corresponding to more than 1.0 logMAR in VA) with the inducing of 4D myopic anisometropia. Stereoacuity statistically impaired at all magnitudes (from 1D to 4D) as the level of myopic anisometropia magnitude increased. The similar findings was found in previous study conducted by Kiran et al. (2022) where they concluded that myopic anisometropia negatively impact binocular interaction, and the level of interference is proportional to the magnitude of anisometropia. In this study, CS started to deteriorate at 3D magnitude of anisometropia in myopic groups. Hence, it could be inferred that for myopic anisometropia, stereoacuity was the most significantly impacted visual function, followed by VA and CS.

Hyperopic anisometropia

For hypermetropic anisometropia, with maximum magnitude of anisometropia induced (4 D), VA degraded to +1.00logMAR. Comparing the hyperopic anisometropia group with this current study, study by Singh et al. (2015) demonstrated insignificant reduction in VA following hyperopic change, in which they postulated that induced hyperopia, especially with the used of lower minus lenses was overcome by the accommodation system. On the other hand, the current study paralysed the accommodation of all participants to avoid any substantial influence on the findings. This might be the reason for the same effect of VA deduction in hyperopic and myopic anisometropia groups manifested in this study. Result obtained from this study showed clinically acceptable stereoacuity measurement (60 sec) was sustained at 1D induced anisometropia in hyperopic but started to reduce with 2D optical blur induced. However, at maximum magnitude, stereoacuity showed greater effect than visual acuity, in which the stereoacuity was diminished or degraded more than 10 lines of acuity. CS started to deteriorate at 3D magnitude of anisometropia in hyperopic anisometropia groups. Similar to myopic anisometropia group, higher hypermetropic anisometropia affected stereoacuity most

as compared to the other visual functions.

Astigmatic anisometropia

With maximum magnitude of anisometropia induced (4D), VA degraded to +0.68logMAR in astigmatic anisometropia, which is better as compared to myopic and hyperopic anisometropia groups. Result obtained from this study showed clinically acceptable stereoacuity measurement (60 sec) was sustained at 1D induced anisometropia in astigmatic groups but started to reduce with 2D optical blur induced. However, the loss of stereoacuity at highest magnitude of anisometropia induced (corresponded to 10 lines of acuity) was comparable to myopic and hypermetropic groups. Related to the axis of astigmatic anisometropia, Atchison et al. (2020) compared the stereoacuity between two meridians of cylindrical anisometropia (90° and 180°) and revealed that stereoacuity become worse as the cylindrical axis rotated from 180° to 90°, proving the axes of meridional anisometropia may also affect the stereoacuity. In this study, astigmatic anisometropia group maintain CS threshold at all magnitudes. Hence, it can be concluded that contrast sensitivity was the least affected by the higher astigmatic anisometropia magnitude, followed by visual acuity and stereoacuity.

Impact of induced anisometropia on visual acuity, stereoacuity, contrast sensitivity and aniseikonia

Upon inducing optical blur, decrease in VA occurred due to inaccurate adjustment of the focal length, causing loss of resolution of the target seen (28). Spang & Fahle (2009) suggested that interruption in spatial resolution, temporal and spatial frequencies in anisometropic eye caused the reduction in VA. Blurred vision in the weaker eye (eye with the higher refractive error) resulted in fewer activated neuron, weakening the activation of primary visual cortex (V1), thus disturbing normal processing in V1 (Spang & Fahle, 2009).

Stereoacuity was affected most compared to other visual functions as magnitude of anisometropia increased (Table II). It is well known that individual with higher anisometropia usually encountered binocularity problem due to the difference in retinal image size formed on the retina, which make it challenging for the brain to fuse the image into a single, three-dimensional image (30). Hoosen et al. (2020) proposed that the reduction in stereoacuity with increasing magnitude of anisometropia resulted from corresponding increment of defocus, reduced contrast and/or retinal image degradation.

Besides, foveal suppression in the eye with higher refractive error and lower density of fusional details were also the possible causes of stereoacuity degradation (31, 32). Current study found greater impact of spherical anisometropia on stereoacuity compared to astigmatic anisometropia, in agreement with previous studies by Nabie et al. (2019). This might be due to the result of global blur induced by spherical defocused, as compared to meridional blur in stimulated astigmatism (31, 34).

Among all the visual functions observed in this study, CS was the least affected by anisometropia. The deterioration of CS might be explained by the reduction in binocular summation of CS with induced optical blur (28, 35, 36). Besides, previous studies concluded that the factors that affect the CS in anisometropia were the monocular contrast deprivation on the eye with higher refractive error (weaker eye) and the optical magnification differences between two eyes, which caused a relative shift in the horizontal CS of the weaker eye (37, 38).

Previous researchers also suggested that the reduction in CS among myopic and hyperopic anisometropia were due to the influence of optical aberration which produce different contrast threshold, thus shifted the optimum focus into negative direction, causing loss of CS (39, 40). As this happens, it may cause difficulties in perceiving fine details and distinguishing objects in the visual environment and affecting activities that require good visual discrimination such as reading and driving. While significantly higher amount of aniseikonia was observed along horizontal meridian in all groups, along vertical meridian, insignificant amount of aniseikonia was demonstrated among astigmatic group. It could possibly be due to the image formed on the retina was parallel to the induced astigmatism of 90-degree axis, thus, minimising the aniseikonia effect along vertical meridian. Aniseikonia was reported to affect the visual function and disrupt binocular function, depending on the magnitude of aniseikonia perceived (41, 42). Clinically, symptoms associated with aniseikonia such as headache and asthenopia started to manifest at the magnitude of 3 to 5% of aniseikonia (16, 41). In this study, most of the findings at 3D and 4D of anisometropia demonstrated aniseikonia ≥ 3 %, which possibly caused binocular problems and discomfort.

Related to this, it is crucial to include the assessment of aniseikonia in vision screening protocols. This consideration could lead to improved diagnostic and therapeutic approaches, especially when considering the correction of aniseikonia as a part of anisometropia correction, which may reduce the risk for developing suppression and improving binocular visual outcomes. Moreover, the utilisation of the validated Smart Optometry application (18) enhances convenience, allowing for easy and efficient test performance of aniseikonia.

In summary, the observed impacts of mimicked anisometropia on visual functions can be attributed to several underlying mechanisms, such as disruption of binocular coordination and induced aniseikonia. Unequal refractive errors induced by the contact lenses increased the disparity in binocular signals between the

two eyes, disrupting the process of binocular integration and interfering the visual processing (43). Besides, blurring effect from the eye with higher refractive error also impede the ability of disparity detectors in the visual system to process depth information effectively (44). Different in interocular retinal image size also is a major factor which complicates the binocular fusion and depth perception, altering the visual functions in anisometropia. The aniseikonia induced will challenge the brain to merge the image into single coherent visual field, altering the visual functions (45).

This study provides valuable insights into how simulated anisometropia, created by inducing varying magnitude of anisometropia through contact lenses, impacts visual performance. However, there are some limitations that should be acknowledged, such as the demographic focus of the participants and the use of contact lenses to simulate anisometropia rather than examining the effects in individuals with naturally occurring anisometropia. Actual impact of anisometropia on visual functions might slightly differ when examine among the actual population, considering the effect of long standing anisometropia on visual functions. Besides, only adult participants were recruited in this study. The effects of anisometropia on visual functions among the usual age of population-based anisometropia, especially in children and presbyopes warrant further investigation. Besides, future studies should explore the long-term consequences of untreated anisometropia on visual function and quality of life, as well as investigate the effectiveness of various clinical interventions in alleviating the effects of anisometropia. Other visual functions such as eye movements parameters among anisometropia can also be included as part of the parameters in future research as interocular refractive error imbalance may as well affect the eye movements.

CONCLUSION

This study demonstrates that anisometropia significantly affects the visual functions and aniseikonia. These findings highlight the significance of managing anisometropia to minimise its negative impact on binocular vision and overall visual functions. Clinically, this research emphasized on the importance of comprehensive visual assessments in patients with anisometropia, especially on binocular function and aniseikonia. Correcting anisometropia and aniseikonia simultaneously, especially at the initial diagnosis of anisometropia, would minimise the risk of suppression and improve overall treatment outcomes for anisometropic patients.

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