The term “ocular dominance” has been used for sighting, sensory, and motor ocular dominance or eye preference (eyedness). Sighting dominance refers to the preferential use of one eye in different forms of visual alignment and for performing monocular activities, such as viewing through a microscope or a key hole. Sensory dominance, which is also called interocular imbalance, cannot be equated with motor eye dominance. A similar mechanism has been suggested to occur between binocular rivalry and strabismic suppression. Motor dominance is observed in the asymmetry of the vergence movements. It appears that these categories are closely related to one another and that spatial attention has a significant role in ocular dominance for these categories. However, no consensus on the relationship between motor dominance and sensory dominance has been reached.

The recent success of monovision for presbyopic correction by contact lens or cataract surgery further demonstrated the importance of ocular dominance on visual outcomes. This clinical practice is based on the assumption that it will be easier to suppress blur in the nondominant eyes than in the dominant eyes, with the dominant eyes usually being corrected for distance and the nondominant eyes for near vision.

Anisometropia, a relative difference in the refractive state of the two eyes, is not uncommon in myopic patients. If the nondominant eye is naturally more myopic than the dominant eye, it could tilt the scales in favor of monovision, in preference to other treatment modalities, as the best option to manage presbyopia.

**ABSTRACT**

**PURPOSE:** To investigate the association between ocular dominance (sighting dominance) and refractive asymmetry in phakic patients.

**METHODS:** This retrospective study included 3,012 patients with a mean age of 29.0 ± 5.3 years (range: 20 to 39 years). Refractive error was determined with cycloplegic refraction and axial length was determined with IOLMaster (Carl Zeiss Meditec, Dublin, CA). Ocular dominance was assessed using the hole-in-the-card test.

**RESULTS:** The right and left eyes were dominant in 77.7% and 22.3% of the patients, respectively. In the high anisometropia group (≥ 2.0 diopters), the nondominant eyes had significantly higher myopic spherical equivalents and longer axial lengths than the dominant eyes (P < .05). However, there were no significant differences in these parameters in the low anisometropia group.

**CONCLUSION:** The current study revealed that nondominant eyes had a greater myopic refractive error and longer axial length than the dominant eyes, especially in the patients who had high amounts of anisometropia.

We investigated the association between ocular dominance (sighting dominance) and refractive asymmetry in patients with phakic eyes.

**PATIENTS AND METHODS**

In this retrospective observational study, 6,024 eyes of 3,012 patients (1,500 men and 1,512 women; age range: 20 to 39 years) were included. The following inclusion criteria were required: orthophoria determined by the cover test, corrected distance visual acuity of 20/20 in each eye (ie, the absence of amblyopia), and corneal astigmatism within 2.50 diopters (D). Because amblyopia is frequently associated with anisometropia, only nonamblyopic patients were included in the study to avoid any effect of amblyopia on ocular dominance. Subjects were excluded from the study if they had any of the following: history of ocular surgery, history of strabismus or ptosis; and presence of any clinically significant retinal pathology, glaucoma, optic neuropathy, optic disc anomalies, or other diseases that might affect visual acuity after correction. Moreover, eyes with keratoconus, determined by the keratoconus screening test using Placido disk videokeratography (TMS-2; Tomey, Nagoya, Japan), were excluded from this study.

The hole-in-the-card test, in which the subject was given a piece of cardboard that had a central circular hole 3 cm in diameter, was used to determine ocular dominance. While wearing their spectacles, the patients were asked to hold the cardboard with both hands and view a target 5 m away through the hole with both eyes open. Each eye was then occluded in turn. When the dominant eye was covered, the target could not be seen through the hole. Alternatively, when the nondominant eye was covered, the dominant eye continued to fix the target through the aperture. This test was repeated at least three times to confirm dominance.

Cycloplegia was achieved by treatment with three drops of 1.0% cyclopentolate hydrochloride (Cylegin 1% ophthalmic solution; Santen Pharmaceuticals, Osaka, Japan) spaced 5 minutes apart. Subjective reaction was measured at least 60 minutes after the last drop of cyclopentolate was instilled. IOLMaster (Carl Zeiss Meditec, Dublin, CA) was then used to determine axial length. Refraction data were converted to spherical equivalents (SEs). The following formula was used to calculate SE: spherical power + (– cylindrical power / 2).

All experimental protocols and procedures complied with the tenets of the Declaration of Helsinki. Informed consent was obtained from each patient.

**STATISTICAL ANALYSIS**

Statistical analysis was performed using IBM SPSS Statistics software (version 20.0; IBM Corporation, Armonk, NY). The Wilcoxon signed-rank test was used to compare refractive errors (SE and cylindrical power) and axial lengths between the dominant and nondominant eyes. Anisometropia calculated at SE was further divided into five subgroups and compared using the Kruskal–Wallis test. Dunnett’s test was used when a significant difference was detected. The relationship of the dominant and nondominant eye was assessed using Spearman signed rank test and a method described by Bland and Altman in which 95% of the differences, or limits of agreement (LoA), lie between ±1.96 × the standard deviation of the mean difference. Spearman signed rank test was used to assess the correlation between the amount of SE anisometropia and the mean myopic SE in each patient. The results are presented as means ± standard deviations (SDs), and the level of statistical significance was set at a *P* value of less than .05.

**RESULTS**

Table 1 shows the results of the ocular dominance test. When the hole-in-the-card test was used to deter-
mine ocular dominance, no patients were classified as having undetermined ocular dominance. Right ocular dominance was present in 2,339 (77.7%) of the 3,012 patients and left ocular dominance was present in 673 (22.3%) patients; males were more right-eye dominant (80.1%) than females (75.3%).

The difference between the SE anisometropia and the mean myopic SE in each patient is shown in Figure 1 ($r = -0.024$, $P = .190$) and Figure 2 ($r = -0.055$, $P = .045$). The mean SE of the two eyes ranged from 0 to -13.75 D, with a population median of -5.25 D. The mean difference was 0.06 (95% LoA: -1.69 to -1.81 D; Figure 1).

The relationship between the amount of SE anisometropia and the proportion of patients with each anisometropia are shown in Figure 3: anisometropia 0.49 D or less = 53.1% (n = 1,599), 0.50 to 0.99 D = 25.0% (n = 753), 1.00 to 1.99 D = 16.5% (n = 496), 2.00 to 2.99 D = 3.7% (n = 110), and 3.00 D or greater = 1.8% (n = 54). For anisometropia less than 0.5 D, the nondominant eye was more myopic in 730 (45.7%), the dominant eye was more myopic in 691 (43.2%), and the difference was less than ±0.25 D in 178 (11.1%) of the 1,159 patients. For anisometropia 0.50 to 0.99 D, 1.00 to 1.99 D, 2.00 to 2.99 D, and 3.00 D or greater, the nondominant eye were more myopic in 407 (54.1%) of the 753 patients, 277 (55.8%) of the 496 patients, 66 (60.0%) of the 110 patients, and 33 (61.1%) of the 54 patients, respectively. The prevalence of more severe myopia in both eyes increased as the amount of anisometropia increased (Table 2). In the high anisometropia group, significantly higher myopic SE and longer axial lengths were found in the nondominant eyes than in the dominant eyes (both $P < .05$). However, in the low anisometropia group, there were no significant differences in these parameters. Additionally, significantly higher myopic SE and higher cylindrical powers and longer axial lengths were found in the high anisometropia group than in the low anisometropia group (both $P < .01$).

**DISCUSSION**

We evaluated the association between ocular dominance and refractive asymmetry. Anisometropia of less than 0.5 D was present in 1,599 (53.1%) patients and anisometropia of 2.0 D or greater was present in 164 (5.4%) patients. The nondominant eyes had a greater myopic refractive error and longer axial length than the dominant eyes, especially in the patients who had a high degree of anisometropia.

Detecting ocular dominance is of great importance when performing refractive surgery to treat presbyopia.6-8 Tests for ocular dominance include the Miles test, Porta test, or hole-in-the-card test, convergence near-point test, lens fogging technique, and camera test. Of these, the advantage of the hole-in-the-card test for testing ocular dominance is that both the clinician and patients know which eye is dominant at the conclusion of the test. We found that the right eye was more dominant than the left eye in 77.7% of the patients, and males were more right-eye dominant than females. The incidence of right-eye dominance in the literature has been reported to be 66.1%2 or 67%,10 which is close to our results demonstrating a predominance of right-eye–dominant male patients over right-eye–dominant female patients. However, although ocular dominance is thought to be stable within a given viewing situation or stimulus arrangement, it is difficult to characterize the strength and reliability of ocular dominance with the hole-in-the-card test only. Seijas et al.11 reported that the hole-in-the-card test only rarely produces results showing undermined dominance.

Originally, ocular dominance was thought to be independent of refraction.12 Eser et al.10 used the hole-in-the-card test in a retrospective study (n = 2,453; mean age: 46 ± 12 years) and found right-eye dominance in 67% and left-eye dominance in 33% of the patients but no difference in the mean SE (mean SE: -2.28 D) of both eyes.
Yang et al.\textsuperscript{13} also reported that there was no significant effect of ocular dominance on the development of myopia over a 2-year period in a population of children with anisometropia of no more than 1 D (n = 178; age range: 7 to 13 years). Thus, these studies give a consistent picture that there was no association between ocular dominance and ocular refraction with low anisometropia.

In contrast, Cheng et al.\textsuperscript{14} were the first to show that dominant eyes, determined by the hole-in-the-card test, had a significantly greater myopic SE (-5.27 ± 2.45 D) than nondominant eyes (-3.94 ± 3.1 D) (P < .001) in adult patients with myopic anisometropia (> 0.5 D) (n = 55; mean age: 30.3 ± 9.5 years). The difference was more evident in those patients with higher anisometropia (> 1.75 D). They also noted that dominant eyes were more myopic in 56% of patients with anisometropia of 1.75 D or greater.

Contrary to Cheng et al.’s findings, we observed that the nondominant eyes had significantly higher myopic SE and longer axial lengths than the dominant eyes in the high anisometropia group (> 2.0 D, n = 164) (both P < .01). However, there were no significant differences in these parameters in the low anisometropia group. In addition, there were statistically significant liner correlations between the amount of anisometropia and the mean myopic SE (Figure 2), but this might have little clinical significance. Linke et al.\textsuperscript{15} also reported a similar trend, with the nondominant eyes having a more myopic SE in anisometropic patients (n = 10,264; mean age: 34.9 ± 9.3 years). The greater likelihood of the nondominant eyes being more myopic than the dominant eyes at higher degrees of anisometropia was reported.

The limitations and pitfalls of our study must be considered. There were no relevant data available

| TABLE 2  
Refractive Error and Axial Length Between Dominant and Nondominant Eyes |
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Parameter</td>
<td>Dominant Eye</td>
<td>Nondominant Eye</td>
<td>P</td>
</tr>
<tr>
<td>All subjects (N = 3,012)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SE (D)</td>
<td>-5.50 ± 2.29</td>
<td>-5.57 ± 2.30</td>
<td>.001</td>
</tr>
<tr>
<td>Cylindrical power (D)</td>
<td>-0.85 ± 0.59</td>
<td>-0.87 ± 0.61</td>
<td>.016</td>
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<tr>
<td>Axial length (mm)</td>
<td>25.78 ± 0.85</td>
<td>25.79 ± 0.84</td>
<td>.383</td>
</tr>
<tr>
<td>Subjects with anisometropia &lt; 0.49 D (n = 1,599)</td>
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<td></td>
<td></td>
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<tr>
<td>SE (D)</td>
<td>-5.51 ± 2.30\textsuperscript{a}</td>
<td>-5.51 ± 2.29\textsuperscript{a}</td>
<td>.823</td>
</tr>
<tr>
<td>Cylindrical power (D)</td>
<td>-0.84 ± 0.60\textsuperscript{b}</td>
<td>-0.87 ± 0.63\textsuperscript{b}</td>
<td>.365</td>
</tr>
<tr>
<td>Axial length (mm)</td>
<td>25.78 ± 0.88\textsuperscript{c}</td>
<td>25.78 ± 0.85\textsuperscript{c}</td>
<td>.363</td>
</tr>
<tr>
<td>Subjects with anisometropia 0.50 to 0.99 D (n = 753)</td>
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<tr>
<td>SE (D)</td>
<td>-5.43 ± 2.09</td>
<td>-5.46 ± 2.20</td>
<td>.584</td>
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<tr>
<td>Cylindrical power (D)</td>
<td>-0.81 ± 0.54</td>
<td>-0.82 ± 0.55</td>
<td>.448</td>
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<tr>
<td>Axial length (mm)</td>
<td>25.76 ± 0.75</td>
<td>25.77 ± 0.79</td>
<td>.647</td>
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<tr>
<td>Subjects with anisometropia 1.00 to 1.99 D (n = 496)</td>
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<td></td>
<td></td>
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<tr>
<td>SE (D)</td>
<td>-5.49 ± 2.41</td>
<td>-5.62 ± 2.19</td>
<td>.197</td>
</tr>
<tr>
<td>Cylindrical power (D)</td>
<td>-0.86 ± 0.59</td>
<td>-0.84 ± 0.58</td>
<td>.448</td>
</tr>
<tr>
<td>Axial length (mm)</td>
<td>25.75 ± 0.82</td>
<td>25.80 ± 0.77</td>
<td>.056</td>
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<td>Subjects with anisometropia 2.00 to 2.99 D (n = 110)</td>
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<td></td>
</tr>
<tr>
<td>SE (D)</td>
<td>-5.49 ± 2.69</td>
<td>-5.95 ± 2.78</td>
<td>.045</td>
</tr>
<tr>
<td>Cylindrical power (D)</td>
<td>-1.07 ± 0.61\textsuperscript{b}</td>
<td>-1.11 ± 0.75\textsuperscript{b}</td>
<td>.331</td>
</tr>
<tr>
<td>Axial length (mm)</td>
<td>25.70 ± 0.94</td>
<td>25.86 ± 0.97\textsuperscript{c}</td>
<td>.023</td>
</tr>
<tr>
<td>Subjects with anisometropia &gt; 3.00 D (n = 54)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>SE (D)</td>
<td>-6.38 ± 2.51\textsuperscript{a}</td>
<td>-7.29 ± 2.98\textsuperscript{a}</td>
<td>.033</td>
</tr>
<tr>
<td>Cylindrical power (D)</td>
<td>-1.03 ± 0.72\textsuperscript{b}</td>
<td>-1.19 ± 0.62\textsuperscript{b}</td>
<td>.194</td>
</tr>
<tr>
<td>Axial length (mm)</td>
<td>26.03 ± 0.86\textsuperscript{c}</td>
<td>26.36 ± 1.03\textsuperscript{c}</td>
<td>.017</td>
</tr>
</tbody>
</table>

SE = spherical equivalent; D = diopters
\textsuperscript{a}Subjects with anisometropia > 3.00 D had significantly more myopic SE than subjects with anisometropia < 0.49 D (P < .01).
\textsuperscript{b}Subjects with anisometropia > 2.00 D had significantly higher cylindrical power than subjects with anisometropia < 0.49 D (P < .01).
\textsuperscript{c}Subjects with anisometropia > 3.00 D had significantly longer axial length than subjects with anisometropia < 0.49 D (P < .01).
in our study on the hand dominance of the patients. However, Cheng et al.\textsuperscript{14} and Mansour et al.\textsuperscript{16} found no correlation between hand dominance and refraction. The strengths of our study include the strict exclusion of ocular diseases and amblyopia, which was the result of a detailed ophthalmic examination of the patients.

It is widely accepted that the prevalence of myopia is increasing,\textsuperscript{17} and the environmental and genetic factors that contribute to its progression remain to be fully elucidated. Cheng et al.\textsuperscript{14} suspected that the extent of ocular dominance varies among individuals, and those with stronger ocular dominance may eventually develop higher amounts of anisometropia, whereas those with less dominance may not. Moreover, retinal defocus is a more recent hypothesis proposed to explain the development of myopia.\textsuperscript{18,19} For those with decreased capacity for accommodation, near work may result in retinal defocus and blur, and it is this chronic blur that leads to myopia.\textsuperscript{20} In addition, the tonic state of the ciliary smooth muscle in the dominant eyes might result in less accommodative accuracy or a greater lag of accommodation compared with that of the nondominant eyes, which could lead to greater defocus. This might explain, in part, why the dominant eyes showed more myopic refraction in the patients with high anisometropia. There is a need for further studies that evaluate the progression of myopia in the dominant eyes compared with that in the nondominant eyes.

There was no statistically significant difference between dominant and nondominant eye for astigmatism of each eye. However, the high anisometropia group had significantly higher cylindrical power than the low anisometropia group (≥ 2.0 D). Linke et al.\textsuperscript{15} reported that comprehensive data on 10,264 myopic patients confirmed the higher astigmatic power in the nondominant eyes. In addition, Chia et al.\textsuperscript{21} did not find a significant effect of ocular dominance on SE in anisometropic myopic children (n = 184) but did show that astigmatism was significantly lower in the dominant eyes of anisometropic patients. They hypothesized that intraocular astigmatic difference may result in eyes with less astigmatism (or better vision) becoming more dominant.

The nondominant eyes had a greater myopic refractive error and longer axial length than the dominant eyes, especially in patients who had a high degree of anisometropia.

**AUTHOR CONTRIBUTIONS**

Study concept and design (HI, MI, MK, KShimizu); data collection (MI, KSunaga); analysis and interpretation of data (MI, TK, KSunaga); drafting of the manuscript (MI); critical revision of the manuscript (HI, MK, TK, KShimizu, KSunaga); statistical expertise (TK); supervision (KShimizu)

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**REFERENCES**