The Relationship Between Corneal Hysteresis and Progression of Glaucoma After Trabeculectomy

Yuri Fujino, CO,*†‡§ Hiroshi Murata, MD,* Masato Matsuura, CO,*† Shunsuke Nakakura, MD, PhD,∥ Nobuyuki Shoji, MD, PhD,† Yoshiataka Nakaoo, CO, PhD,* Yoshiki Kiuchi, MD, PhD,¶ and Ryo Asaoka, MD, PhD*‡#

*Department of Ophthalmology, The University of Tokyo, Tokyo; †Department of Ophthalmology, Seirei Hamamatsu General Hospital; ‡Seirei Christopher University, Shizuoka; §Department of Ophthalmology, School of Medicine, Keio University, Tokyo; ¶Department of Ophthalmology, School of Medicine, Kitasato University, Kanagawa; ∥Department of Ophthalmology, Seirei Hamamatsu General Hospital

ORIGINAL STUDY

The purpose of this study was to investigate the association of corneal hysteresis (CH) measured with Ocular Response Analyzer on the progression of glaucoma after trabeculectomy.

Materials and Methods: Twenty-four eyes of 19 patients with primary open-angle glaucoma underwent trabeculectomy. A series of visual fields (Humphrey Field Analyzer 24-2 SITA-standard) were measured starting after 6 months after trabeculectomy (4.2 ± 0.5 months). The mean total deviation (mTD) of the 52 test points were calculated. In addition, the mTD was divided into the following areas: central area (within central 10 degrees), superior area and inferior area: mTDcentre, mTDsuperior, and mTDinferior, respectively. The relationship between each area’s progression rate of mTD and the 7 variables of baseline age, central corneal thickness, baseline mTD, mean intraocular pressure (IOP), SD of IOP divided by mean IOP, and CH were analyzed using the linear mixed model, and the optimal model was selected using the model selection method with the second order Akaike Information Criterion.

Results: In the optimal model for mTD progression rate, only CH was selected with the coefficient of 0.11. The optimal model for the mTDcentre progression rate included mean IOP with the coefficient of 0.089, and that for mTDsuperior included only CH with the coefficient of 0.089. There was no variable selected in the optimal model for the mTDinferior progression rate.

Conclusion: CH is a useful measure in the management of glaucoma after trabeculectomy.

Key Words: glaucoma, trabeculectomy, visual field, corneal hysteresis, ocular response analyzer

(J Glaucoma 2020;29:912–917)
progression of glaucoma after trabeculectomy should be investigated, in particular in relation with CCT and IOP control.

The purpose of this study was to investigate the possible association of CH with the long-term progression of glaucoma after trabeculectomy, in relation with CCT and IOP control. In addition, the clinical factors related to VF progression in superior and inferior hemifields may not be identical. For instance, previous studies have reported that the inferior VF is predominantly affected in nonarteritic anterior ischemic optic neuritis.48 We have recently reported that smoking habit was found to be related to progression predominantly in the inferior VF, suggesting that ischemia may be related to VF damage in the inferior hemifield.49 Thus, we also investigated the effect of CH on VF progression after trabeculectomy, in the superior and inferior hemifields, separately.

MATERIALS AND METHODS

This study was approved by the Research Ethics Committee of the Graduate School of Medicine and Faculty of Medicine at the University of Tokyo. Written informed consent was given by participants for their information to be stored in the hospital database and used for research. This study was performed in accordance with the tenets of the Declaration of Helsinki.

Subjects

The study population consisted of 24 eyes of 19 POAG patients. All study participants underwent trabeculectomy and had at least 5 subsequent VF measurements starting after at least 6 months after trabeculectomy between 2003 and 2018 at the University of Tokyo Hospital. All subjects underwent complete ophthalmic examinations, including biomicroscopy, gonioscopy, IOP measurement, funduscopy, refraction, best-corrected visual acuity measurements, and axial length measurements, as well as ORA and VF testing [Humphrey Field Analyzer (HFA); Carl Zeiss Meditec Inc., Dublin, CA]. IOP measurement was conducted at each of patients’ visits (>3 mo interval), whereas VF measurement was performed approximately once every 2 visits. ORA measurement was carried out once in the observation period.

POAG was defined as (1) presence of typical glaucomatous changes in the optic nerve head such as a rim notch with a rim width ≤0.1 disc diameters or a vertical cup-to-disc ratio of >0.7 and/or a retinal nerve fiber layer defect with its edge at the optic nerve head margin greater than a major retinal vessel, diverging in an arcuate or wedge shape and (2) gonioscopically wide open angles of grade 3 or 4 based on the Shaffer classification. Exclusion criteria were: (1) age below 20 years; (2) possible secondary ocular hypertension in either eye; (3) visual acuity ≤0.5 LogMAR.50 Thus, the diagnosis of POAG was made irrespective of the presence of glaucomatous VF change, so that patients with a large range of glaucomatous damage were enrolled in the study, including those without measurable VF damage. Subjects with other systemic or ocular disorders that could affect the VF results were carefully excluded. IOP measurements were performed using GAT. Eyes with surgical intervention, including needling bleb revision and reoperation of trabeculectomy during the VF observation period were included. Eyes with surgical procedures performed at least 6 months before the initiation of the VF observation period were excluded. Eyes with cataract were carefully excluded, except for clinically insignificant cataract. Being extracted from patients’ history in the clinical charts, baseline IOP was measured twice on different days without any antiglaucomatous medication or surgical treatment including laser treatment, and the average value was calculated. Mean IOP was calculated as the mean value of the all IOP record during the observation period. The difference between baseline IOP and mean IOP was calculated (ΔIOP).

VF Data

VF testing was performed using the HFA with the 24-2 or 30-2 program (SITA-Standard and Goldmann III target). All participants were subjected to near-refractive correction and had previous experience in VF examinations. Unreliable VFs defined as fixation losses >20%, or false-positive responses >15% were excluded, following the manufacturer’s recommendation. The mean total deviation (mTD) of the 52 test points in the 24-2 HFA VF test pattern was calculated. In addition, the VF was divided into the following 3 areas: central area (central 12 points within 10 degrees), superior area and inferior area. The mTD was calculated for each area (Fig. 1, mTD_centre, mTD_superior, and mTD_inferior).

ORA (CH) Data

ORA records 2 unplanned measurements, before and after application of an air puff. The cornea resists the air puff because of its viscoelastic property, which results in a measurement difference between the 2 application pressures. This difference is called CH.52 All ORA data had a quality index of >7.5 as recommended by the manufacturer. The ORA measurement was performed thrice on the same day within 3 months from the last VF measurement, and the average value was used for analysis.

CCT Data

CCT was derived from the CorvisST tonometry (Oculus, Wetzlar, Germany) measurement. It was considered reliable according to the “OK” quality index displayed on the CorvisST tonometry monitor. The average value of 3 measurements was used for analysis.
Statistical Analysis

Univariate analyses between the mTD progression rate and the 7 variables of baseline age, CCT, baseline mTD, mean IOP, SD of IOP divided by the mean IOP (SD/mean IOP), ΔIOP and CH were performed using the linear mixed model in which patients were treated as a “random effect.” Subsequently, as a primary analysis, a multivariate analysis using the linear mixed model was performed to examine the relationship between the progression rate of mTD and the aforementioned 7 variables where both the intercept and slope was explained using the 7 variables. The optimal model was selected using the model selection method with the second ordered Akaike Information Criterion (AIC), from 27 combinations. The AIC is a well-established statistical measure used in model selection, and the AIC is its corrected type, providing an accurate estimation especially when the sample size is small. In a linear regression model, the degree of freedom decreases as the number of variables increases. Hence, model selection methods should be used when the number of variables is large.

As subanalyses, similar analyses were performed using the regional progression rates of mTDcentre, mTDsuperior, and mTDinferior.

All statistical analyses were performed using the statistical programming language R (version 3.1.3; The R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

The characteristics of the study population are summarized in Table S1 (Supplemental Digital Content 1, http://links.lww.com/IJG/A402). The mean (± SD) patient age at baseline was 54.8 ± 10.6 years (range: 38 to 79 y). Ten patients were male, while 9 patients were female. All VFs [11.7 ± 4.4 (range: 5 to 21) VFs per eye] were measured over an average period of 8.9 ± 3.8 years (range: 2.2 to 14.2 y). The mTD value at baseline was −13.5 ± 5.41 dB (range: −22.4 to −3.81 dB) and the progression rate of mTD was −0.22 ± 0.47 dB/y (range: −1.23 to 1.04 dB/y) (Fig. 2). The CH value was 9.4 ± 1.2 mm Hg (range: 7.5 to 12.5 mm Hg). During this observation period, IOP measurement was conducted [49.3 ± 20.3 (range: 11 to 82) times per eye]. Baseline IOP was 17.6 ± 3.65 mm Hg (range: 13.0 to 29.3 mm Hg), whereas mean IOP was 12.1 ± 3.12 mm Hg (range: 5.63 to 17.4 mm Hg) and ΔIOP was 5.57 ± 3.27 mm Hg (0.093 to 14.3 mm Hg) and SD of IOP divided by the mean IOP was 0.17 ± 0.68 (0.08 to 0.29). Fifteen eyes of 13 patients received IOP lowering medications (2.1 ± 0.88 medications) at the last visit in the observation period.

Table S2 (Supplemental Digital Content 1, http://links.lww.com/IJG/A402) shows the results of the univariate relationships between various mTD progression rates (mTD, mTDcentre, mTDsuperior, and mTDinferior) and baseline age, CCT, baseline mTD, mean IOP, SD/mean IOP, ΔIOP, and CH, using the linear mixed model.

As shown in Table S3 (Supplemental Digital Content 1, http://links.lww.com/IJG/A402), in the optimal model for the mTD progression rate, only CH was selected with the coefficient of 0.12 (SE: 0.054). The remaining 6 variables (baseline age, CCT, baseline mTD, mean IOP, SD/mean IOP and ΔIOP) were not included in this model.

The variables selected in the optimal models for the mTDcentre progression rate, were mean IOP with the coefficient of −0.043 (linear mixed model, SE: 0.024) and CH with the coefficient of 0.12 (linear mixed model, SE: 0.066).

FIGURE 2. Histogram of the mean of total deviation (mTD) progression rate. The progression rate of mTD was −0.22 ± 0.47 dB/y.

There was no variable selected in the optimal model for the mTDsuperior progression rate.

The only variable selected in the optimal models for the mTDinferior was CH with the coefficient of 0.089 (linear mixed model, SE: 0.040).

DISCUSSION

In the current study, the relationship between CH and VF progression after trabeculectomy was investigated in 24 eyes of 19 POAG patients. The results showed that ORA-CH was useful when analyzing the mTD progression rate; rapid mTD progression rate with low CH. The remaining variables (ie, baseline age, CCT, baseline mTD, mean IOP during the follow-up period, SD/mean IOP during the follow-up period and baseline IOP before treatment were not included in the optimum model for the mTD progression rate. In addition, in the current study, this analysis was iterated by dividing the VF into 3 regions: central 10 degrees, superior and inferior hemifields outside 10 degrees. Consequently, CH and mean IOP were shown to be related to VF progression in the central region, whereas CH was related to VF progression in the outer inferior hemifield. In contrast, none of the investigated variables were included in the optimum model for the progression rate in the outer superior hemifield.

We recently reported the usefulness of CH in the analysis of mTD progression in glaucomatous eyes without trabeculectomy, in which the mean CH value was 9.2 mm Hg. Previous reports have suggested that CH increases after trabeculectomy. Nonetheless, a very similar average CH value was observed in the current study and the previous studies, respectively. This may be because the change in CH after trabeculectomy is largely dependent on the change of IOP, whereas the mean IOP values were similar between our previous and current studies (13.5 vs. 12.1 mm Hg, respectively). Of note, the mean IOP, as well as the fluctuation of IOP, is likely to influence the progression of VF, whereas the values of the SD of IOP were similar between our previous and current studies (1.6 vs. 1.9 mm Hg). Previous studies have suggested that IOP is less...
variable after trabeculectomy. However, this finding was not observed in the current study. Moreover, in the current study, ΔIOP was not related to VF progression. As shown in many previous studies, there is no doubt that high IOP is a risk factor for the progression of glaucoma. In contrast, the current results suggested that the mean IOP was not significantly related to the progression of mTD. However, this finding does not deny the importance of IOP in the management of glaucoma since, in the current study, IOP was well controlled in all eyes through trabeculectomy. This result is consistent with our previous findings from a multicentre study, in which real-world clinical data were analyzed, and there was no relationship identified between the mean IOP and progression of glaucoma. It should also be noted that in the previous study, it was suggested that the SD of IOP was significantly related to the progression of mTD. However, this finding was not observed in eyes with a mean IOP < 15 mm Hg. Although in the current study the mean IOP value was at this level, the SD of IOP was not significantly related to the progression of VF.

Furthermore, CCT and AL have been reported to influence the progression of VF. and and age were very similar between our previous and current studies (531 vs. 533 μm and 25.1 vs. 25.2 mm, respectively). In contrast, the baseline mTD value (~6.8 vs. ~13.5 dB, respectively) and age were very different between the 2 studies (63.2 vs. 54.8 y, respectively). These differences may reflect differences in the nature of the 2 studies. Despite these differences, similar tendencies were observed in these studies: CH was significantly related to the progression of mTD in both studies, whereas mean IOP, SD of IOP, baseline mTD and CCT were not. This observation indicates the usefulness of CH in the management of glaucoma both in eyes without trabeculectomy and after trabeculectomy. We recently reported different patterns of VF damage and progression rate between POAG and primary angle-closure glaucoma, agreeing with other previous reports. This implies the presence of different pathologic mechanisms in the development of superior and inferior VF damages. Indeed, inferior VF is predominantly affected in nonarteritic anterior ischemic optic neuritis. In addition, smoking was found to be related to progression predominantly in the inferior VF. The current study suggested that CH was significantly related to VF progression in the inferior hemifield, unlike in the superior hemifield. Previous studies suggested that superior VF damage is more attributed to elevated IOP than that occurring in the inferior hemifield. As described above, the effect of IOP on the progression of VF was no longer observed because of the nature of the current study. However, CH was significantly related to VF progression in the inferior hemifield. The primary reason for the relationship between CH and the progression of glaucoma remains unclear. However, daily life activities such as postural change, eyeh blink, ocular pulsatility due to ocular hemodynamics, Valsalva maneuver and eye movement exert stress to eyes and may lead to deformation. An eye with high hysteresis is more likely to absorb these external strains with the damping capacity, and indeed we recently reported a significant relationship between the damping capacity and progression of VF. In addition, the cornea and sclera are continuous collagenous structures of an eye with similar biomechanical characteristics, because embryologically, the sclera and Bruch membrane are both derived from the neural crest. Our results suggest that the VF progression in the inferior hemifield is related not only to elevated IOP, but also other factors, such as CH.

In contrast to the outer VF regions, the mean IOP was significantly related to VF progression in the central 10 degrees. The number of retinal ganglion cells corresponds to the area is much larger than those of the outer regions. Indeed, this region usually maintains visual sensitivity until the last stage of glaucoma. The reason for the significant effect of the mean IOP in this region—despite the probable larger number of remaining retinal ganglion cells—is unclear. However, this may be attributed to treatment decision bias, as the indication of trabeculectomy was decided mainly using the tendency in the entire VF with the HFA 24-2 test. This area is directly related to the deterioration of the quality of life in patients with glaucoma. Thus, the inclusion of CH in the optimal model for the progression rate in this region suggests further usefulness of CH in the management of glaucoma. Furthermore, the importance of this region can be more emphasized after trabeculectomy, since only this area may remain in the last stage of the disease.

A limitation of the current study is the lack of CH measurement before trabeculectomy. Further studies including this parameter are warranted. Another limitation of the current study is the effect of antiglaucoma eye drops on corneal biomechanical properties. This is because it has been reported that anti-IOP agents can change the cornea’s biomechanical properties.

In conclusion, the current results suggested that CH is a useful measure in the management of glaucoma, even after trabeculectomy.

REFERENCES

1. Congdon N, O’Colmain B, Klaver CC, et al. Causes and prevalence of visual impairment among adults in the United States. Arch Ophthalmol. 2004;122:477–485.
2. Quigley HA. Number of people with glaucoma worldwide. Br J Ophthalmol. 1996;80:389–393.
3. The AGIS Investigators. The Advanced Glaucoma Intervention Study: the relationship between control of intraocular pressure and visual field deterioration. Am J Ophthalmol. 2000;130:429–440.
4. Garway-Heath DF, Crabb DP, Bunce C, et al. Latanoprost for open-angle glaucoma (UKGTS): a randomised, multicentre, placebo-controlled trial. Lancet. 2015;385:1295–1304.
5. Heijl A, Leske MC, Bengtsson B, et al. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. Arch Ophthalmol. 2002;120:1268–1279.
6. Holmin C, Thorburn W, Krakau CE. Treatment versus no treatment in chronic open angle glaucoma. Acta Ophthalmol (Copenh). 1988;66:170–173.
7. Leske MC, Heijl A, Hyman L, et al. Predictors of long-term progression in the early manifest glaucoma trial. Ophthalmology. 2007;114:1965–1972.
8. Musch DC, Gillespie BW, Lichter PR, et al. The CIGTS Study Investigators. Visual field progression in the Collaborative Initial Glaucoma Treatment Study: the impact of treatment and other baseline factors. Ophthalmology. 2009;116:200–207.
9. Pajic B, Pajic-Eggspuehler B, Halfliger IO. Comparison of the effects of dorzolamide/timolol and latanoprost/timolol fixed combinations on intraocular pressure and progression of visual field damage in primary open-angle glaucoma. Curr Med Res Opin. 2010;26:2213–2219.
10. Cairns JE. Trabeculectomy. Preliminary report of a new method. Am J Ophthalmol. 1968;66:673–679.
11. Lichter PR, Musch DC, Gillespie BW, et al. Interim clinical outcomes in the Collaborative Initial Glaucoma Treatment Study comparing initial treatment randomized to medications or surgery. Ophthalmology. 2001;108:1943–1953.
12. Membrety WL, Bunce C, Poinoosawmy DP, et al. Glaucoma surgery with or without adjunctive antiproliferatives in normal
tension glaucoma: a visual field progression. Br J Ophthalmol. 2001;85:696–701.
13. Werner EB, Durance SM, Schulzer M. Trabeculotomy and the progression of glaucomatous visual field loss. Arch Ophthalmol. 1977;95:1374–1377.
14. Greve EL, Duke CL. Four year follow-up of a glaucoma operation. Prospective study of the double flap Scheie. Int Ophthalmol. 1979;1:139–145.
15. Kidd MN, O’Connor M. Progression of field loss after trabeculotomy: a five-year follow-up. Br J Ophthalmol. 1985;69:827–831.
16. Aoyama A, Ishida K, Sawada A, et al. Target intraocular pressure for stability of visual field loss progression in normal-tension glaucoma. Jpn J Ophthalmol. 2010;54:117–123.
17. Fujino Y, Asaoka R, Murata H, et al. Evaluation of Glaucoma Progression in Large-Scale Clinical Data: The Japanese Archive of Multicentennial Databases in Glaucoma (JAMDIG). Invest Ophthalmol Vis Sci. 2016;57:2012–2020.
18. Musch DC, Gillespie BW, Niziol LM, et al. CIGTS Study Group. Intraocular pressure control and long-term visual field loss in the Collaborative Normal-Tension Glaucoma Treatment Study. Ophthalmology. 2011;118:1766–1773.
19. Collaborative Normal-Tension Glaucoma Study Group. Comparison of glaucomatous progression between untreated patients with normal-tension glaucoma and patients with therapeutically reduced intraocular pressures. Am J Ophthalmol. 1998;126:487–497.
20. Bhan A, Tan AC, Shah MA. Effect of corneal thickness on intraocular pressure measurements with the pneumotonometer, Goldmann applanation tonometer, and Tono-Pen. Invest Ophthalmol Vis Sci. 2002;43:1389–1392.
21. Ehlers N, Bramsen T. Importance of corneal thickness in applanation tonometry [proceedings]. Acta Ophthalmol Suppl. 1975;32:33–32.
22. Feltgen N, Leifert D, Funk J. Correlation between central corneal thickness, applanation tonometry, and direct intraocular pressure IOP readings. Br J Ophthalmol. 2001;85:85–87.
23. Foster PJ, Baasanhu J, Alsibk PH, et al. Central corneal thickness and intraocular pressure in a Mongolian population. Ophthalmology. 1998;105:969–973.
24. Gunvant P, Baskaran M, Vijaya L, et al. Effect of corneal parameters on applanation tonometry using the pulsatile ocular blood flow tonograph and Goldmann applanation tonometer. Br J Ophthalmol. 2004;88:518–522.
25. Kotecha A, Elsheikh A, Roberts CR, et al. Corneal thickness and age-related biomechanical properties of the cornea measured with the ocular response analyzer. Invest Ophthalmol Vis Sci. 2006;47:3337–3347.
26. Liu J, Roca-Cortes O. Corneal biomechanical properties on intraocular pressure measurement: quantitative analysis. J Cataract Refract Surg. 2005;31:146–155.
27. Shah S, Chatterjee A, Mathai M, et al. Relationship between corneal thickness and measured intraocular pressure in a general ophthalmology clinic. Ophthalmology. 1999;106:2154–2160.
28. Shimmyo M, Ross AJ, Moy A, et al. Intraocular pressure, Goldmann applanation tension, corneal thickness, and corneal curvature in Caucasians, Asians, Hispanics, and African Americans. Am J Ophthalmol. 2003;136:603–613.
29. Stodmeister R. Applanation tonometry and correction according to corneal thickness. Acta Ophthalmol Scand. 1998;76:319–324.
30. Tonu PA, Ho T, Newton T, et al. The influence of central corneal thickness and age on intraocular pressure measured by pneumotonomometry, non-contact tonometry, the Tono-Pen XL, and Goldmann applanation tonometry. Br J Ophthalmol. 2005;89:851–854.
31. Whittacre MM, Stein R. Sources of error with use of Goldmann-type tonometers. Surv Ophthalmol. 1993;38:1–30.
32. Wolfs RC, Klaver CC, Vingerling JR, et al. Distribution of central corneal thickness and its association with intraocular pressure: The Rotterdam Study. Am J Ophthalmol. 1997;123:767–772.
33. Jonas JB, Holbach A. Central corneal thickness and the thickness of the lamina cribrosa in human eyes. Invest Ophthalmol Vis Sci. 2005;46:1275–1279.
34. Ku JY, Danesh-Meyer HV, Craig JP, et al. Comparison of intraocular pressure measured by Pascal dynamic contour tonometry and Goldmann applanation tonometry. Eye. 2006;20:191–198.
35. Weinreb RN, Brandt JD, Garway-Heath D, et al. Intraocular pressure: Kugler Publications; 2007.
36. Lascaratos G, Garway-Heath DF, Russell RA, et al. Intraocular pressure (IOP) measured with the Ocular Response Analyzer is a better predictor of glaucoma progression than Goldmann IOP in the United Kingdom Glaucoma Treatment Study (UKGTS). Invest Ophthalmol Vis Sci. 2014;55:128.
37. Abitbol O, Bouden J, Doan S, et al. Corneal hysteresis measured with the Ocular Response Analyzer and patients with normal-tension glaucoma: Acta Ophthalmol. 2010;88:116–119.
38. Congdon NG, Broman AT, Bandeen-Roche K, et al. Central corneal thickness and corneal hysteresis associated with glaucoma damage. Am J Ophthalmol. 2006;141:868–875.
39. De Moraes CVC, Hill V, Tello C, et al. Lower corneal hysteresis is associated with more rapid glaucomatous visual field progression. J Glaucoma. 2012;21:209–213.
40. Mansouri K, Leite MT, Weinreb RN, et al. Association between corneal biomechanical properties and glaucoma severity. Am J Ophthalmol. 2012;153:419–427.
41. Medeiros FA, Meira-Freitas D, Liboa R, et al. Corneal hysteresis as a risk factor for glaucoma progression: a prospective longitudinal study. Ophthalmology. 2013;120:1533–1540.
42. Wells AP, Garway-Heath DF, Poostchi A, et al. Corneal hysteresis but not central corneal thickness correlates with optic nerve surface compliance in glaucoma patients. Invest Ophthalmol Vis Sci. 2008;49:3262–3268.
43. Hirasa K, Matsuura M, Murata H, et al. Association between corneal biomechanical properties with corneal response analyzer and also CorvisST tonometry, and glaucomatous visual field severity. Trans Vis Sci Technol. 2017;6:18.
44. Matsuura M, Hirasa K, Murata H, et al. The usefulness of CorvisST tonometry and the Ocular Response Analyzer to assess the progression of glaucoma. Sci Rep. 2017;7:40798.
45. Pillunat KR, Spoerl E, Terai N, et al. Corneal biomechanical changes after trabeculotomy and the impact on intraocular pressure measurement. J Glaucoma. 2017;26:278–282.
46. Pakravan M, Afrozifard M, Yazdani S. Corneal biomechanical changes following trabeculectomy and phaco-trabeculectomy, Ahmed glaucoma valve implantation and phacoemulsification. J Ophthalmic Vis Res. 2014;9:7–13.
47. Huang C, Zhang M, Huang Y, et al. Corneal hysteresis is correlated with reduction in axial length after trabeculectomy. Curr Eye Res. 2012;37:381–387.
48. Hayreh SS, Zimmerman B. Visual field abnormalities in nonarteritic anterior ischemic optic neuropathy: their pattern and prevalence at initial examination. Arch Ophthalmol. 2005;123:1554–1562.
49. Asaoa K, Murata H, Fujino Y, et al. Effects of ocular and systemic factors on the progression of glaucomatous visual field damage in various sectors. Br J Ophthalmol. 2017;101:1071–1075.
50. Matsuura K, Hirasa K, Murata H, et al. The relationship between visual acuity and the reproducibility of visual field measurements in glaucoma patients. Invest Ophthalmol Vis Sci. 2015;56:5630–5635.
51. Bengtsson B, Heijl A. False-negative responses in glaucoma perimetry: indicators of patient performance or test reliability? Invest Ophthalmol Vis Sci. 2000;41:2201–2204.
52. Terai N, Raikup F, Haustein M, et al. Identification of biomechanical properties of the cornea: the ocular response analyzer. Curr Eye Res. 2012;37:553–562.
53. Burnham KP, Anderson DR. Multimod her inference: understanding AIC and BIC in model selection. Socio Methods Res. 2004;33:261–304.
54. Tibshirani RJ, Taylors J. Degrees of freedom in lasso problems. J Curr Eye Res. 2004;10:78–107.
55. Mallow C. Some comments on Cp. Technometrics. 1973;15:661–675.
56. Krupin T, Liebmann JM, Greenfield DS, et al. Low-Pressure Glaucoma Study Group. A randomized trial of brimonidine versus timolol in preserving visual function: results from the
Low-Pressure Glaucoma Treatment Study. *Am J Ophthalmol.* 2011;151:671–681.

57. Migdal C, Gregory W, Hitchings R. Long-term functional outcome after early surgery compared with laser and medicine in open-angle glaucoma. *Ophthalmology.* 1994;101:1651–1656; discussion 1657.

58. Jay JL, Murray SB. Early trabeculectomy versus conventional management in primary open angle glaucoma. *Br J Ophthalmol.* 1988;72:881–889.

59. Fujino Y, Asaoka R, Murata H, et al. Evaluation of glaucoma progression in large-scale clinical data: the Japanese Archive of Multicentral Databases in Glaucoma (JAMDIG). *Invest Ophthalmol Vis Sci.* 2016;57:2012–2020.

60. Aoki S, Murata H, Matsuura M, et al. The effect of air pulse-driven whole eye motion on the association between corneal hysteresis and glaucomatous visual field progression. *Sci Rep.* 2018;8:2969.

61. Aoki S, Murata H, Nakakura S, et al. Correlation between elastic energy stored in an eye and visual field progression in glaucoma. *PLoS One.* 2018;13:e0204451.

62. Yousefi S, Sakai H, Murata H, et al. Asymmetric patterns of visual field defect in primary open-angle and primary angle-closure glaucoma. *Invest Ophthalmol Vis Sci.* 2018;59:1279–1287.

63. Yousefi S, Sakai H, Murata H, et al. Rates of visual field loss in primary open-angle glaucoma and primary angle-closure glaucoma: asymmetric patterns. *Invest Ophthalmol Vis Sci.* 2018;59:5717–5725.

64. Lee YH, Kim CS, Hong SP. Rate of visual field progression in primary open-angle glaucoma and primary angle-closure glaucoma. *Korean J Ophthalmol.* 2004;18:106–115.

65. O’Brien C, Schwartz B. The visual field in chronic open angle glaucoma: the rate of change in different regions of the field. *Eve.* 1990;4(pt 4):557–562.

66. Quigley HA, Tielsch JM, Katz J, et al. Rate of progression in open-angle glaucoma estimated from cross-sectional prevalence of visual field damage. *Am J Ophthalmol.* 1996;122:355–363.

67. Yang CB, Myers JS, Herndon LW, et al. Rate of progression in open-angle glaucoma estimated from cross-sectional prevalence of visual field damage. *Am J Ophthalmol.* 1997;123:426–428.

68. Wang YX, Jiang R, Wang NL, et al. Acute persipillary retinal pigment epithelium changes associated with acute intraocular pressure elevation. *Ophthalmology.* 2015;122:2022–2028.

69. Ritch R. A unification hypothesis of pigment dispersion syndrome. *Trans Am Ophthalmol Soc.* 1996;94:381.

70. Singh K, Dion C, Wajsalber M, et al. Measurement of ocular fundus pulsation in healthy subjects using a novel Fourier-domain optical coherence tomography. *Invest Ophthalmol Vis Sci.* 2011;52:8927–8932.

71. Kim YW, Girard MJ, Mari JM, et al. Anterior displacement of lamina cribrosa during valsalva maneuver in young healthy eyes. *PLoS One.* 2016;11:e0159663.

72. Wang X, Rumpel H, Lim WEH, et al. Finite element analysis predicts large optic nerve head strains during horizontal eye movementseye movements induce optic nerve head strains. *Invest Ophthalmol Vis Sci.* 2016;57:2452–2462.

73. Yildirim R, Dikkaya F, Arici C, et al. Corneal viscoelastic properties in patients with angiod streaks. *Curr Eye Res.* 2016;41:299–304.

74. Schoenwolf GC, Larsen WJ. *Larsen’s Human Embryology*, 4th ed. Philadelphia, PA: Churchill Livingstone/Elsevier; 2009:687.

75. Garway-Heath DF, Caprioli J, Fitzke FW, et al. Scaling the hill of vision: the physiological relationship between light sensitivity and ganglion cell numbers. *Invest Ophthalmol Vis Sci.* 2000;41:1774–1782.

76. Hood DC, Raza AS, de Moraes CG, et al. Glaucomatous damage of the macula. *Prog Retin Eye Res.* 2013;32:1–21.

77. Blumberg DM, De Moraes CG, Prager AJ, et al. Association Between Undetected 10-2 Visual Field Damage and Vision-Related Quality of Life in Patients With Glaucoma. *JAMA Ophthalmol.* 2017;135:742–747.

78. Murata H, Hirasawa H, Aoyama Y, et al. Identifying areas of visual field damage of the macula. *Prog Retin Eye Res.* 2013;19:298–312.

79. Sumi I, Shirato S, Matsumoto S, et al. The relationship between visual disability and visual field in patients with glaucoma. *Ophthalmology.* 2003;110:332–339.

80. Zhong Y, Shen X, Yu J, et al. The comparison of the effects of latanoprost, travoprost, and bimatoprost on central corneal thickness. *Acta Ophthalmol (Copenh).* 1985;63:351–354.

81. Nielsen CB, Nielsen PJ. Effect of alpha- and beta-receptor active drugs on corneal thickness. *Acta Ophthalmol (Copenh).* 1985;63:351–354.

82. Inoue K, Okugawa K, Oshika T, et al. Influence of dorzolamide on corneal endothelium. *Jpn J Ophthalmol.* 2003;47:129–133.

83. Kaminiski S, Hommer A, Koyuncu D, et al. Influence of dorzolamide on corneal thickness, endothelial cell count and corneal sensitivity. *Acta Ophthalmol Scand.* 1998;76:78–79.

84. Sawada A, Yamamoto T. Switching efficacy on intraocular pressure from latanoprost to bimatoprost in eyes with open angle glaucoma: implication to the changes of central corneal thickness. *Jpn J Ophthalmol.* 2014;58:423–428.