Correction for the Erroneous Compensation of Anterior Segment Birefringence with the Scanning Laser Polarimeter for Glaucoma Diagnosis

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PURPOSE. To evaluate whether erroneous compensation for anterior segment retardation can be estimated and used to correct peripapillary (PP) retinal nerve fiber layer (RNFL) retardation measurements.

METHODS. Retardation measurements (for the 780-nm wavelength), given as RNFL thickness by the scanning laser polarimeter, were obtained at the macula and PP retina in 45 eyes of 45 normal subjects and 53 eyes of 53 patients with early glaucoma. The correlation of macula and PP retardation was assessed. The normal range for RNFL retardation was defined as 97.5th minus 2.5th percentile (normal subjects). This was calculated for uncorrected PP RNFL retardation and for PP RNFL retardation corrected by retardation measurements taken in the macula (analysis 1) and in the temporal aspect of the PP measurement annulus (analysis 2). Further ranges were defined at different percentile cutoffs, and normal and glaucomatous eyes were classified as abnormal if retardation measurements were below each cutoff. The accuracy of classification by uncorrected and corrected measurements was assessed by receiver operating characteristic curve analysis. Uncorrected and corrected RNFL retardation was correlated with visual field mean deviation (MD).

RESULTS. PP retardation correlated significantly with that of macular retardation in normal ($r^2 = 0.71$, $P < 0.000$) and glaucomatous ($r^2 = 0.41$, $P < 0.000$) eyes. The normal range for uncorrected PP retardation was 25.4° and for corrected retardation, 18.0° ($r^2 = 0.21$; analysis 1) and 14.6° ($r^2 = 0.22$; analysis 2), a reduction of 29% and 43%, respectively. For a specificity of 85%, the sensitivity to identify PP retardation was 25.4° and for corrected retardation, 18.0° ($r^2 = 0.21$; analysis 1: $r^2 = 0.22$; analysis 2: $r^2 = 0.18$) than did uncorrected measurements ($r^2 = 0.05$).

CONCLUSIONS. Erroneously corrected anterior segment birefringence significantly affects PP RNFL retardation measurements. Retardation arising from the cornea–corneal compensator interaction can be partially estimated from the macula and temporal aspect of the PP measurement annulus, allowing correction of PP RNFL retardation. This provides a narrower normal range and greater sensitivity for glaucoma diagnosis. (Invest Ophthalmol Vis Sci. 2002;43:1465–1474)

Glaucoma causes the death of retinal ganglion cells and their axons.1-4 It is assumed that axonal loss results in a reduction in the thickness of the retinal nerve fiber layer (RNFL). Measurements of RNFL thickness should therefore be a reliable means of predicting the loss of visual function in glaucomatous eyes. Scanning laser polarimetry is a relatively recent innovation designed to allow in vivo measurement of the thickness of the RNFL.5,6

The scanning laser polarimeter (SLP) is a confocal scanning laser ophthalmoscope with polarization and polarization-analyzing optics. A laser beam is scanned across the fundus, and reflected light returns through the pupil to a detector. The theoretical basis of scanning laser polarimetry is that the state of polarized light is changed as it passes through a linearly birefringent material. Linear birefringence refers to the property of a material in which light polarized in one direction travels faster through the material than light polarized in the perpendicular direction (it has a “fast” and a “slow” axis). This difference in speed causes a phase shift between the perpendicular light beams (retardation). Principal ocular structures exhibiting birefringence are the cornea,7-9 RNFL,10 and Henle fibers,11 with the cornea contributing the largest component to measurements of retardation in whole eyes.12 The cornea, in fact, behaves as a biaxial crystal with three different indices of refraction (in the x, y, and z directions).7,11 However, the fastest principle axis is normal to the corneal surface so that, when light passes through the cornea perpendicular to the surface, only the difference in refractive index between the x and y directions give rise to retardation.7,11 The cornea, therefore, acts as a fixed retarder, with its slow axis nasally downward,7,11 when light passes perpendicularly through the central cornea. In the parafovea, the symmetrical arrangement of Henle fibers acts as a uniaxial birefringent crystal with the slow axis arranged radially around the fovea.13 The retardation arising when polarized light passes through the birefringent cornea and Henle fibers is explained by Klein Brink and van Blokland.10 Retardation arising from two linear retarders is added or subtracted, depending on the angle the slow axes of the retarders make with each other. Thus, as the beam passes around a circle in the perifoveal area, corneal and retinal retardations are alternately summed and subtracted, with troughs 180° apart and peaks 180° apart.10

To quantify the component arising from the RNFL, it is necessary to neutralize the corneal component. A commercial SLP (GDx Nerve Fiber Analyzer; Laser Diagnostic Technologies, Inc., San Diego, CA), incorporates a proprietary “cornea polarization compensator” designed to cancel the polarization
effects of the cornea.12 When the fast axis of the compensator is aligned to the slow axis of the cornea, and the magnitude of birefringence is equal to the population mode, the effect of corneal birefringence is neutralized. If the fast axes of the two are aligned, the birefringent effects are additive. Thus, if the slow axis of the cornea is rotated (in either direction) and out of alignment with the fast axis of the corneal compensator, measured retardation from the combined cornea and corneal compensator is greater than zero. The corneal compensator has a fixed optic axis and a fixed magnitude of retardation, so that the corneal component of retardation is eliminated only if the position of the slow axis and magnitude of retardation of the cornea in the eye being imaged are identical with the mode of the population. However, if either the axis or magnitude of retardation of the cornea lies outside the normal range, retardation resulting from the interaction of the cornea and the compensator manifests in measurements made from the fundus. Because the retardation pattern arising from the Henle fibers around a fovea-centered annulus in the perifoveal retina is radially symmetrical,10 any modulation of the measured retardation around an annulus in the perifoveal retina must result from uncompensated, or erroneously compensated, retardation elsewhere in the laser pathway. The slow axis of corneal birefringence is typically 15° nasally downward.7,11 Greenfield et al.11 established that there is considerable interindividual variation in the axis of corneal birefringence and demonstrated that the corneal axis is significantly associated with RNFL and macular summary retardation parameters. Furthermore, they suggested that the macular measurements might be helpful in estimating the corneal polarization axis. Knighton et al.13 have shown that there is also a wide variation in the magnitude of corneal retardation. Recently, Zhou et al.14 have demonstrated that the axis and magnitude of anterior segment retardation estimated from the macular retardation pattern agrees well with direct measurement of the same variables, using a corneal polarimeter (although the magnitude of retardance was generally lower when measured from macular polarimetry). The corneal polarimeter enables a view of the fourth Perkinje image through crossed polarizers and a variable waveplate.15

Retardation measured from the macula represents the combined effects of the birefringence of the corneal compensator, the cornea, and the radially arranged Henle fibers. Because the axons of the RNFL are approximately radially arranged in the peripapillary retina, the interaction of the corneal compensator–corneal birefringence with peripapillary RNFL birefringence may be similar to that occurring in the macula. It may therefore be possible to use the macular retardation pattern to correct the peripapillary measurements for erroneously compensated corneal birefringence.

The purpose of this study was to apply this empiric approach to determine whether the ability of peripapillary RNFL measurements to discriminate between normal and glaucomatous eyes can be improved by correcting the peripapillary retardation measurements with retardation measurements made in the macula.

**Methods**

**Subjects**

Forty-five eyes of 45 normal subjects and 53 eyes of 53 subjects with glaucoma were imaged with the SLP. Normal subjects were members of staff, friends or spouses of patients, or volunteers, attending the Glaucoma Division of the Jules Stein Eye Institute, Los Angeles (37 subjects), and the Glaucoma Research Unit of Moorfields Eye Hospital, London (8 subjects). Inclusion criteria were ametropia less than 5 D, visual acuity of 20/30 or better, normal visual fields, intraocular pressure of less than 21 mm Hg, no previous history of ocular disease, and no family history of glaucoma involving a first-degree relative. All subjects performing a normal field test were included, regardless of optic disc appearance. One eye was included in the study, chosen at random if both were eligible.

Subjects with glaucoma were patients attending the Glaucoma Division of the Jules Stein Eye Institute, Los Angeles (51 subjects), and the Glaucoma Research Unit of Moorfields Eye Hospital, London (2 subjects). Restriction criteria were ametropia less than 5 D, visual acuity of 20/30 or better, a reproducible visual field defect, normal open anterior chamber angle, and no other disorders that might cause visual field loss. One eye was included in the study, chosen at random if both were eligible.

All normal subjects and patients with glaucoma had consented to take part in prospective research into the early detection of glaucoma with new imaging technology at their respective institutions. Research protocols had been approved by the relevant institutional ethics committees and conformed to the tenets of the Declaration of Helsinki.

**Visual Field Testing**

All visual field testing was performed with the Humphrey Field Analyzer (model 640 or 750; Allergan Humphrey, San Leandro, CA) and the 24-2 full-threshold or 24-2 Swedish interactive test algorithm (SITA) standard programs. Reliability criteria applied were fixation losses less than 30%, false-positive responses less than 15%, and false-negative responses less than 30%.

A glaucomatous visual field was defined as two or more contiguous points with a P < 0.01 loss or greater, or three or more contiguous points with a P < 0.05 loss or greater, in the superior or inferior arcuate areas, compared with perimeter defined age-matched control subjects, or a 10-dB difference across the nasal horizontal midline at two or more adjacent locations.15 In addition, all patients had abnormal findings in a glaucoma hemifield test (Allergan Humphrey).

A normal visual field was taken to be one in which there were no sensitivity losses matching the criteria for glaucoma.

**Image Acquisition and Analysis**

Technical details of the SLP have been described elsewhere.6,16 Subjects were imaged with the SLP, software (GDx NFA, Ver. 1.0.16; Laser Diagnostic Technologies). All subjects had a series of one to three images centered on the fovea and three images centered on the optic disc. A subjective assessment of image quality of macular images was made on the basis of the brightness and evenness of fundal illumination, motion artifact, and centration on the fovea. Poor-quality images were discarded. Software-generated image quality assessment was used to evaluate images centered on the optic disc, and these were also checked by subjective assessment.

If more than one good-quality macular image was available for each eye, images were aligned manually in the SLP software to generate a mean image. Optic disc-centered images were also aligned manually to generate a mean image. Software-generated RNFL thickness measurements were recorded at the macula, with the vessel-removal algorithm switched off, and in the peripapillary retina, with the vessel-removal algorithm switched on. Data were collected using the software facility Tools/Region Special/Write Region Data to File.

Retardation measurements were made at the macula in 30° and 10° sectors around a 1000-μm-diameter annulus (width, 10 pixels) centered on the fovea (Figs. 1a, 1c). The Henle fibers form a radially arranged, almost symmetrical, birefringent layer at this location at the edge of the fovea, but the RNFL is only just detectable.17,18 Modulation in macular retardation measurements are likely, therefore, to reflect the presence of birefringent structures elsewhere in the laser pathway.

Retardation measurements were made in the peripapillary retina in 30° and 10° sectors around a 2880-μm-diameter annulus (width, 10 pixels) centered on the optic disc (Figs. 1b, 1d). RNFL thickness measurements reach a maximum in annuli of 1.5 to 2.0 disc diameters,19 and measurements are more reproducible at the 1.5-disk-diameter annulus when compared with 1.0 and 2.0.20 An annulus diameter

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the perifoveal and peripapillary annuli and differences in the retardation modulation (maximum value minus minimum value for 10° sectors) around the perifoveal and peripapillary annuli and the modulation of the measure “peripapillary minus perifoveal retardation.”

The association of the mean magnitude of retardation around the perifoveal annulus and the mean magnitude of retardation around the peripapillary annulus was explored by linear regression analysis. Similarly, the association of the magnitude of retardation modulation around the perifoveal annulus and the magnitude of retardation modulation around the peripapillary annulus was explored by linear regression analysis. The analyses were performed separately in the normal and glaucomatous eyes.

A series of normal ranges for retardation measurement around the peripapillary annulus was defined from the normal eyes at cutoff levels ranging from the 1st to the 50th percentiles in each 30° segment. A normal or glaucomatous eye was categorized as abnormal if the retardation measurement fell below the cutoff level in any 30° segment.

The macular retardation measurement in each 30° segment was then subtracted from the retardation in the equivalent peripapillary 30° segment in all eyes, to give an adjusted retardation value. A second series of normal ranges was defined from cutoffs at the 1st to the 50th percentiles. Similarly, a normal or glaucomatous eye was categorized as abnormal if the retardation measurement declined below one of the percentile cutoffs in any 30° segment.

The categorization of eyes at the cutoffs from the 1st to 50th percentiles was used to generate receiver operating characteristic (ROC) curves.

Analysis 2. The association of the mean magnitude of retardation around the perifoveal annulus and the magnitude of retardation in the temporal 40° of the peripapillary annulus (Fig. 1) was explored by linear regression analysis.

Because the mean retardation in the temporal 40° of the peripapillary annulus was found to be significantly related to the mean magnitude of retardation around the perifoveal annulus, the possibility of using this measure to correct for erroneously compensated corneal birefringence was explored. The mean retardation in the temporal 40° of the peripapillary annulus was therefore subtracted from the retardation in each 30° segment of the peripapillary annulus in all eyes, to give an adjusted retardation value. A third series of normal ranges was defined from cutoffs at the 1st to the 50th percentiles. Again, a normal or glaucomatous eye was categorized as abnormal if the retardation measurement fell below one of the percentile cutoffs in any 30° segment.

RESULTS

Demographic data on the subjects are summarized in Table 1. The diagnosis of the glaucomatous eyes was as follows: 36 primary open-angle glaucoma, 13 normal-tension glaucoma, 3 pigment-dispersion glaucoma, and 1 glaucoma secondary to uveitis (inactive).

Analysis 1

The mean ± SD retardation around the perifoveal annulus was 15.5 ± 3.9° in the normal eyes and 14.6 ± 3.0° in the glaucomatous eyes. The difference was not statistically significant.

| Table 1. Demographic Data |
|---------------------------|-------------------|-------------------|
|                           | Normal Eyes       | Glaucomatous Eyes |
| Number                    | 45                | 53                |
| Age (y)                   | 51.4 ± 11.9       | 64.2 ± 14.1       |
| Sex (% male)              | 48.9              | 41.5              |
| Refractive error (D)      | −0.5 ± 1.5        | −0.7 ± 2.7        |
| Eye (% right eyes)        | 55.6              | 41.5              |
| Visual field MD (dB)      | 0.31 ± 1.0        | −3.92 ± 2.1       |

![Image](https://via.placeholder.com/150)
The distribution of mean perifoveal retardation values in normal eyes is given in Figure 2. The mean retardation around the peripapillary annulus was 22.2 ± 6.2° in the normal eyes and 19.0 ± 4.2° in the glaucomatous eyes. The difference was statistically significant (P = 0.01, Mann-Whitney).

A plot of mean retardation around the perifoveal annulus against mean retardation around the peripapillary annulus in normal eyes is given in Figure 3. The R² for the correlation was 0.71 (P < 0.0001). In glaucomatous eyes, the R² for the correlation was 0.41 (P < 0.0001). The retardation modulation around the peripapillary annulus was 10.1 ± 3.7° in the normal eyes and 8.9 ± 3.2° in the glaucomatous eyes. The difference approached statistical significance (P = 0.06). The mean retardation modulation around the peripapillary annulus was 21.5° in the normal eyes and 18.3° in the glaucomatous eyes. The difference was statistically significant (P = 0.0001).

A plot of retardation modulation around the perifoveal annulus against retardation modulation around the peripapillary annulus in normal eyes is given in Figure 4. The R² for the correlation was 0.29 (P < 0.0001). In glaucomatous eyes, the R² for the correlation was 0.02 (P = 0.28).

The normal range (between the 2.5th and 97.5th percentiles) for unadjusted peripapillary retardation is illustrated in Figure 5 and the normal range for adjusted peripapillary retardation in Figure 6. The mean range between the 2.5th and 97.5th percentiles was 25.4° for unadjusted retardation and 18.0° for adjusted retardation. The difference was statistically different (P = 0.0006). The unadjusted and adjusted peripapillary retardation modulation in normal eyes was 21.5° and 18.3°, respectively. The difference was statistically significant (P = 0.0005).

**Analysis 2**

A plot of mean retardation around the perifoveal annulus against the mean retardation in the temporal 40° of the peripapillary annulus in normal eyes is given in Figure 7. The R² for
the correlation was 0.61 (P < 0.0001). In glaucomatous eyes, the $R^2$ for the correlation was 0.24 (P = 0.0002). The mean range between the 2.5th and 97.5th percentiles was 14.6° for adjusted retardation. The difference between the unadjusted and adjusted retardation was statistically significant (P = 0.0001). The normal range for adjusted peripapillary retardation is illustrated in Figure 8.

Classification accuracy for unadjusted and adjusted (analyses 1 and 2) retardation values is illustrated by the ROC curves in Figure 9 and in Table 2. The sensitivity values for adjusted retardation analyses 1 and 2 were not significantly different. The sensitivity for analysis 1 was significantly better than unadjusted retardation at 85% specificity. The sensitivities for analysis 2 were significantly better than unadjusted retardation at 80% and 85% specificity.

The $R^2$ values for the correlation of retardation measurements with visual field MD were as follows: unadjusted 0.05, adjusted (analysis 1) 0.21, and adjusted (analysis 2) 0.18.

**DISCUSSION**

There is good evidence that retardation measurements made with the SLP correlate with RNFL thickness. The peripapillary retardation measurements have been validated histologically in primate eyes with the cornea and lens removed. There was a correlation between retardation measurements and RNFL thickness measurements, with 1° of retardation equivalent to 7.4 μm of RNFL with a laser source of 514 nm wavelength. For a given birefringent material, the magnitude of retardation of light passing through it is wavelength-dependent. The current NFA instrument uses a laser source of 780-nm wavelength, and the manufacturers have adopted a conversion factor of 3 μm RNFL thickness per degree of retardation (Zhou Q, Laser Diagnostic Technologies, written communication). Good correspondence between retardation measurements and RNFL thickness has also been reported in intact primate eyes, although the strength of the correlation and gradient of the
relationship varies around the circumference of the optic nerve head. In human deceased-donor eyes with the cornea and lens removed, the pattern of peripapillary retardation is consistent with known properties of the RNFL, with the slow axes of the birefringent structures in the peripapillary retina arranged radially around the optic disc. In addition, an age-related decline in retardation has been reported that may result from the age-related loss of axons that has been demonstrated histologically.

It is well established that loss of the RNFL is an early sign in glaucoma and frequently precedes visual field loss. A histologic study estimated that a 5-dB sensitivity loss in the visual field was associated with a 20% ganglion cell loss for the whole field and a 50% ganglion cell loss in the central 12 degrees. A recent analysis of the physiological relationship between ganglion cell numbers and visual field sensitivity has suggested that a 3-dB sensitivity loss may equate to as much as a 50% loss of ganglion cells across the central 24 degrees. In this context, it is disappointing that estimates of RNFL thickness with the SLP did not have a greater ability to discriminate between normal and glaucomatous eyes. Many studies involving scanning laser polarimetry have found considerable overlap between RNFL thickness measurements between normal and glaucomatous eyes.

Other, mathematically contrived parameters separate normal and glaucomatous eyes with greater sensitivity and specificity. These parameters include the ratios between the RNFL thickness at the poles of the peripapillary measurement ellipse and that in the nasal or temporal part, the difference between the RNFL thickness (or cross-sectional area) at the poles of the peripapillary measurement ellipse and that in the nasal or temporal part, the variability of height measurements around the measurement ellipse, the symmetry between upper and lower halves of the disc and between eyes, a discriminant function using RNFL thickness and modulation.
measurements,\textsuperscript{35} and the output of a trained neural network, using 128 input parameters, called “The Number.”\textsuperscript{35,38,39,42} In addition, estimations of RNFL thickness with the SLP have a significant, though rather poor, correlation with equivalent structural parameters estimated with other imaging devices.\textsuperscript{29,43} Correlation of retardation measurements with clinical grading of RNFL photographs is poor, with RNFL grading demonstrating a better correlation with visual field data.\textsuperscript{41}

The original histologic validation studies were performed on eyes with the cornea and lens removed,\textsuperscript{6,9} and the only study in intact eyes reported a poorer correlation between retardation and RNFL thickness and variation in the relationship between the two around the circumference of the optic disc.\textsuperscript{25} The effects of the axis of corneal birefringence and the interaction of the cornea and corneal compensator on measured posterior segment retardation have recently been demonstrated by Greenfield et al.\textsuperscript{11} In the present study, the interindividual variability in the magnitude of retardation around the perifoveal measurement annulus and the intraindividual modulation in magnitude of retardation around the perifoveal measurement annulus (double-hump pattern) in normal eyes suggests an erroneously corrected component of retardation...
induced by the interaction of the cornea and corneal compensator. The very high correlation between retardation measured around the perifoveal annulus and retardation measured around the peripapillary annulus (Fig. 3) suggests that a substantial part of the retardation measured at the peripapillary annulus arises from the interaction of the cornea and corneal compensator. In addition, the significant correlation between the modulation of retardation in the perifoveal and peripapillary retina supports the empiric approach taken in this study to subtract the former from the latter to make a correction for erroneously compensated anterior segment birefringence. Because it has been demonstrated that the axis and magnitude of anterior segment retardation estimated from the macular retardation pattern agrees well with direct measurement, the approach of subtracting the macular pattern from the peripapillary pattern may provide a simple solution to the problem compared with measuring corneal birefringence. The correlation between perifoveal and peripapillary retardation measurements was slightly worse in the glaucomatous eyes. A possible explanation for this is that retardation measurements in glaucomatous eyes have been shown to be less reproducible than those in normal eyes, and this greater variability would lessen the strength of the relationship. An alternative explanation is that, in normal eyes, the pattern of peripapillary retardation is, by coincidence, similar to the macular retardation pattern. Thus, in glaucomatous eyes, with the loss of the regular double-hump pattern of the RNFL in the peripapillary region, there is a lower correlation with the macular retardation pattern.

The results demonstrate that the (empiric) approaches taken in this study provide a narrower normal range of retardation measurements (Figs. 7, 9) and improved discrimination between normal and glaucomatous eyes. The results also demonstrate that by adjusting peripapillary retardation measurements, with either perifoveal or temporal peripapillary measurements, the correlation with visual function is greatly enhanced. The normal ranges derived in this study do not demonstrate that by adjusting peripapillary retardation measurements in the perifoveal retardation to make a correction may not be wholly justified. Birefringence has an axis as well as a magnitude, and differences in the orientation of the axons around the optic disc and Henle fibers around the fovea may result in an inappropriate correction. However, the method shows promise, and a study is in progress to evaluate the agreement between the empiric approach (subtracting perifoveal from peripapillary retardation) and the approach of making peripapillary retardation measurements with a variable compensator to neutralize corneal birefringence for each eye imaged.

Analysis 2 identified a very high correlation between retardation measured in the perifovea and retardation measured at the temporal 40° of the peripapillary measurement annulus (Fig. 7). Correction for corneal retardation on the basis of the amount of retardation measured in the temporal 40° of the peripapillary measurement annulus performs better than the first analysis, although it accounts only for the variability in the average magnitude of erroneously compensated birefringence and not the modulation around the measurement annulus. A reason for this may be that the second method makes an estimate of the anterior retardation and of the peripapillary retardation from within the same image, whereas the first analysis requires a correction derived from one image to be applied to the second. Occasional patients with horizontal peripapillary double-hump patterns have been described. Analysis 2 would give rise to a nonphysiological pattern of retardation in these rare cases.

The results of analysis 2 explain why the discriminative ability of SLP measurements in previous investigations were improved by taking the difference between the polar and the nasal-temporal cross-sectional areas, the difference between the polar and the nasal-temporal RNFL thickness, or the ratio of the polar and nasal RNFL thicknesses. The magnitude of the minimum retardation (in the temporal and nasal parts of the peripapillary retina) is related to the magnitude of retardation arising from anterior birefringent structures.

The effect of the cornea–corneal compensator interaction on the measurement of RNFL-induced retardation has a considerable impact on the ability of the polarimeter to distinguish between normal and glaucomatous eyes. It probably has little effect on the instrument's ability to detect change over time, in that measurement reproducibility has been shown to be high, and, although there are no long-term data, corneal birefringence has been shown to remain stable for a year.

In the current study, a considerable proportion of measured peripapillary retardation was induced by birefringent structures anterior to the retina, and accounting for this anterior component resulted in a narrower range of measurements in normal eyes and a greater ability of retardation measurements to discriminate between normal and glaucomatous eyes.

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References


43. Waldock A, Potts MJ, Sparrow JM, Karwatowski WS. Clinical evaluation of scanning laser polarimeter. T intraoperator reproducibil...