Eye scan therapy for astigmatism

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ABSTRACT—Two earlier papers introduced a new model of functional astigmatism. The investigation that led to the model indicated a relationship exists between meridional eye scanning habits and the formation of functional astigmatism. The present paper reviews the model and describes a clinical study involving the prescribing of specific eye scan therapy on a meridional basis. The results demonstrate that therapy involving eye scanning procedures has a positive effect on the control and reduction of astigmatism.

KEY WORDS—astigmatism, eye scan, head scan, head posture, eye scan therapy

Introduction

Conflicting views exist concerning astigmatism and its possible causes. Many have conceived it to be primarily a structural anomaly that follows genetic and age patterns. This view has been influenced by longitudinal, demographic and cross-sectional studies.

Early studies have indicated horizontal or with-the-rule astigmatism is more prevalent during the pre-school years.[1] More recent studies have revealed a greater incidence of against-the-rule astigmatism in pre-school children which tends to either become gradually eliminated or changes to with-the-rule astigmatism by school age.[2,3,4]

Vertical or against-the-rule astigmatism tends to reappear as one gets older, especially after the age of forty.[5,6,7,8,9] This type of astigmatism has also been shown to relate to the development of myopia.[10]

It has been speculated that these changes in astigmatism are precipitated by changes in lid pressure, with increased lid pressure accounting for greater with-the-rule and reduced against-the-rule astigmatism. Loose lids or lid retraction has been thought to possibly account for increased against-the-rule or decreased with-the-rule astigmatism.[2,11,12]

Others hypothesize a functional origin for most forms of astigmatism viewing its formation as a reaction to accommodative or convergence stress,[13,14,15,16] postural distortion,[17] or work-related viewing demands.[18]

Clinically, exceptions for almost all the rules are found. Myopic or presbyopic individuals are seen who do not demonstrate increased against-the-rule astigmatism or reduced with-the-rule astigmatism.

Some even exhibit a reduction in against-the-rule astigmatism or an increase in with-the-rule astigmatism. In terms of postural distor-

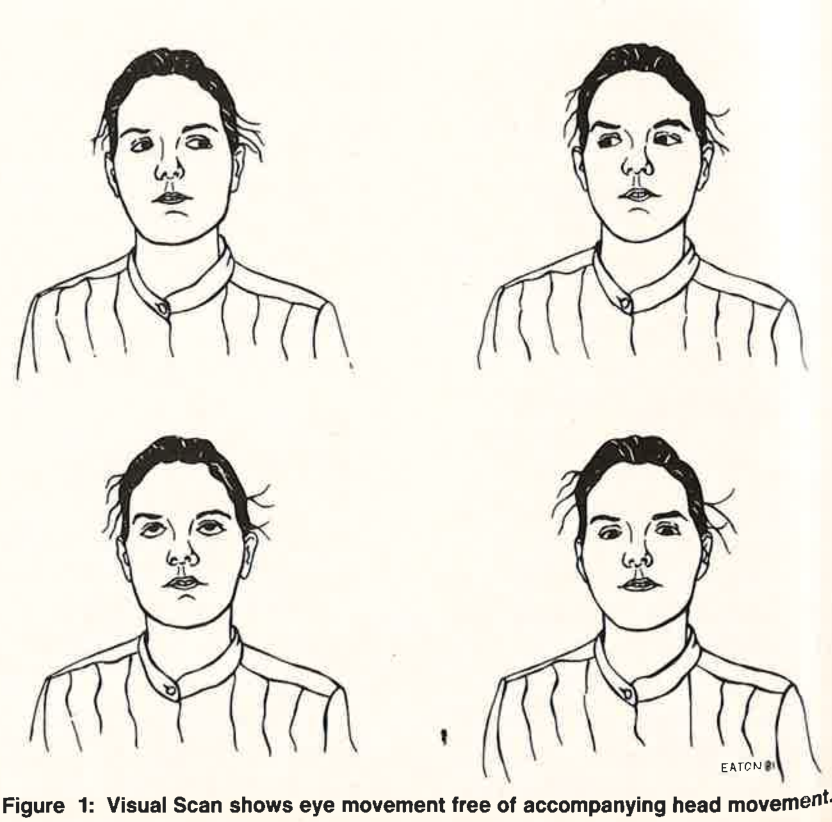


Figure 1: Visual Scan shows eye movement free of accompanying head movement.

tions, it is possible for individuals with pronounced and persistent head tilts to demonstrate little or no astigmatism at all.

Inconsistencies such as these stimulated the start of a long-term investigation leading to the formation of a new model of functional astigmatism.[19,20] This new model suggests there is a direct relationship between changes in astigmatism and an observable triad consisting of eye scan, head scan and head posture.

Eye scan refers to eye movement that is free of head movement. (See Figure 1). Head scan refers to eye movement that is yoked to head movement. (See Figure 2). Head posture refers to a combination of head angle, rotation, and tilt in relation to gravity. (See Figure 3).

Key elements of the model

The key elements of the model are as follows:

1. With-the-rule astigmatism appears to relate to a greater preference for visual (eye) scanning, free of head movement, in the horizontal meridian than in the vertical meridian.
2. Against-the-rule astigmatism appears to relate to a greater preference for visual scanning in the vertical meridian than in the horizontal meridian.

The term preference does not indicate a conscious decision on the part of the individual. It represents a spontaneous type of movement that can be precipitated by specific causative factors such as the mechanics of a task situation.

Interestingly, it can often be correlated with a subjective feeling of an inner ease in visually scanning in one principal meridian as opposed to the other rather than by an objective measurement of the actual quality of eye movement in different meridians of gaze.

3. The overall magnitude of astigmatism appears to be related to the degree of difference between the type of scanning in one principal meridian as compared to the other. Less astigmatism is associated with less of an operational difference between meridians. Little or no astigmatism appears to be related to eye scan/head scan preferences that are approximately equal in all meridians.
4. Astigmatic anisometropia, in which there is a difference in the amount of astigmatism in each eye, appears to relate either to a persistent rotation of the head to one side or a decentration of the task in relation to the head.

In either case, there is a resulting mismatch between the structural midline of the head and the operational visual midline (the line that bisects the angle formed by the lines of sight as they fixate a target).

The eye that then persistently fixates across the facial midline (e.g. the nose) tends to develop more astigmatism (See Figure 4).

The degree of astigmatism in the other eye, the one with the lesser astigmatism, appears to be related to how close that eye is to being aligned perpendicularly to the task. The closer that eye is to being perpendicular to what is being fixated, the less astigmatism that eye will have (See Figure 5).

5. Parallel oblique astigmatism appears to be due to a consistent obliquity in visual scanning resulting either from a persistent head tilt, an angled task placement or a combination of both. (See Figure 6).
6. Symmetrical oblique astigmatism appears to result from a consistent mismatch between the plane of regard and the primary position plane (a plane at the level of the two eyes perpendicular to the frontal or facial plane). This is due to a persist-

Figure 2: Head Scan with eye movement yoked to head movement.

Figure 3: Head posture showing major elements of head movement around the x, y and z axes.

ent up or down angling of the head (See Figure 7).

In binocular individuals, a persistent scanning pattern above the primary position plane results in intorted axes. Binocular individuals, whose scanning pattern is consistently below the primary position plane, tend to reveal extorted axes.

Monocular individuals, on the other hand, tend to demonstrate the reverse obliquity. Persistent scanning above the primary position plane results in extorted axes while persistent scanning below the primary position plane results in intorted axes.

7. Asymmetrical astigmatism, in which the axes are neither parallel nor symmetrical, appears to be due to an oblique eye scan/head tilt relationship superimposed on an angled head positioning.

The extreme of asymmetry, perpendicularly-opposing astigmatism, where the astigmatism is close to vertical in one eye and close to horizontal in the other, appears to be evident in those who utilize a different type of eye scan for things done on the right side as opposed to the left. In most instances, it is seen to be accompanied by inefficient binocularity.

Initial studies

An initial paper introduced the model and reported two clinical studies designed to test the hypothesis.[19] The first study involved a total of 45 consecutively seen patients between ages 20 and 40 who had demonstrated a recent change in astigmatism of at least 0.50 D in one eye (except for a few where a significant shift in axis was concerned).

In almost every case, some change was reported to have taken place within the previous 4 to 12 months in work and/or study habits (on the job, in school or at home) that appeared to be consistently related to specific alterations in eye scan/head scan/head posture usage.

The second clinical study indicated it was also possible to make reasonable predictions of an individual's habitual and preferred eye scan/head scan/head posture relationship based on the astigmatic component of the refractive findings.

Implications of the model

The essential element in the formation of functional astigmatism appears to be the individual's eye movement/head movement style and how it differs in the vertical as compared to the horizontal meridian.

Persistently skewed head posture superimposed upon this meridional eye scan/head scan relationship tends to result in the various types of astigmatic anisometropias and oblique astigmias that are seen.

Early in the investigation, it was assumed the preferred eye scan meridian more directly affected astigmatic status. It was thought that the horizontal toroidal elongation of the cornea in with-the-rule astigmatism could be due to the molding effect created by "freer" eye movements in the horizontal meridian, especially in relation to the possible action of the eyelids.

In comparing changes in astigmatism with concurrent changes in task demand, it became apparent the meridian of restraint (the meridian in which eye movement more often tended to be yoked to head movement) appeared to trigger the change in astigmatism by becoming more myopic or less hyperopic.

Another important factor concerned the relation of primary causes to mediating causes. It became apparent many factors could influence the eye scan-head scan relationship.

These predisposing factors include accommodative stress, convergence difficulty, poor bilateral organization, presbyopia, improper lighting, distorted posture, mechanics of the task demand, psychological desire for clearer vision over a wider range of space, and mechanics inherent in the use of bifocals, especially the narrower ones.

The actual change in astigmatic status, however, appears to occur primarily in individuals who, in the course of adapting to environmental or physiological demands, change the habitual meridional interplay between eye movement and head movement.

If the visual scan habits do not change, the chances are the individual's astigmatic status will remain the same regardless of the primary causative factors. This would account for myopes and presbyopes who do not develop less with-the-rule or more against-the-rule astigmatism as the statistical trends would indicate they should.

Because eye movements appear to play an important role in this model, there is the further implication that the action of the extraocular muscles is a major factor in forming astigmatism and its various types. A rationale for such possible extraocular muscle involvement was presented in a second paper on the subject.[20]

A more clinically relevant implication is if functional astigmatism is molded by use, then it should be capable of being reversed or minimized by specific therapy directed at changing eye scanning habits. This would be accomplished mainly by encouraging more eye movement free of head movement in the meridian where both have become relatively more yoked. This inference led to the prescribing of specific eye scan therapy for astigmatic patients.

In addition, because in clinical practice astigmatism is often seen to change within short periods of time (e.g. 6 to 12 months), at almost any age and regardless of correction worn, it was hypothesized if results were to be achieved through appropriate therapy, they should similarly be achieved in short periods of time, at almost any age and regardless of correction.

Eye scan therapy

The major aim of eye scan therapy for astigmatism is to attempt to equalize eye scanning in the principal meridians by increasing eye movement (free of head movement) in the head scanning meridian. A secondary aim, in the case of astigmatic anisometropia, is to correct head alignment so the structural midline of the head is kept more consistently coincident with an imaginary line bisecting the lines of sight of the two eyes.

Many different techniques were originally used to accomplish these goals. These included eye scanning with the head tilted, angled or rotated to compensate for various oblique axes of astigmatism.

It also included using bi-temporal occluders (for with-the-rule astigmats), superior-inferior occluders (for against-the-rule astigmats), asymmetrically placed bi-temporal occluders (for head rotators) and yoked lateral prisms.

For various reasons, most of these approaches were not found to be universally practical or effective. It was out of experience with these various procedures a simplified protocol evolved.

1. To reduce the magnitude of astigmatism, full pursuit and/or wide saccadic fixations are prescribed. Instructions are given for this to be done effortlessly, without head movement, for a minimum of two minutes per session. Saccadic fixations are done at the rate of approximately one scan per second.

It is recommended this procedure be done ten to fifteen times per day and to be associated with key activities such as brushing teeth, eating, or watching T.V. to serve as reminders.

Predominantly against-the-rule astigmats are instructed to scan horizontally. Predominantly with-the-rule astigmats are instructed to scan vertically.

If the obliquity of the astigmatism cannot be determined to be predominantly vertical or horizontal, (e.g. O.D. axis 135, 0.S. axis 45) or in cases of perpendicularly opposing astigmatism, then general motility in the major meridians is prescribed. This encompasses doing an equal amount of horizontal, vertical and oblique scanning during each two minute training period.

2. To reduce astigmatic anisometropia, the patient is instructed to become aware of his nose, thinking of it perhaps as a

Figure 4: A mismatch between the structural midline of the head and the operational visual midline.

Figure 5: The eye closest to being directly in line with and perpendicular to the task develops less astigmatism.

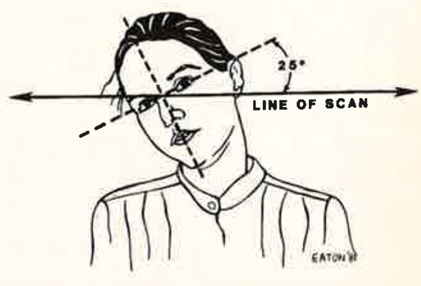
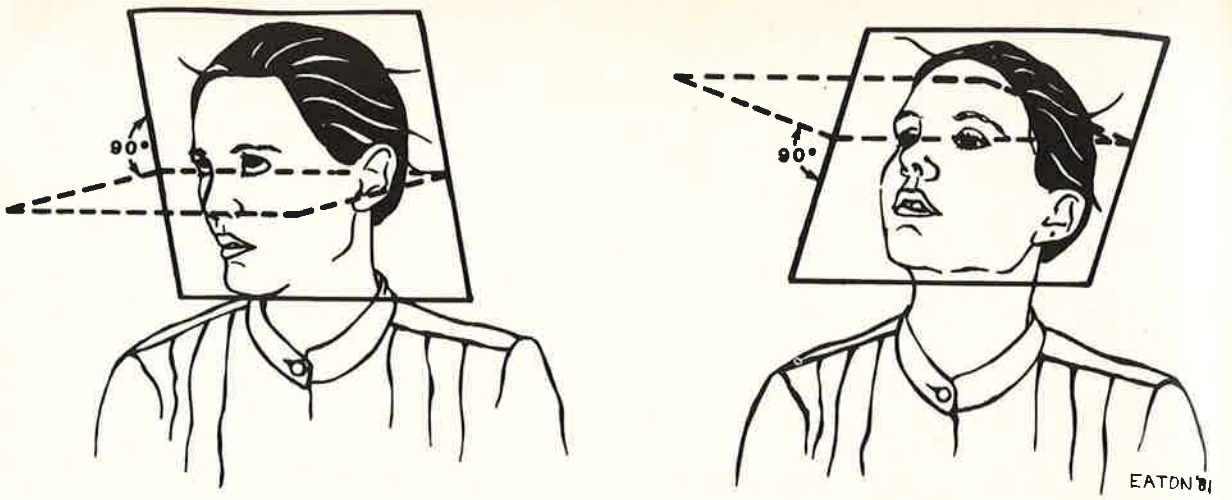


Figure 6: A mismatch between the line of scan and the "horizontal" plane of the eyes.



SCANNING ABOVE PRIMARY POSITION PLANE **SCANNING BELOW PRIMARY POSITION**

Figure 7: Scanning above or below the primary position plane as a result of persistent head angling.

pointer, an arrow or an expanding Pinocchio-type nose and remembering to point it towards whatever requires prolonged visual attention. Because of habit, there might be a tendency to undercorrect and not actually align the nose as it should be.

Patients are instructed to exaggerate and overshoot the target area, if necessary, rather than take the chance of an undercorrection.

Testing the hypothesis

After numerous instances of clinical improvement were demonstrated using this protocol, it was decided to institute a third clinical study to test, in a more formal manner, the effect of specific eye scanning procedures on the magnitude of astigmatism and the effect of more appropriate head alignment on astigmatic anisometropia.

This third investigation was organized around clinical patients seen routinely. Those with astigmatism, or their parents in the case of children, were told what the condition represented according to the model and how it might be possible to reverse the condition to some degree using simple home training procedures. Those who expressed interest and appeared motivated were given specific instructions relative to their particular type of astigmatism and were requested to return for a re-evaluation in approximately six months.

As these patients returned, the first 45 who claimed to have done the procedures became part of the experimental group. The minimum requirement for inclusion was that the procedures were done conscientiously as prescribed for at least three consecutive months, even if they were discontinued afterwards.

An additional 45 patients who admitted to not doing the procedures, or who did them intermittently for only the first few weeks, became the control group.

To ensure operator consistency, all pre- and post-test measurements were done by the same examiner. Both subjective and retinoscopic refractive measurements were taken.

The final reference was to the astigmatism found under subjective testing with the exception of a small number of very young children in whom subjective testing was unreliable. In these instances, the astigmatic status was established by retinoscopic findings which were repeated at least twice to verify their consistency.

Subjective measurements were taken to best visual acuity where more minus or less plus would not improve the ability to discern additional letters on the Snellen chart. Subjective astigmatic tests were always repeated to ensure accuracy of measurements.

An American Optical company Micromatic Ophthalmometer (Model 11000) was used for central keratometry. The calibration of the instrument was checked routinely and each measurement was taken at least three times with the median used as the final recorded finding. Keratometry was done on all but a small group of children, mainly pre-schoolers.

Even though the study was not double-blind, efforts were taken to minimize operator error. This included waiting until post-examination was completed before asking patients (or parents) whether the programmed procedures were done.

Results

Before reporting results of the interaction, it should be noted that because the subjects did, in effect, self-select their category placement (e.g. control group vs. experimental group), the investigator was especially sensitive to pre-test group differences and, in fact, to any differences that might have biased the results. The groups were compared for sex, age, and pre-test differences in astigmatism.

There were 45 subjects in both the control and experimental groups. The control group had 23 females and 22 males. The experimental group had 21 females and 24 males.

The groups were almost identical with respect to sex. As for age, the mean of the control group was 17.64 years, with the standard deviation being 7.44. The mean age of the experimental group was 17.38 years, with a standard deviation of 11.67.

The groups did not differ on this variable by the Wilcoxon test, but variances for the two distributions were different, with $F(44, 44) = 2.46$, $p < .0035$. Because of this, and, again, due to the problem of subject self-assignment, the experimenter was especially careful to control for age as a possible causal variable.

The total sample for each condition was compared to a sample comprised only of individuals age 10 or older (there were four children in the control group 9 years old or younger and 15 in the experimental condition). In no case, either for pre-test scores or for change scores, did the exclusion of children from the sample make any difference, by the Wilcoxon test.

Another variable examined was the amount of time subjects in both groups took in reporting for their post-test examinations. Even though all subjects were asked to return in approximately six months, there was a strong difference between the groups, with control subjects reporting back an average of 13.5 months later (SD = 3.89), and experimental subjects an average of 9.0 months later (SD = 3.87).

Three measures of astigmatism were considered in this study: (1) the magnitude of total astigmatism as determined by refractive procedures, primarily the subjective finding; (2) the magnitude of corneal astigmatism as determined by central keratometry; and (3) the degree of astigmatic anisometropia (the difference in the magnitude of astigmatism between the two eyes) as determined by refractive measurements.

The control and experimental groups were first compared in respect to data from the initial (pre-test) examination. The means for each group are presented in Table 1.[a] The number of subjects listed in the experimental group for the keratometry measurements is 36 rather than 45. This difference is accounted for by the group of chil-

TABLE 1

Means and group comparisons for pre-test astigmatism.

	Mean (N)		Wilcoxon
	Group C	Group E	
Refraction, O.D.	1.300 (45)	1.194 (45)	Z = .589, NS
Refraction, O.S.	1.290 (45)	1.000 (45)	Z = 1.61, p < .11
Keratometry, O.D.	1.401 (45)	1.149 (36)	Z = 1.94, p < .05
Keratometry, O.S.	1.345 (45)	0.960 (36)	Z = 1.97, p < .05
Anisometropia	0.417 (45)	0.472 (45)	Z = 1.54, NS

TABLE 2

Mean change scores for astigmatism and comparisons between the groups.

	Mean Change		Wilcoxon
	Group C	Group E	
Refraction, O.D.	+0.056	−0.461	Z = 7.15, p < .0001
Refraction, O.S.	+0.106	−0.417	Z = 6.69, p < .0001
Keratometry, O.D.	−0.072	−0.199	Z = 1.42, NS
Keratometry, O.S.	−0.005	−0.170	Z = 2.44, p < .015
Anisometropia	+0.039	−0.156	Z = 2.97, p < .005

dren on whom keratometry was not done.

In Table 1, the only pre-test difference between the control and experimental groups is for the magnitude of corneal astigmatism, as measured by keratometry for both left and right eyes. In both cases, the subjects in the control group start out with significantly more measurable corneal astigmatism.

Whether this is due to the self-assignment problem or to the exclusion of the group of younger children is impossible to determine. There is a marginal difference noted between the groups for the left eye as measured on refractive tests, and although the difference is not significant for the right eye as measured by refraction, the trend is in the same direction. Control subjects revealed slightly more total astigmatism than experimental subjects.

Generally, in both the refractive and keratometric tests, the control group began with an initially higher amount of astigmatism, and this fact must be considered when interpreting the data.

The degree of change in astigmatism found in comparing pre- and post-test results is presented in Table 2. Again, data are based on changes in magnitude of astigmatism as determined by refractive and keratometric measurements, and for the degree of anisometropia as determined by refractive measurements.

The means are reported with (+) and (−) signs reflecting direction of astigmatic change. A positive number represents an increase in magnitude of astigmatism or in degree of astigmatic anisometropia over the period of time between pre- and post-examinations. A negative number represents a decrease in magnitude of astigmatism or in degree of astigmatic anisometropia.

As can be seen, magnitude of astigmatism and degree of astigmatic anisometropia decreased in every category for the experimental group. In the control group, there was an increase in the magnitude of astigmatism, on average, for both eyes as measured by refraction, and an increase in the degree of astigmatic anisometropia.

In every case but one (the right eye as measured by keratometry), there was a significant difference in change scores between control and experimental groups. Even for corneal astigmatism of the right eye (as measured by keratometry), the difference was in the predicted direction, although not significantly so ($Z = 1.42$, $p < .16$).

One final analysis was performed. Because subjects in the control condition did have initially higher astigmatism (see Table 1), there is a possibility the greater decrease in astigmatism, as reflected by the change scores (see Table 2) might be due to that initially higher level. Individuals with more astigmatism might be more resistant to change over time.

Correlations were computed between absolute level of astigmatism, at pre-test, and the change scores. Please remember a negative change score means astigmatism was reduced by that amount. The more negative the number, the greater the reduction in astigmatism at the post-exam.

The correlation for refraction, O.D., was almost zero. Correlation for refraction, O.S., and keratometry O.D. and O.S., were negative, in the range −.16 to −.29. The correlation between anisometropia at initial and post exams was quite strong, with $r = -.44$

These correlations indicate subjects that began with a higher degree of astigmatism actually had a slight edge, in terms of reduction, over those with lower initial astigmatism. The lower initial level of astigmatism in the experimental condition did not account for the greater change scores in that condition, and, if anything, slightly biased against that possibility.

Discussion

A number of factors should be considered for their influence on interpretation of these data.

First, there are generally accepted error ranges in regard to refractive and keratometric measurements of approximately ±0.25 to ±0.50 diopters. Even though mean dioptric shifts in this study did not exceed these error limits, the fact that astigmatism in the experimental group shifted in the predicted direction as compared to the control group could be an indication that even small dioptric shifts in astigmatism in patients who have previously demonstrated consistent and stable measurements over many years can be meaningful. The shifts may be a sensitive indicator of recent meridional changes in eye scanning.

The second consideration relates to subject self-assignment in regard to their group placement. Because the subjects assigned themselves to conditions, it is possible some other variable might possibly account for differences in change scores between control and experimental groups.

For example, subjects in the group that chose to undertake the therapy (the experimental group) might have been more highly motivated than those individuals who did not do the recommended procedures. This is supported by the difference in the mean pre-to-post examination interval between groups.

Control subjects took, on the average, about 4½ months longer to report back. And, in addition, the control group did differ from the experimental group in the amount of astigmatism its subjects began with at the time of the initial examination.

Because of these factors, we are not justified in drawing unequivocal causal inferences about the effect of the manipulation.

Realizing the limitations of this study, the author extends an invitation to other researchers to conduct similar investigations using the suggested basic methodology and to compare results with this study to determine the true bias factor, if any. Suggestions to minimize experimental bias would include using different examiners for pre-training and post-training refractions and the possible use of a questionnaire administered by a third party to determine placement of subjects in either the experimental or control groups.

Another approach would be to use an experimental group that does the training procedures and a control group that simply returns at the appointed time for re-examination without having been prescribed any scanning therapy. Of importance, is that the subjects be assigned to their respective groups on a strictly random basis.

This approach would also necessitate using a second, "blind" examiner. For example, one examiner could do the initial examination on half the patients and the post examination on the other half, and vice versa.

Another suggestion would be to use both central and peripheral keratometry on each subject. There are implications that changes in peripheral keratometric measurements as a result of therapy may be more consistently related to refractive astigmatic changes than are changes in central keratometric measurements.

On the other hand, regardless of the limitations in experimental design of the present study, there appears to be a reasonable indication that the data indicating a decrease in astigmatism in the experimental subjects can be explained on the basis of the scanning therapy. Again, though it is not appropriate to draw strong causal inferences, the data does appear to offer support for the model and to the possible clinical use of specific scanning therapy for astigmatism. It also suggests this therapy procedure is certainly worth further investigation. **AOA**

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FOOTNOTE

a. Rank scores are available for inspection.

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