Visual Stabilization of Posture in Retinitis Pigmentosa and in Artificially Restricted Visual Fields

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Purpose. To investigate the relationship between retinitis pigmentosa (RP) progression and the visual contribution to posture stabilization; to examine the extent to which visual-field diameter affects the visual contribution to posture stabilization.

Methods. Posture information was recorded in 35 subjects with well-characterized RP and in 20 subjects with normal vision. Data were collected as each subject stood in a dark environment and as each subject viewed a stationary visual display. In both conditions, somatosensory feedback was concurrently altered. Data were also collected on 10 additional subjects with normal vision wearing field-restricting goggles (visual-field diameters ranged from 26.5 down to 6°).

Results. RP progression is accompanied by a steady decrease of the visual stabilization of posture, from normal values at the onset of the disease to the absence of visual stabilization and, eventually, to visual destabilization of posture. Decreasing visual field diameter in the subject with normal vision resulted in a linear decrease of the visual stabilization of posture. However, subjects with RP with comparable visual-field loss showed significantly lower visual stabilization than normal subjects with artificially restricted fields. Moreover, subjects with normal vision with restricted visual fields as small as 6° failed to show visual destabilization of posture.

Conclusions. Most likely, the additional reduction in the visual stabilization of posture shown in subjects with RP, as well as the visual destabilizing effect manifest in the late stages of RP, is caused by anomalous processing of visual information in the remaining visual field. Invest Ophthalmol Vis Sci 1993;34:3004-3010.

Retinitis pigmentosa (RP) is the joint name for a group of pigmentary retinal degenerations whose most salient visual manifestation is progressive visual field loss.1 This loss typically begins in the midperiphery, goes on to form a ring scotoma, and spreads both centrally and peripherally, eventually leading to severely contracted central visual fields. Other changes of visual performance include loss of night vision, reduced spatial2 and temporal3 contrast sensitivity, and, in late stages, reduced visual acuity. In their residual visual fields, many patients with RP experience visual abnormalities: increased thresholds for motion discrimination,4 large errors in judgments of spatial position,5,6 and poor spatial position precision.5

On the basis of such perceptual distortions, one may expect patients with RP to be impaired in their use of optic flow patterns to guide mobility and to maintain postural stability, in addition to limitations imposed by their restricted visual fields. Indeed, patients with advanced RP commonly report orientation and mobility problems.

Postural stability is maintained with a combination of vestibular (e.g., changes in angular and translational changes in head position), somatosensory (e.g., proprioception via cutaneous and muscle pressure, muscle length, and joint angle), and visual cues. Visual cues include flow movement of the entire environment, as well as changes in image size and retinal disparity. Motion information has been reported to provide a major contribution to visual posture stabilization7 because it allows observers to detect body oscillations relative to a stable background.8 Subjects with normal vision sway up to 50% more after closing
their eyes.9 This instability becomes even more pronounced if somatosensory feedback is altered. Studies of visual contributions to postural stability have been performed in elderly observers with normal vision10,11 and in younger observers with artificially degraded vision.8 When somatosensory information was reduced, postural sway was shown to covary with visual acuity,8,10 contrast sensitivity,10 and visual field restrictions.8,11 Approximately half the subjects with visual deficits in one study9 swayed more, by approximately 50%, with eyes open than with eyes closed; for these subjects, therefore, visual information provided a destabilizing effect. Unfortunately, this study provided no information on the nature of these subjects' visual deficits.

In our study, we examined the relationship between RP progression and the visual contribution to posture stabilization. Posture information was recorded in subjects with normal vision and in patients with well-characterized RP under conditions of a dark environment and a stationary visual display, while concurrently reducing somatosensory feedback.

MATERIALS AND METHODS

Apparatus and Display

We used the EquiTest System (NeuroCom International, Inc., OR) to measure postural sway. The apparatus is a three-sided booth in which the subject normally stands facing the rear. In our study, however, the subject faced the open side, toward a video display. The subject's feet were placed approximately 15 cm apart, and the subject wore a harness attached to an overhead support as a safety precaution.

Postural sway was estimated from the output of four pressure transducers that are located within the corners of the platform on which the subject stood. The pressure transducers measure vertical forces applied to the forceplate. The total vertical force is calculated as

\[ F = \sum_j F_j \]

where \( F_j \) is the supporting force of the \( j \)th transducer. The anterior-posterior center of vertical force, \( PY \), is the distance between the vertical projection of the subject's COG (center of gravity) and the \( x \)-axis. (The \( y \)-axis is the anterior-posterior axis relative to the center of the subject's body.)

\[ PY = \left[ \sum_j F_{j1} - \sum_k F_{k1} \right] / F \cdot d \]

where \( F_{j1} \) is the supporting force of the \( j \)th front transducers, \( F_{k1} \) is the supporting force of the \( k \)th rear transducers, and \( d \) is the distance between the force transducers and the \( x \)-axis, which is 10.7 cm. The vertical force position is assumed to be centered directly above a point on the forceplate that is equivalent to the vertical projection of the subject's center of gravity. The current value of the instantaneous \( y \)-axis position of the total vertical forces, \( PY(i) \), is passed through a second-order, low-pass filter to obtain an estimate of the position of the vertical projection of the COG, \( P_{COG} \).

When a subject stands in an upright manner, the COG is on a line inclined 2.3° forward from a vertical line passing through the ankle joints, and the height of the COG, \( H_{COG} \), is estimated as 0.55 of the subject's total height.12,13 When the body moves about the ankles, in an inverted pendulum fashion, the anterior-posterior COG sway angle is the angle formed by a line extending vertically from the center of foot support and a line extending from the center of foot support through the COG (Fig. 1). Thus, COG sway angle, \( \theta \), can be calculated as

\[ \theta(i) = \arcsin \left[ \frac{P_{COG}}{H_{COG}} \right] - 2.3° \]

COG sway angles effectively compensate for differences in subject height. The angular limits of stability are approximately the same for all adults regardless of height, whereas the limits of stability measured in
terms of the linear position of COG are not. In our study, for each trial, the pressure transducers sampled at a rate of 100 samples per second over a 20-second period. We use a standard deviation measure, the root-mean-square (RMS) error, of the COG sway angle sampled over the last 15-second period to index the magnitude of sway.

When an observer stands on a stable support surface, the normal anterior-posterior body sway results in inflection at the ankle joint, a reliable somatosensory cue to postural instability (Fig. 2A). In our study, subjects stood on a support surface that moved parallel with the subject’s anterior-posterior body sway (Fig. 2B). In this way, we were able to measure postural sway under conditions in which somatosensory information was altered. When the support surface is referenced to body sway, cues such as changes in cutaneous and muscle pressure, muscle length, and joint angle become less reliable in maintaining balance. The EquiTest System uses information about body sway to generate, by means of a servomotor, a rotation of the support surface around an axis collinear with the ankle joint, in proportion to the subject’s anterior-posterior sway. The servomotor has an output sensitivity of 1°/volt and a range of ±10°. Because the servomotor takes 50 msec to reach maximum velocity (50°/s at 4°/rotate), somatosensory feedback is not nulled, but it is altered and is thus less reliable.

A video display was used to show computer-generated patterns of high-contrast, randomly positioned dots. The luminance of the dots was 82.5 cd/m², and the luminance of the background was 0.2 cd/m². The average number of visible dots was 400, and each dot subtended a visual angle of 12.5 arcmin horizontally and 10.3 arcmin vertically. The large number of high-contrast, stationary visual elements makes the display an ideal visual stimulus for postural stabilization. The patterns were generated using a MATROX high-resolution graphics board (1024 × 1024 × 8 bits) controlled by an IBM-AT computer, and they were displayed on a rear-projection television (Mitsubishi model VS-403R). The display was refreshed at a rate of 50 Hz. The subjects viewed the display binocularly at a distance of 80 cm. At this viewing distance, the display subtended a visual angle of 53° horizontally and 41° vertically. However, all subjects wore field-restricting goggles, limiting their field of view to 51° horizontally and 38° vertically.

**Procedure**

Sway was measured in all subjects under two conditions: Subjects either viewed the stationary-dot display, or they stood in the dark. Each subject participated in three trials of each condition, yielding a total of six measures of postural sway. Subjects were instructed to look straight ahead and stand as still as possible with arms down at their sides. In the stationary dot condition, the visual pattern was displayed for 20 seconds. Viewing was binocular. In the dark environment condition, the display was turned off and the room remained dark for 20 seconds.

**Subjects**

Twenty people with normal vision and 35 people with RP served as subjects. The age range of the subjects with normal vision was 26 to 66 years (mean, 46.1 years), and the age range of the subjects with RP was 19 to 68 years (mean, 43.8 years). The subjects with RP were evaluated and diagnosed as typical RP* by a retina specialist. Atypical patients with RP, as well as patients with RP with cystoid macular edema, were excluded from the study. Visual acuity was measured binocularly on all subjects using the ETDRS acuity chart that was back-illuminated at 130 cd/m². Snellen acuity was converted to LogMAR values (the log of the minimum angle of resolution is the same as the log of the decimal equivalent of the Snellen acuity). LogMAR values ranged from −0.19 to 0.0 in the subjects with normal vision and from 0.0 to 0.9 in the subjects with RP. Visual fields were measured on each of the subjects with RP by kinetic perimetry with the Goldmann perimeter using the II/4e target (0.25° test spot at 320 cd/m²) and the V/4e target (1.75° test spot at 15 Snellen acuity was converted to LogMAR values (the log of the minimum angle of resolution is the same as the log of the decimal equivalent of the Snellen acuity). LogMAR values ranged from −0.19 to 0.0 in the subjects with normal vision and from 0.0 to 0.9 in the subjects with RP. Visual fields were measured on each of the subjects with RP by kinetic perimetry with the Goldmann perimeter using the II/4e target (0.25° test spot at 320 cd/m²) and the V/4e target (1.75° test spot at

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* Typical signs and symptoms of retinitis pigmentosa are night blindness; midperipheral ring-like scotoma or contracted visual fields; intraretinal bone spicule-like pigmentation; narrowed retinal arterioles; vitreous degeneration with cellular debris and pigment dusting; reduced or unrecordable electroretinogram; loss of foveal reflex; and no known systemic, metabolic, inflammatory, dietary, toxic, or traumatic cause of retinal degeneration.
320 cd/m²) on a background of 10 cd/m². The visual fields were used in combination with previously measured visual fields to estimate disease progression. The age of visual-field-loss onset, i.e. critical age, was derived from the longitudinal samples of log visual field area obtained with the Goldmann H/4e target under a model of first order exponential decay. The number of visual field measurements per subject with RP ranged from 1 to 12 (mean, 5.7), with 29 of the 35 subjects with RP having more than 1. The multiple visual field measurements for the 29 subjects with RP took place over a span of 1.5 to 16 years (mean, 10.0 years). Disease progression was indexed by the number of years past critical age (YPCA). All subjects wore their refractive corrective lenses during the experiment. Informed consent was obtained from each subject after the nature and possible consequences of the study were described. The research followed the tenets of the Declaration of Helsinki, and it was approved by the institutional human experimentation committee.

RESULTS

Figure 3 shows graphs of COG sway angle plotted over time. Figure 3A shows one of the three sets of sway paths of a subject with normal vision, and Figure 3B shows one of the three sets of sway paths of a subject with RP. The thick line represents the sway angle of the subject while viewing a visual display, and the thin line represents the sway angle of the subject in the dark. Note the difference in sway angle deviations between the visual display and the dark conditions of the subject with normal vision. The computed RMS values are 0.69 for the visual display condition and 1.19 for the dark condition. Apparently, the visual display served as a sway-reducing agent. There was little difference in sway angle deviations, on the other hand, between the visual display and the dark conditions in the subject with RP. The computed RMS values are 1.19 for the visual display condition and 0.96 for the dark condition. Apparently, no visually mediated sway reduction occurred.

In Figure 4, the magnitude of sway in the dark is plotted against the magnitude of sway with the visual display. The data of the subjects with RP and normal vision are plotted as open and closed symbols, respectively. Each data point is the median sway magnitude of three trials. The solid line in the figure represents equivalent sway for the two conditions. Data falling along this line indicate the absence of a visual contribution to postural stability. Data falling above the line indicate a visual stabilizing effect; subjects swayed less with the presentation of a visual display than in the dark. Data falling below the line indicate a visual destabilizing effect; subjects swayed more with a visual display than in a dark environment.

To determine whether the subjects with normal vision or the subjects with RP used visual information to stabilize their posture, we used a nonparametric test, the Wilcoxon test for two correlated samples, to assess the similarity in distributions of sway in the dark and sway with a visual display. The normal vision group showed a significant difference in sway between the visual and dark conditions ($w_{obs} = 12$, $P < 0.01$). The RP group showed only a marginally significant difference in sway between the visual and dark conditions ($w_{obs} = 192.5$, $P < 0.05$). The lack of a strong effect of vision on performance in the RP group is demonstrated in Figure 4 by the clustering of...
group is significantly different from the distribution of visual stabilization of posture than the subjects with RP, on the average, showed less variation toward the lower end of the visual stabilization indices; acuity is artificially degraded by semitransparent plastic foils. Another visual characteristic reportedly associated with sway magnitude is visual field area. In the Paulus et al study, visual field defects were simulated in younger observers by means of a coverplate attached to a helmet. As the visual field area decreased, so did postural stability.

We ran a hierarchical multiple regression analysis to determine which variable or variables among the set of putative causal variables—logMAR, log visual field area, age, and disease progression—affects the visual stabilization index significantly and appreciably. Disease progression, indexed by YPCA, has the highest correlation of any independent variable with the visual stabilization index ($r = -0.585$). The coefficient of determination, $R^2$, which is the proportion of the variation of one variable determined by the variation of the other, is 0.345 and is highly significant ($P < 0.0005$). When we regress the visual stabilization index on age as well as YPCA, we obtain $R^2 = 0.419$. Again, this coefficient is highly significant ($P < 0.0002$). The results of a significance test for additional independent variables reveal that the increase in $R^2$ due to the addition of age to YPCA in the regression equation is marginally significant ($P < 0.05$). Including logMAR does not change $R^2$, and including log visual field area to the other three independent variables decreases $R^2$ to 0.395.

Figure 5 illustrates RP progression accompanied by a steady decrease in the visual stabilization of posture, from normal values at the onset of the disease to the absence of visual stabilization and, eventually, to visual destabilization of posture. The solid line indicates the regression line, the dashed line indicates the mean of the normal-vision distribution, and the dotted line indicates the absence of a visual effect on stabilization. Six of the seven subjects with RP with an estimated YPCA of 32 years or more showed a visual destabilization of posture; they swayed more with a visual display than they did in the dark.

In RP, disease progression is accompanied by a steady decrease of the peripheral visual field. In the late stages of the disease, subjects with RP typically have severely contracted fields. To examine the extent to which visual field diameter alone affects the visual contribution to posture stabilization, data were collected on subjects with normal vision wearing field-restricting goggles.

**ARTIFICIALLY-RESTRICTED VISUAL FIELDS**

**Methods**

The apparatus, display, and procedure were the same as described above, with the exception that postural onset ages are clustered in the first few decades of life, and thus age and YPCA covary significantly. Thus, a significant correlation between the age of the subjects with RP and the magnitude of the visual stabilization index is expected and, in our study, confirmed ($r = -0.52, P < 0.000$). That age per se is not a critical factor is supported by the significant partial correlation between the visual stabilization index and YPCA, with age held constant ($r = -0.46, P < 0.001$).
FIGURE 5. Visual stabilization index plotted against years past critical age. The solid line represents the best fitting linear regression. The dashed line indicates the normal distribution mean, and the dotted line indicates the absence of a visual effect on stabilization.

 sway was measured as each subject wore goggles to restrict field of view to visual-field diameters of 26.5, 15.5, and 6°.

Subjects
Ten people with normal vision served as subjects. The ages ranged from 34 to 59 years (mean, 46.4 years). LogMAR values ranged from -0.19 to 0.0. Viewing was binocular, and all subjects wore their refractive corrective lenses during the experiment.

Results
In Figure 6, the visual stabilization index is plotted against visual field diameter. The closed symbols represent the mean of the 10 subjects with normal vision with artificially restricted visual fields (error bars are ±2 SEM). (The closed data point at 51° is the mean of the 20 subjects with normal vision in the first experiment.) As the visual field diameter decreases, the stabilizing effect of vision decreases linearly. The data are well fit by the line, visual stabilization index = 1.082 + 0.013 x visual field diameter. With no vision (visual field diameter = 0), the stabilizing effect of vision is approximately nil (visual stabilization index ~1). Of particular importance is the finding that visual field diameters of 6° (smaller than any of the V/4e Goldmann visual fields of the subjects with RP) never resulted in a destablizing visual effect on posture.

Superimposed on the artificially restricted field data in Figure 6 are the data of the subjects with RP from the first experiment. The visual-field diameters of the subjects with RP were estimated to the nearest 2° of the central-most intact region of the Goldmann visual fields using the V/4e target, right eye. (All visual fields are truncated at 51° because of the field-restricting goggles worn by the subjects in the first experiment.) As shown in Figure 6, there is a decreasing trend in the visual stabilization index with decreasing visual field diameter, similar to the artificially restricted field data. However, the majority of the RP data fall below 2 SEM of the visual field-matched normal vision data. And, unlike the artificially restricted field data, several subjects with RP show a destabilizing effect of vision (some with visual-field diameters as large as 30°, and one with a visual-field diameter of 51°).

Different symbols differentiate the data of the subjects with RP with (open triangles) and without (open squares) peripheral islands. Two points are worth noting. One is that visual information extending no further than 3° to 7° in the retinal periphery (6° to 14° diameter) is sufficient for visual stabilization of posture. The other is that the presence of a peripheral island does not assure a visual stabilizing effect. (As an aside, the visual stabilization distribution of the subjects with RP with peripheral islands is not significantly different from the visual stabilization distribution of the subjects with RP without peripheral islands (z_{obs} = 1.85, P > 0.05.)

DISCUSSION
The results of this study demonstrate that the extent to which visual information affects postural stability differs between subjects with RP and normal vision. On average, subjects with normal vision show greater visual stabilization than subjects with RP. Taken individually, not every subject with RP shows the same
effect as the group as a whole. Some individuals with RP had visual stabilization indices as high as subjects with normal vision, whereas others showed destabilization. Statistically, however, the regression line in Figure 5 shows that RP progression is accompanied by a steady decrease of the visual stabilization index, from normal values at the onset of the disease to unity (absence of visual stabilization) and, eventually, to values below unity (visual destabilization of posture).

The statistically significant correlation of log visual field area and the magnitude of the visual stabilization, together with the field restriction results, suggest that visual field loss can partially account for the decreased visual stabilization of posture in subjects with RP. Visual stabilization decreased linearly with increases in visual field restriction (Fig. 6). But, visual field loss, alone, cannot totally explain the RP results. Approximately two-thirds of the subjects with RP had visual stabilization values lower than 2 SE away from the mean of visual field-matched subjects with normal vision. Moreover, subjects with normal vision with restricted visual fields as small as 6° failed to show the visual destabilization effect manifest in the late stages of RP (Fig. 5). Most likely, the additional reduction in the visual stabilization of posture found in subjects with RP, as well as the visual destabilization, is the result of anomalous processing of visual information in the remaining visual field. Photoreceptors throughout the RP retina gradually deteriorate, as is demonstrated by changes in spatial and temporal contrast sensitivity, acuity, motion thresholds, and perception of spatial position. A combination of any of the above may be the cause of decreased visual stabilization in people with retinitis pigmentosa. Thus, it may be more accurate to describe the loss of a visual contribution to posture stabilization—and, likewise, other problems of orientation and mobility experienced by patients with RP—as a consequence of multiple retinal changes.

Key Words
retinitis pigmentosa, low vision, postural stability, self-motion, visual fields

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