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# Vertical Heterophoria and Susceptibility to Visually-induced Motion Sickness

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## Abstract

Motion sickness is reported to be a common symptom in patients with vertical heterophoria. The goal of this study was to assess the relationship between vertical phoria and susceptibility to motion sickness in a non-clinical sample of 43 subjects. Vertical phoria was measured with a Maddox rod after 30 s of occlusion. To evaluate susceptibility to motion sickness, subjects read text while sitting inside a rotating optokinetic drum for 10 min. Subjects rated their level of motion sickness at 1 min intervals during drum rotation and the magnitude of 13 motion-sickness symptoms after drum rotation ended. The magnitude of vertical phoria ranged from 0 to 2.13 prism diopters (pd) with a mean of 0.46 pd and correlated significantly with both the maximum rating of motion sickness during drum rotation and the summed symptom score following rotation. A vertical phoria of 0.75 pd discriminated best between subjects with low vs. high summed motion-sickness-symptom scores ($p < 0.0001$). Introducing a prism to artificially increase the phoria of 12 subjects with vertical phorias < 0.75 pd increased motion-sickness symptoms in only 1 subject. Prisms that reduced the phoria of subjects with vertical phorias > 0.75 pd reduced motion-sickness symptoms in 2 of the 4 subjects tested. The results confirm an association between vertical phoria and motion sickness, but suggest the relationship may not be causal.

### Keywords

hyperphoria; hypophoria; motion sickness; vertical phoria

## INTRODUCTION

Hyperphoria, or vertical phoria, is the tendency of one eye to deviate vertically when fusional vergence is interrupted and not available to maintain single vision. Scobee and Bennet (1950) reported that 35% of their sample of 1476 clinical patients had a vertical phoria greater than or equal to 0.5 prism diopters (pd). The prevalence of vertical phoria in asymptomatic clinical patients and in non-clinical samples typically is found to be in the range of 10 – 20% (Abraham, 1931; Amos & Rutstein, 1987). According to Amos and Rutstein (1987), who summarized the published data on vertical phorias, the presence of hyperphoria is unrelated to age or refractive error.

Even small amounts of vertical phoria have been associated with symptoms such as asthenopia, diplopia, loss of place while reading, drowsiness, fatigue, postural deficiency, vertigo, nausea, and motion sickness (Amos & Rutstein, 1987; Duke-Elder, 1949; Hansell,

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1892; Marlow, 1934; Matheron & Kapoula, 2008; Rosner & Feinberg, 2005; Scheiman & Wick, 2008; Scobee & Bennet, 1950). Further, the introduction of a 2 pd vertical prism was reported to produce changes in the postural stability of subjects with normal vertical eye alignment (Matheron et al., 2007). Clinical testing for a vertical phoria is usually performed by dissociating the eyes and measuring the misalignment between them with an alternating cover test or a hand held Maddox rod, or using the von Graefe technique in conjunction with the phoropter (Amos & Rutstein, 1987; Scheiman & Wick, 2008). If a vertical misalignment is found, the most common treatment is vertical prism to neutralize the phoria or to balance the vertical vergence range (Amos & Rutstein, 1987; Benjamin, 2006; Scheiman & Wick, 2008). Treatment usually is reserved for patients who have troublesome symptoms (Amos & Rutstein, 1987; Scheiman & Wick, 2008; Scobee & Bennet, 1950). In their clinical sample, Scobee and Bennet (1950) determined that only 17% percent of patients with 0.5 pd or more of vertical phoria exhibited clinically significant symptoms.

Motion sickness is a feeling of dizziness or nausea that can occur when discordance exists between different neural sources of information about movement, for example between visual and vestibular signals of self motion (Probst & Schmidt, 1998; Reason, 1978). Common associated symptoms are malaise, cold sweats, and vomiting. Motion sickness can occur naturally, as in the cabin of a boat or the passenger seat of a car, or can be stimulated experimentally, for example, using moving visual displays (Kennedy et al., 2010). In particular, OKN drums have been used for over 30 years in experiments of visually induced motion sickness (Lackner & Teixeira, 1977; Webb, 2000).

In the clinical literature, the presence of a hyperphoria is associated with symptoms of motion sickness. Hansell (1892) documented motion sickness as a prominent symptom in a case series of 13 patients with hyperphoria. An association between hyperphoria and motion sickness continues to be reported in current clinical texts (Benjamin, 2006; Scheiman & Wick, 2008). Although the relationship between hyperphoria and motion sickness frequently is considered to be causal, to our knowledge no published data specifically address either the strength of the association between hyperphoria and motion sickness or the underlying basis of this relationship. The goal of the present study was to evaluate the relationship between hyperphoria and the susceptibility to visually-induced motion sickness in a non-clinical sample of subjects. Susceptibility to motion sickness was evaluated during full-field rotation of the visual scene with respect to the stationary observer.

## MATERIAL AND METHODS

The sample consisted of forty-three adult subjects, twenty-one females and twenty-two males who ranged in age from twenty two to sixty one years old. Subjects were recruited non-systematically by word of mouth from the faculty, students, and staff of the University of Houston College of Optometry and their family members. All had normal ocular motility and reported normal corrected or uncorrected vision. No other selection criteria were applied and subjects with or without a history of hyperphoria and motion sickness symptoms were included. None of the subjects exhibited an obvious head tilt. In the subjects who wore their habitual spectacles, vertical alignment of the optical lens centers was assumed to be correct. The project was reviewed by the University of Houston Committee for the Protection of Human Subjects and subjects provided written informed consent before participating.

### Experiment 1: Vertical phoria and motion sickness scores

Testing of each subject began with a measurement of the magnitude of his or her vertical phoria using a Maddox rod and vertical prism bar. The subject was seated in a dark room with his or her head erect and stabilized by a chinrest. After allowing approximately 2 minutes for dark adaptation, the subject fixated a 5 mm (4.3 arc min) yellow light-emitting

diode presented at eye level at a distance of four meters. These few minutes of dark adaptation ensured that the fixation target was readily visible. The subject held a vertically oriented Maddox rod in front of the left or right eye, assigned by the experimenter in a pseudo-random order. When the subject confirmed that he or she was viewing binocularly and could clearly see a horizontal yellow streak as well as the fixation light, the eye that was viewing through the Maddox rod was occluded for thirty seconds. After thirty seconds of dissociation, this eye was uncovered briefly, and the subject described the position of the horizontal streak of light in relation to the fixation light. If the streak did not pass through the middle of the fixation light, the experimenter introduced a vertical prism bar (Astron International, Naples, FL) between the occluder and the Maddox rod to neutralize the perceived misalignment. The eye that viewed now through the Maddox rod and the prism was uncovered again briefly. Vertical prism was adjusted in half prism-diopter steps until the subject perceived the horizontal streak to pass through the middle of the fixation light. In some cases it was necessary to bracket this endpoint and take the midpoint between two prism values. The measurement then was repeated with the Maddox rod and prism bar in front of the fellow eye. The vertical phoria of each eye was measured twice and averaged.

Approximately 5 min after completing the phoria measurements, each subject's susceptibility to motion sickness was evaluated. The subject was seated at the center of a 145-cm diameter OKN drum, the inside of which was covered by alternating gray and white vertical 1-cm stripes and randomly distributed 7.5 cm × 10 cm black rectangles. A black cloth covered the floor so that no external stationary contours were visible during the drum rotation. The subject was instructed to read aloud a passage of text, presented at eye level at a distance of 40 cm on a clear Lucite stand placed inside the drum. Six passages of text were available (Texas Education Agency, 2003), each written at an $8^{th}$ grade reading level and typed in fourteen point Calibri font with 1.5 line spacing. When the subject was positioned inside of the drum, the room lights were extinguished and the drum began to rotate at 37 deg/s in either the clockwise or counter-clockwise direction. The experimenter turned on a light inside the drum and the subject was signaled to begin reading. Although the rotating OKN drum induces a perception of circular rather than linear self motion, the testing condition was intended to simulate reading while in a moving vehicle, which is known to produce motion sickness in susceptible individuals (Probst et al., 1982; Turner & Griffin, 1999). In addition to reading aloud, the subject indicated when he or she perceived self-rotation to begin and rated his or her level of motion sickness on a scale of zero to six at one minute intervals, with zero being no symptoms and six being moderate nausea with a need to stop testing (Webb & Griffin, 2003; also see Table 1). Clockwise or counterclockwise rotation of the drum lasted for 5 minutes, followed by a brief interlude of darkness and then 5 minutes of drum rotation in the opposite direction. After a total of ten minutes of drum rotation, the subject rated thirteen symptoms of motion sickness on a scale of 0 to 3, using a previously validated instrument (Webb, 2000). The subject was informed of the list of symptoms (Table 2) before the start of OKN-drum rotation. Afterwards, the subject was instructed to report the *maximum* rating for each symptom during the preceding ten-minute period of drum rotation. The subject's ratings for these symptoms were summed to give a total post-exposure symptom score. This score, as well as the maximum rating of motion sickness during the ten minutes of rotation of the OKN drum, was compared to the subject's average vertical phoria.

## Experiment 2: Induction and correction of phoria and motion sickness scores

After the results of Experiment 1 were collected, the subjects were divided into two categories, based on the magnitude of their vertical phoria. Subjects with an average vertical phoria less than and greater than 0.75 pd were categorized as having a “low” or a “high” phoria, respectively. The purpose of Experiment 2 was to add vertical prism in front of one

eye to increase the vertical phoria of low-phoria subjects or to decrease the vertical phoria of high-phoria subjects, and determine whether these manipulations produce a change in the subjects' ratings of motion sickness.

Twelve subjects in the low-vertical-phoria category (mean phoria = 0.27 ± 0.22 [SD] pd) and four subjects in the high-vertical-phoria category (mean phoria = 1.33 ± 0.55 pd) were retested. First, phoria measurements were repeated in the same manner as in Experiment 1, above. For the subjects in the low-vertical-phoria category, the Maddox rod was then removed and the subject fixated on the yellow light. Using the vertical-prism bar, base-up prism was introduced in front of the subject's more hyperphoric eye until the subject was unable to fuse the fixation light. In two subjects with the same measured phoria in the left and right eyes, the eye that received the prism was chosen randomly. The largest magnitude of base-up prism that the subject could fuse was then introduced, typically in front of the subject's more hyperphoric eye. For the subjects in the high-vertical-phoria category, a correcting prism was defined as the base up or base down prism, rounded to the nearest half pd, that was needed to correct the eye with the higher average vertical phoria measurement. This correcting prism was placed in front of the eye with the larger vertical phoria. For subjects who did not wear spectacles, the vertical prism (35 mm in diameter) was introduced in front of the eye using a trial frame. For subjects who wore spectacles, the vertical prism was attached to the spectacle frame using a Halberg clip. Both the trial frame and the Halberg clip had 32-mm apertures, to minimize truncation of the peripheral visual field. As soon as the inducing or corrective prism was introduced, the subjects were placed in the OKN drum and retested for susceptibility to motion sickness as described for Experiment 1, above.

After wearing the modifying prism in the OKN drum for ten minutes, each subject was tested for prism adaptation (Ellerbrock, 1950; Henson & North, 1980; Ogle & Prangen, 1953). Adaptation was quantified by re-measuring each subject's vertical phoria following the rotation of the optokinetic drum, immediately after the subject removed the inducing or the correcting prism. The same procedure that was described above for Experiment 1 was used. Adaptation was defined as the 100% × (post-testing phoria – pre-testing phoria) / power of the inducing or correcting prism.

## Data Analyses

Hyperphoria of the right eye and hypophoria of the left eye were considered to be positive, and hypophoria of the right eye and hyperphoria of the left eye were considered to be negative. Signed values were used to compute the correlation between the phoria measured for each subject when the right and left eye was viewing. Average vertical phorias were then calculated for each subject by combining the pairs of measurements obtained during fixation with the right and left eye. Signed values of the average phoria were used to calculate the magnitude of prism adaptation in Experiment 2. All of the other analyses that are reported below were performed using the (unsigned) absolute values of the subjects' average vertical phorias. Although the distributions of the (unsigned) vertical phorias and maximum motion sickness rating are skewed (see Results), the Pearson product-moment correlation coefficient, r, and the t test are robust to moderate levels of skew if the sample is not small (Faber, 1988; Ratliff, 1968). The correlational and comparative analyses reported below therefore were performed using these parametric statistics.

# RESULTS

## Experiment 1

The 43 subjects in the study had average unsigned hyperphorias that ranged from 0 to 2.13 pd, with a mean value of 0.46 and a standard deviation of 0.40 pd (Figure 1). Because the histogram shown in Figure 1 plots unsigned values of the hyperphoria, the distribution is skewed significantly to the right (skew = 1.95, 95% confidence interval = 0.80 to 3.68). For the 43 subjects, the Pearson product-moment correlation between the average (signed) vertical phoria in the two eyes is 0.81, which is highly significant ($t_{df=41} = 8.86$, $p = 5.7 \times 10^{-11}$).

The maximum rating of motion sickness during rotation of the OKN drum ranged from 0 to 6, with a mean of 2.5 ± 1.4 (SD). The summed post-exposure symptom scores ranged from 0 to 23 out of a possible 39, with a mean value of 9.1 ± 6.2. The two measures of motion sickness are correlated ($r = 0.69$, $p = 3.3 \times 10^{-7}$). The distributions of the maximum motion sickness rating and the total post-exposure scores are positively skewed (for maximum rating, skew = 0.68, 95% confidence interval = 0.24 to 1.27; for summed post-exposure scores, skew = 0.48, 95% confidence interval = -0.01 to 1.02), although to a lesser extent than the distribution of the subjects' hyperphorias.

A significant positive correlation exists between the magnitude of hyperphoria and the severity of motion sickness, as quantified by the maximum motion-sickness rating in the OKN drum (r=0.41, p=0.0058) and the summed post-exposure symptom ratings (Figure 2; r = 0.33, p=0.031). Both of these correlations remain significant if one subject with a vertical phoria of 2.13 pd is eliminated from the analysis (for maximum motion-sickness rating, r = 0.32, p = 0.039; for the summed post-exposure symptom ratings, r = 0.31, p = 0.047). To evaluate these relationships further, we compared the symptom scores of subjects with hyperphorias that were larger and smaller than a criterion value, which we varied systematically from 0.5 to 0.9 pd. This analysis indicated that a criterion hyperphoria of 0.75 pd yields the greatest difference in symptom scores between the low and high phoria groups. Specifically, 5 of the 8 subjects with an average hyperphoria greater than 0.75 pd had a maximum motion-sickness rating in the OKN drum of 5 or more, compared to only one of the 35 subjects with hyperphorias less than 0.75 pd. Similarly, 7 of the 8 subjects with a hyperphoria greater than 0.75 pd had a summed post-exposure symptom score of 12 or more, compared to 10 of the 35 subjects with hyperphorias less than 0.75 pd. Student t tests confirmed that the differences between the subjects with phorias less than and greater than 0.75 pd in maximum motion-sickness ratings ($t_{df=41} = 4.79$, $p=2.2 \times 10^{-5}$) and the summed post-exposure symptom scores ($t_{df=41} = 3.90$, $p=3.5 \times 10^{-4}$) are highly significant.

On the other hand, no significant relationship exists between the magnitude of hyperphoria and the latency of perceived self motion in the optokinetic drum ($r = 0.01$, $p = 0.93$). Similarly, the latency to perceive self motion is unrelated to the maximum motion sickness rating ($r = 0.17$, $p = 0.32$) and to the summed post-exposure symptom score ($r = 0.06$, $p = 0.74$).

Neither gender nor age are related significantly with the average hyperphoria measurements or with the susceptibility to visually-induced motion sickness. The average phoria measurements are 0.49 ± 0.12 (standard error) pd for women and 0.44 ± 0.04 pd for men ($t_{df=41} =0.48$, $p=0.63$). The maximum motion-sickness ratings for women and men average 2.91 ± 0.32 and 2.14 ± 0.27, respectively ($t_{df=41} =1.85$, $p=0.07$), and the summed post-exposure symptom totals are 10.05 ± 1.29 for women and 8.11 ± 1.39 for men ($t_{df=41} =1.02$, $p=0.31$). When the results for both genders are combined, the correlation between hyperphoria and age is 0.02 ($p = 0.91$). The correlations between the maximum motion-

sickness ratings in the optokinetic drum and the summed post-exposure symptom ratings and the subjects' age are 0.08 (p = 0.60) and -0.11 (p=0.50), respectively.

### Experiment 2

Twelve subjects with a hyperphoria less than 0.75 pd were re-tested in the optokinetic drum while wearing a vertical prism that was intended to increase their phoria. The magnitude of the inducing prism ranged from 1 – 3.5 pd (average = 2.1 pd), producing an average of 1.7 ± 0.5 pd of hyperphoria. In addition, 4 subjects with a hyperphoria greater than 0.75 pd (range = 0.9 to 2.1 pd) were re-tested while wearing a vertical prism that reduced their vertical phoria to between 0 and 0.4 pd. As shown in Figure 3, a substantial increase in the summed post-exposure symptom score was observed in response to a prism-induced increase in hyperphoria in only one of the twelve subjects. A reduction of the summed post-exposure symptom score occurred in two of the four subjects who wore prism to decrease their vertical phoria. Similar changes were observed in the maximum motion-sickness ratings in the optokinetic drum for the two groups of subjects.

The expected impact of the inducing or correcting prisms would be reduced if the subjects adapted to the vertical prisms (Ellerbrock, 1950; Henson & North, 1980; Kono et al., 1998; Ogle & Prangen, 1953). Adaptation averaged 17 ± 22% (SD) among the twelve subjects who wore an inducing prism and 43 ± 21% among the four subjects who wore a correcting prism. The difference in adaptation between the two groups of subjects does not reach statistical significance ($t_{df=14} = 2.08$, p = 0.057). The one subject whose symptoms increased substantially while wearing an inducing prism exhibited 35% adaptation. The two subjects whose symptoms decreased while wearing correcting prisms exhibited 31% and 73% adaptation.

## DISCUSSION

Previous clinical studies reported a high prevalence of symptoms of motion sickness in clinical patients with vertical phoria (Doble et al., 2010; Hansell, 1892; Schrier, 1997). Although findings for clinical and non-clinical samples are not always similar, our results for a *non-clinical* sample of 43 subjects are consistent with these reports. In particular, subjects with vertical phorias greater than or equal to 0.75 pd exhibited significantly more severe symptoms of motion sickness, when exposed to full-field visual motion.

Previous studies also indicated that symptomatic patients with hyperphoria report improvement when prism is used to reduce or neutralize the vertical phoria (Doble et al., 2010; Hansell, 1892; Rosner & Feinberg, 2005; Sethi, 1986). In addition, Matheron and Kapoula (2008) showed that prismatic correction improved postural stability when normal subjects with only small amounts of vertical phoria (0.25 - 0.75 pd) viewed a target at 2 m. The results of our Experiment 2 are not definitive, as only two of four subjects with a vertical phoria greater than 0.75 pd reported a reduction in the symptoms of visually-induced motion sickness while wearing a corrective prism.

In Experiment 2, the amount of phoria produced by the introduction of an inducing or correcting prism could have decreased or increased during optokinetic stimulation as the result of prism adaptation. In the literature, the reported rates of adaptation to vertical phoria vary considerably (Brautaset & Jennings, 2005; Eskridge, 1988; Henson & North, 1980; Kono et al., 1998; Ogle & Prangen, 1953; Rutstein & Eskridge, 1986; Sethi, 1986). Some studies suggest that symptomatic subjects with hyperphoria adapt less completely to vertical prism than subjects without hyperphoria (Ogle & Prangen, 1953; Rutstein & Eskridge, 1986). On average, the subjects in Experiment 2 averaged approximately 25% adaptation after 10 minutes, which is less than the value of approximately 50% reported after a 10-

minute period of adaptation by Kono et al. (1998). However, our subjects sat inside of a moving OKN during prism wear and a large portion of the visual field consisted of vertical stripes that provide no vertical disparity information. Future studies that document the subjects' vertical eye position during optokinetic stimulation could provide additional information about the relationship between the magnitude of vertical phoria and symptoms of motion sickness.

Doble et al. (2010) suggested that maintaining fusion in the presence of a vertical phoria fatigues the vertical extra-ocular muscles and that the reported symptoms, including motion sickness, are a result of this fatigue. In particular, the authors hypothesized that a consequence of muscle fatigue is tremor, which generates unintended movements of the retinal image and lead to motion sickness. Matheron et al. (2007) reported that postural stability worsens when normal subjects view either a near or distant target with a 2 pd vertical prism in front of their non-dominant eye. This observation is relevant because postural instability has been suggested to be an important precursor to the development of motion sickness (Smart et al., 2002; Stoffregen & Smart, 1998). However, the influence of vertical prism on posture appears to be complicated, as the study by Matheron et al. (2007) indicated that postural stability *improves* significantly, in comparison to the no-prism condition, if a target at 2 m (but not at 40 cm) is viewed with a vertical prism in front of the dominant eye. Previously, the same research group found that the introduction of a non-fusable 5 pd vertical prism in front of the dominant eye increases the magnitude of body sway during binocular viewing at 90 cm (Isotalo et al., 2004). However, increased body sway was observed also when a 5 pd prism was placed in front of the dominant eye during *monocular* viewing.

The outcome of our Experiment 2 suggests that the presence of a vertical phoria and susceptibility to visually-induced motion sickness may *not* be related causally, as a prism-induced increase of the phoria produced a substantial worsening of motion-sickness symptoms in only one of the twelve subjects tested. This outcome is consistent with the possibility that a vertical phoria and susceptibility to motion sickness represent separate manifestations of a common supra-nuclear condition, for example, abnormal information from the vestibular otolith organs (Diamond & Markham, 1992; Helling et al., 2003; Karmali et al., 2006; Maxwell & Schor, 1996). This possibility is consistent with the observation (related to us by one of the reviewers of this paper) that patients with congenital superior oblique palsy, who can have extremely large vertical phorias, rarely or never report symptoms of motion sickness.

Although vertical phoria and susceptibility to visually-induced motion sickness are related, 30% of our subjects with vertical phorias less than 0.75 pd had summed post-exposure symptom scores greater than 12. Two of these subjects had total symptom scores that were higher than the average score (15.5) of the subjects with vertical phorias greater than 0.75 pd. Susceptibility to motion sickness in the subjects with low vertical phorias may be attributable to a different physiological mechanism than in the subjects with higher vertical phorias. Our finding that an artificial increase in vertical misalignment did not increase the symptom scores of most subjects with low vertical phorias is consistent with this interpretation.

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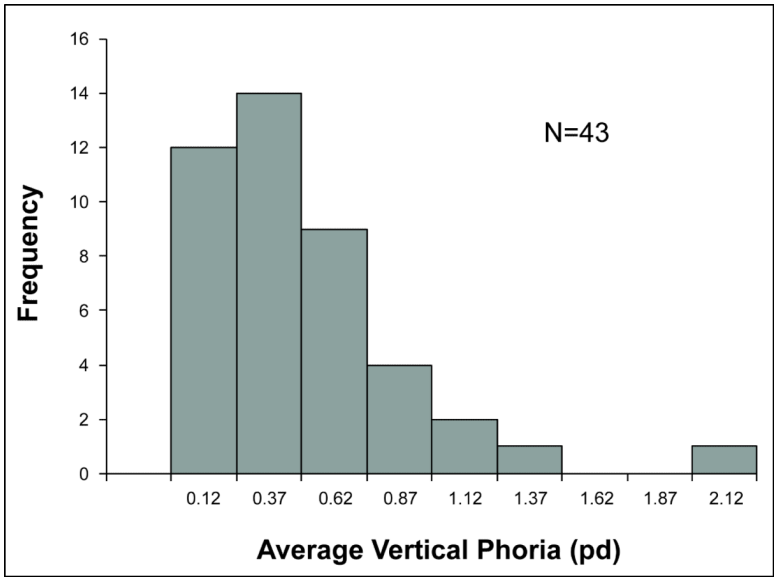
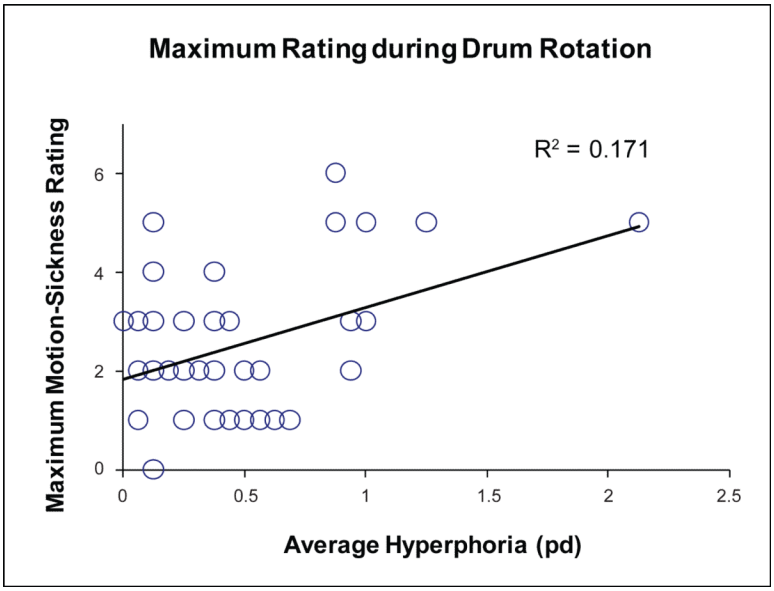



**Figure 1.**
Histogram showing the frequencies of average vertical phorias in 43 subjects.



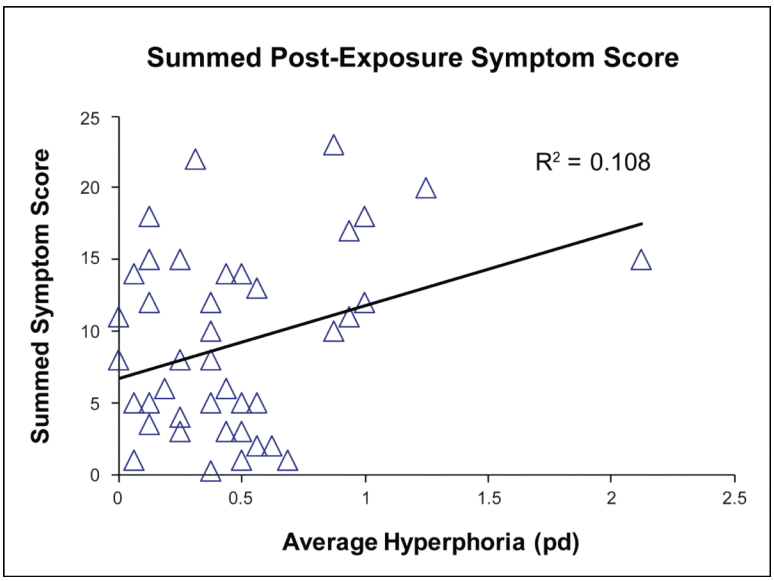




**Figure 2.**
Scatter plots of each subject's average vertical phoria *vs.* (a) maximum motion sickness ratings during optokinetic drum rotation and (b) summed post-exposure symptom scores.

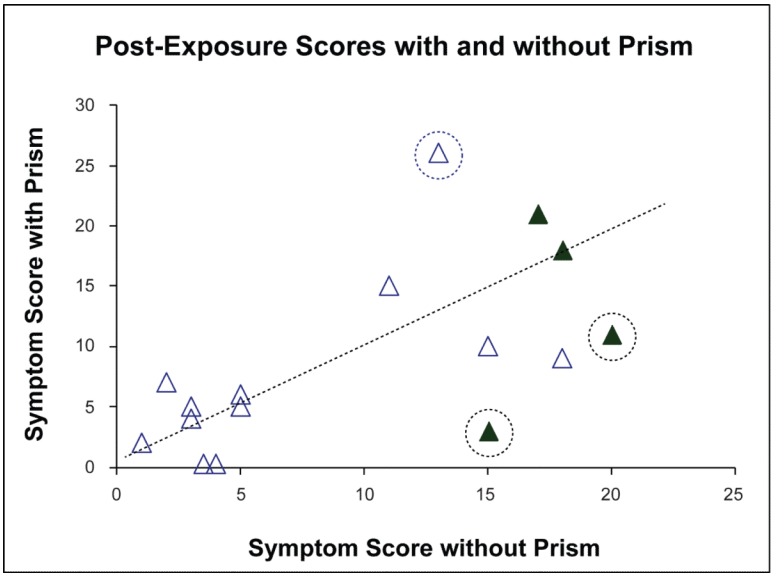



**Figure 3.**

Summed post-exposure symptom scores of 12 low-phoria subjects (unfilled symbols) and 4 high-phoria subjects (filled symbols), determined with and without inducing or correcting vertical prisms. The dashed line indicates no change in the post-exposure symptom score with prism. Data points for one low-phoria subject whose post-exposure symptom score increased with an inducing prism and two high-phoria subjects whose post-exposure symptom scores decreased with a correcting prism are circled.