

Effect of Developmental Binocular Vision Abnormalities on Visual Vertigo Symptoms and Treatment Outcome

Marousa Pavlou, PhD, James Acheson, MRCP (UK), FRCS (Glas), FRCOphth, Despina Nicolaou, BMedSci (Hons), Orthoptics, MMedSci Orthoptics, Clare L. Fraser, MBBS (Hons), MMed, FRANZCO, Adolfo M. Bronstein, MD, PhD, FAAN FRCP, and Rosalyn A. Davies, FRCP, PhD

Background and Purpose: Customized vestibular rehabilitation incorporating optokinetic (OK) stimulation improves visual vertigo (VV) symptoms; however, the degree of improvement varies among individuals. Binocular vision abnormalities (misalignment of ocular axis, ie, strabismus) may be a potential risk factor. This study aimed to investigate the influence of binocular vision abnormalities on VV symptoms and treatment outcome.

Methods: Sixty subjects with refractory peripheral vestibular symptoms underwent an orthoptic assessment after being recruited for participation in an 8-week customized program incorporating OK training via a full-field visual environment rotator or video display, supervised or unsupervised. Treatment response was assessed at baseline and at 8 weeks with dynamic posturography, Functional Gait Assessment (FGA), and questionnaires for symptoms, symptom triggers, and psychological state. As no significant effect of OK training type was noted for any variables, data were combined and new groups identified on the basis of the absence or presence of a binocular vision abnormality.

Results: A total of 34 among 60 subjects consented to the orthoptic assessment, of whom 8 of the 34 had binocular vision abnormalities and 30 of the 34 subjects completed both the binocular function assessment and vestibular rehabilitation program. No significant between-group differences were noted at baseline. The only significant between-group difference was observed for pre-/post-VV symptom change ($P = 0.01$), with significant improvements noted only for

the group without binocular vision abnormalities ($P < 0.0005$). Common vestibular symptoms, posturography, and the FGA improved significantly for both groups ($P < 0.05$).

Discussion and Conclusions: Binocular vision abnormalities may affect VV symptom improvement. These findings may have important implications for the management of subjects with refractory vestibular symptoms.

Video Abstract available for insights from the authors regarding clinical implication of the study findings (see Video, Supplemental Digital Content 1, <http://links.lww.com/JNPT/A115>).

Key words: *binocular vision, vestibular rehabilitation, visual vertigo* (JNPT 2015;39: 215–224)

INTRODUCTION

Visual vertigo (VV), or visually induced dizziness, is a term used to describe symptoms of dizziness, disorientation, and/or unsteadiness in situations involving visual-vestibular conflict or intense visual motion (eg, walking down supermarket aisles).^{1,2} A subset of people with a vestibular disorder are more susceptible to visual motion than others,^{1,3–5} which is believed to be due to an overreliance on visual cues for both perception and postural responses (ie, visually dependent).⁶ Perceptual preferences for spatial orientation vary even within a healthy population, with certain individuals relying more on vision and others on vestibule-proprioceptive cues.⁷ In disease, VV may be a trait that is enhanced or acquired (perhaps as a consequence of compromised function in other sensory systems), whereby perceptual preferences may develop and become inappropriate compensatory strategies for balance.⁸ Binocular vision abnormalities have been associated with increased postural responses to optokinetic stimuli in people with vestibular dysfunction.¹ Recently, a greater incidence of fixation disparity (a small misalignment of the eyes when viewing with binocular vision) and reduced stereopsis (binocular depth perception) has been identified in people with vestibular dysfunction compared with healthy controls. However, only individuals with additional VV symptoms also had fusional vergence dysfunction (inability to effectively use and/or sustain binocular vision because of disjunctive eye movements in which the visual axes move toward or away from each other) and experienced greater difficulty with visual fixation in the

Centre of Human and Aerospace Physiology (M.P.), School of Biomedical Sciences, King's College London, London, United Kingdom; Department of Neuro-Ophthalmology (J.A., D.N.) and Department of Neuro-Otology (A.M.B., R.A.D.), National Hospital for Neurology and Neurosurgery, Queen Square, London, United Kingdom; Moorfields Eye Hospital NHS Foundation Trust (J.A., D.N.), London, United Kingdom; Save Sight Institute (C.L.F.), University of Sydney, Sydney, Australia; and Neuro-Otology Unit (A.M.B.), Division of Brain Sciences, Imperial College London, London, United Kingdom.

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Correspondence: Marousa Pavlou, PhD, BA, Centre of Human and Aerospace Physiology, School of Biomedical Sciences, King's College London, London, United Kingdom (marousa.pavlou@kcl.ac.uk).

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presence of optokinetic stimuli.⁹ Despite the aforementioned findings, no studies to date have investigated the effect of binocular vision abnormalities on vestibular rehabilitation outcomes, specifically pre-/posttreatment changes in VV symptoms.

Customized vestibular rehabilitation, promoting desensitization and increased tolerance to visual stimuli through optokinetic exposure, has been identified as particularly beneficial for improving VV symptoms in people with a vestibular disorder.^{10–12} It is believed that graded exposure to optokinetic stimulation reduces the abnormal overreliance on visual input for perceptual and postural responses,^{6,13} with recent findings showing that short-term repeated exposure to visuo-vestibular exercises induces adaptive changes, thereby decreasing (improving) the magnitude of visual dependency in healthy controls.¹³ The degree of VV symptom improvement varies among individuals^{11,14} and a subset of patients do not report a pre-/posttreatment change.¹⁴

The primary aim of this study was to compare the presence and severity of VV symptoms at baseline and pre-/posttreatment VV score changes in people with refractory vestibular symptoms with and without a binocular vision abnormality. Secondary aims were to compare baseline and pre-/posttreatment change scores for common vestibular and psychological symptoms, dynamic computerized posturography and the Functional Gait Assessment (FGA)¹⁵ in the same participant cohort, and to assess the relationship between pre-/posttreatment changes for objective and self-report measures, migraine, and the presence of a binocular vision abnormality. The authors hypothesized that the presence of a binocular vision abnormality would be associated with higher baseline scores and less (ie, worse) pre-/posttreatment improvement on all outcome measures, particularly for VV symptoms and that pre-/post-treatment changes for psychological symptom scores will show a positive correlation with those for common vestibular and VV symptoms.

METHODS

Design

This study was a secondary analysis of data from a single-blinded randomized, controlled parallel-group clinical trial with pre-/postcomparisons.¹⁰ The original study investigated the effect of supervised versus unsupervised optokinetic exercises on vestibular rehabilitation treatment outcomes.¹⁰ Subjects were randomly assigned to an 8-week customized vestibular rehabilitation exercise program incorporating optokinetic stimulation training via (a) a full-field visual environment rotator, (b) a video display supervised, or (c) a video display unsupervised. The therapist and subjects were informed of exercise program allocation after completion of the baseline assessment. As no significant differences were noted between the 3 exercise programs for any outcome measures,¹⁰ data from all subjects were combined. Groups for the current study were based on the presence (Group P) or absence (Group A) of a binocular vision abnormality. The flow of subjects from the original study to group allocation in the current study is illustrated in Figure 1.

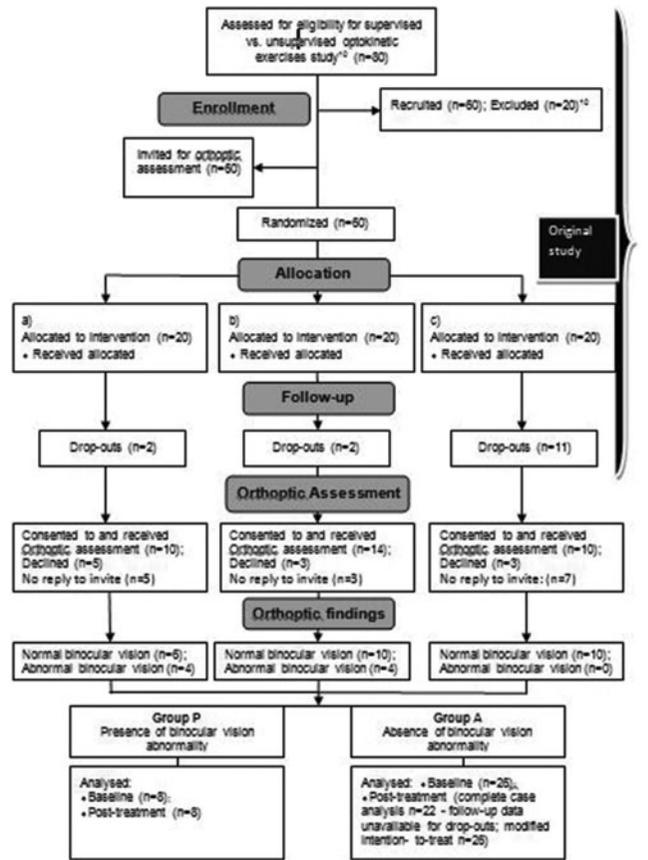


Figure 1. Flow diagram summarizing the flow of subjects from the original study to group allocation in the current study.

Sixty subjects were recruited during a 3-year period from neuro-otology clinics at the National Hospital for Neurology and Neurosurgery (NHNN), Queen Square and Charing Cross Hospital, London, UK. Subjects were recruited after a neurological and neuro-otological examination. Inclusion criteria were (a) clinical diagnosis of a peripheral vestibular disorder; (b) chronic dizziness and/or unsteadiness (symptom history of at least 2 months^{16,17}); (c) 18 to 80 years old; and (d) previous vestibular rehabilitation program completed with partial/no improvement. Subjects with (a) central nervous system involvement excluding migraine; (b) fluctuating symptoms (eg, active Meniere's disease), (c) orthopedic deficit affecting balance and gait, or (d) inability to attend sessions were excluded. Subjects with severe migraine (ie, >3 migrainous headaches per month) or untreated severe depression (ie, Beck Depression Inventory score of >29) were excluded.

The neuro-otological examination included the following assessments:

1. Fitzgerald-Hallpike bithermal caloric stimulation using a 40-second irrigation in each ear at 44°C and 30°C. The British Society of Audiology Recommended Procedure Caloric Test document¹⁸ states that individual departments should obtain their own normative duration or slow-phase velocity data to identify a clinically significant canal paresis.

The threshold for the Neuro-otology Department, NHNN, London, UK, is more than 8% in the absence of optic fixation under direction observation¹⁹ and is lower than that obtained using the slow-phase velocity parameter.²⁰

2. Horizontal direct current electronystagmography (objective recording of eye movements using Easygraph recorder, Gould Instrument Systems, Ilford, Essex, UK) of gaze ($\pm 30^\circ$) with/without optic fixation, saccades (at 0° and $\pm 30^\circ$, assessing for velocity and accuracy), smooth pursuit at 0.2, 0.3, and 0.4 Hz (with peak velocities of 38, 56.5, and 76°/s, respectively, assessing for saccadic intrusions), optokinetic responses to a full-field striped curtain rotated at 40°/s (assessing for symmetry). Sinusoidal rotation at 0.2 Hz with/without fixation and impulsive rotation (until nystagmus subsided, approximately 45 seconds, maximum 100 seconds) with an initial 140°/s acceleration/deceleration and a 60°/s fixed-chair velocity tested the vestibulo-ocular reflex, including vestibulo-ocular reflex suppression. The latter was considered normal when no measurable nystagmus was recorded during visual fixation.
3. Dix-Hallpike test.²¹

Subject diagnosis was based on clinical history and/or neuro-otological findings, according to published normal data and limits;²² migraine was diagnosed on the basis of the International Headache Society criteria for migraine²³ and Neuhauser criteria^{24,25} for vestibular migraine. Diagnoses, vestibular findings, and presence of migraine headache for each group are listed in Table 1. In accordance with previous findings, 50% of subjects with chronic peripheral vestibular disorders²⁶ or vestibular migraine²⁷ had normal test results. Three subjects were diagnosed with motorist disorientation syndrome,²⁸ a symptom descriptor referring to symptoms of dizziness, disorientation, nausea, and/or unsteadiness together with an illusion that the car is veering off course when driving a car on open roads (ie, a highway) particularly when going around a curvy bend, over the brow of or descending a hill and at faster speeds (>40 mph miles per hour). Subjects were diagnosed by physicians with advanced expertise in neuro-otology.

Measurements

Orthoptic Assessment

All subjects were seen for orthoptic screening in the Department of Neuro-Ophthalmology, NHNN, by the senior orthoptist. The binocular vision assessment included a history of childhood-onset squint, amblyopia (decreased vision in one or both eyes because of abnormal development of vision in infancy or childhood and treatment for amblyopia), other visual and ocular disorders, and documentation of current use of corrective lenses. The objective assessment investigated motor and sensory binocular visual function. Specific tests included corrected monocular visual acuity^{29–31} at 0.33 and 6 m; color vision, focimetry (use of a focimeter or lensmeter instrument to verify the correct prescription of a pair of eyeglasses), cover test at 0.33 and 6 m;^{32–34} near point of convergence;^{35–37} prism fusion range;^{38–40} Frisby stereotest;^{41–43} and ocular motility.^{44,45} Please refer to Table 2 for specific information relevant to each test.

Table 1. Participant Characteristics^a

Variable	Group A ^a (n = 26)	Group B ^b (n = 8)
Age, y, mean (range)	49.04 (29-73)	53.88 (43-70)
Sex		
Female, n (%)	19 (73)	6 (75)
Male, n (%)	7 (27)	2 (25)
Symptom duration, mo, mean (range)	89.1 (8-600)	73.5 (24-156)
Presence of migraine, n (%)	14 (54)	4 (50)
Diagnosis, n		
VN	7	2
VM	11	3
BVH (+M) ^c	3 (1)	1
BPPV	0	1
Post-traumatic dizziness (+M) ^c	0	0 (1)
Acoustic neuroma (+M) ^c	0 (1)	0
MDS ²⁵ (+M) ^c	1 (2)	0
Vestibular findings, n		
CP	7	3
Bilateral CP	4	1
BPPV	0	1
No abnormal findings	15	3

Abbreviations: BPPV, benign paroxysmal positional vertigo; BVH, idiopathic bilateral vestibular hypofunction; CP, canal paresis; M, meets IHS diagnostic criteria for migraine; MDS, motorist disorientation syndrome; VM, vestibular migraine according to Neuhauser criteria; VN, idiopathic peripheral vestibular disorder, compatible with a history of past vestibular neuritis.

^aParticipants with refractory vestibular symptoms but no binocular vision abnormality.

^bParticipants with refractory vestibular symptoms plus the presence of a binocular vision abnormality.

^cThe inclusion of (+M) indicates a history of migraine headache in addition to the neuro-otological diagnosis. The number included next to each diagnosis refers to the number of individuals who had that diagnosis but no migraine history in the first column and those with migraine history in the parentheses.

If a subject was found to have signs of a binocular vision abnormality, this was classified further with ocular motility examination. A binocular vision abnormality included reduced stereopsis (binocular depth perception) on the Frisby stereoacuity test, double vision at near fixation, or an abnormal head posture^{46,47} with a reduced field of binocular single vision.

Vestibular Rehabilitation Outcome Measures

Subjective symptoms, balance, and gait were assessed at baseline and at 8 weeks (end of treatment). The primary outcome measure was the Situational Characteristics Questionnaire (SCQ),^{3,6} a 19-item questionnaire that yields a normalized score (sum/19-number “not tried”) between 0 (never) to 4 (always) measuring frequency of symptom provocation or exacerbation in environments with visual-vestibular conflict or intense visual motion (eg, walking down a supermarket aisle and watching moving television scenes). Scores of 0.7/4 or more were used to identify the presence of VV symptoms.⁵ The SCQ has been found to corroborate an initial clinical diagnosis of VV and quantify its severity in subjects with vestibular dysfunction.⁵ All subjects also completed 2 validated questionnaires, the Vertigo Symptom Scale,⁴⁸ and the Becks Anxiety⁴⁹ and Depression Inventory.⁵⁰ Balance and gait measures included computerized dynamic posturography⁵¹ and the FGA.¹⁵

Table 2. Details of Orthoptic Tests Performed

Orthoptic Test	What Does It Assess?	Brief Description	Possible Findings	Reliability and Validity
Monocular visual acuity	Visual acuity	Subject is asked to read the smallest possible letters on a standardized chart for a near target (reading distance 0.33 m; success vision test) and a distance target (6 m; Snellen test) Tested with spectacles on, one eye at a time	Amblyopia should be considered if best corrected visual acuity <6/9 in one eye; often coexists with strabismus and microtropia (small-angle unilateral strabismus)	The success vision test assesses near visual acuity to levels as low as N4. Snellen test: high test-retest reliability, similar to that for the logMar chart, which is considered the gold standard; ^{26,27} sensitive to most common sources of visual impairment but limited reliability when visual acuity is less than 6/24 m (20/80 ft). ²⁸ As all our subjects had visual acuity of 6/18 m (20/60 ft) or better, the reliability of this test was considered adequate for the purpose of the study.
Cover test (including alternate cover and cover-uncover test) ³¹	The presence and degree of strabismus or ocular misalignment	A cover is placed over one eye then removed while observing both eyes for movement. It is repeated on both eyes for a near (0.33 m) and distant target (6m).	In amblyopia, the eye will deviate inwards or outwards. A constant, intermittent, or manifest ocular misalignment can be described to aid diagnosis.	With experienced examiners, both the cover-uncover and alternate cover tests are reliable methods for assessing ocular alignment with high interexaminer and intraexaminer repeatability. ^{29,30}
Ocular motility	All extraocular muscles to be examined in both their primary and secondary positions of action.	Subjects look at a torchlight in 9 specific gaze positions while the cover and cover-uncover tests are performed.	Any extraocular muscle abnormality can be detected, described, and quantified using an orthoptic grading scale. During this test, the examiner may observe discomfort or pain on movements (common in mechanical strabismus) or any symmetrical limitations of movement. Superior oblique palsy (SOP) is the most common isolated cranial nerve palsy. ⁴¹	Reported as the simplest objective method of determining ocular alignment. ⁴²
Near point of convergence	The ability to maintain fixation on a target as it approaches the nose.	Determined by measuring the point at which the eyes can no longer maintain binocular fusion on a target as it is brought toward the face.	In a near point of convergence >10 cm from the nose, symptoms such as diplopia are common when reading.	Reliable and diagnostic in an adult population. ^{32,33} A receded near point of convergence is an important criterion for diagnosis of convergence insufficiency. ³⁴
Compensatory or abnormal head posture ⁴³	Abnormal head posture as a compensatory mechanism to eliminate double vision	The abnormal head posture is determined on the basis of observation by an experienced ophthalmic assessor.	Head tilt and face turned toward the less affected eye is observed in superior oblique palsy. (In all cases a hypertropia consistent with a congenital superior oblique palsy was found in the current study).	The most common ocular cause of abnormal head posture is superior oblique palsy. ⁴⁴ Assessment can be biased by the assessor's training, experience and the spectrum of patients seen. ⁴³
Prism fusion range	The ability to maintain binocular single vision during vergence movement simulated by prisms	The prism fusion range is tested by placing either horizontal or vertically aligned prisms in front of either eye, to determine whether binocular single vision can be maintained.	Normal values have been reported for adults. ³⁵ A large prism fusion range is consistent with a long standing ocular misalignment.	This is the only test for measuring motor fusion in free space that the authors are aware of. A vertical fusion amplitude range of >10Δ supports diagnosis of a congenital superior oblique palsy. ^{36,37}
Frisby stereoaucuity test ³⁹	Assesses a subject's ability to use binocular vision to achieve stereopsis (3D vision).	An actual depth stereotest. Disparity is caused by printing a circle of coarse texture elements from 1 of the 4 squares on the opposite side of a perspex plate. The subject is asked to pick the square containing the actual depth object.	Reduced stereopsis provides information regarding vision development and is absent in patients with long-standing strabismus.	Tests stereoaucuity levels of 600 to 15 arc sec, is identified as being clinically useful, ³⁸ and has best intraexaminer repeatability compared with other stereoaucuity tests. ⁴⁰ It enables test-retest reliability as the correct response cannot be learned.

Intervention

Full details of the optokinetic equipment and exercise program may be found in Pavlou et al.¹⁰ In brief, the video recording comprises 13 two-minute sessions of an optokinetic disc or drum rotating, at constant velocities or sinusoidally, at peak velocities of 40°/s or 60°/s. Exercises were divided into a progressive sequence (beginners, intermediate, and advanced level) with exercises in sitting, standing, and walking with or without additional vertical or horizontal head movements. Local ethics committee approval was obtained from the NHNN and Institute of Neurology Joint Ethics Committee. All subjects provided written informed consent to participate in the study.

Statistical Analysis

IBM SPSS Statistics 20 (IBM Corporation, New York) was used for statistical analysis. Data are presented as mean ± standard deviation (SD). Between-group differences were determined using Mann-Whitney *U* tests; the nonparametric equation $r = Z/\sqrt{N}$, where *N* is the total number of samples, was used as the measure of effect size.⁵² Within-group differences between pre- (baseline) and post-(week 8) intervention data were analyzed using Wilcoxon signed rank tests. Results for pre-/posttreatment changes are presented for both a complete case (only data for subjects who completed both the orthoptic assessment and vestibular rehabilitation program are included) and a modified intention-to-treat analysis (baseline measures forwarded as final values for the 4 subjects in Group A who did not complete the vestibular rehabilitation program). Covariate and fixed-factor effects were assessed. Preliminary screening with Spearman bivariate correlations was performed to enable the selection of covariates (age, symptom duration, and migraine) and a fixed factor (sex) to be tested with analysis of covariance models of ranked data; results will only be reported if significant. Spearman correlation assessed the relationship between the presence of a binocular vision abnormality and migraine as well as pretreatment and posttreatment changes for objective and self-report measures.

Table 3. Orthoptic Examination Details on Patients Found to Have Binocular Vision Abnormalities

Ocular Motility Diagnosis	Objective Binocular Vision Abnormality
Congenital superior oblique palsy (n = 3)	Cover test: hypertropia Compensatory abnormal head posture to eliminate diplopia Reduced stereopsis in primary position
Convergence insufficiency (n = 2)	Near exophoria with intermittent diplopia at near fixation Reduced near point of convergence Reduced stereopsis
Microtropia (n = 3)	Esotropia with reduced motor fusion (n = 2) Esotropia since childhood with reduced stereopsis (n = 1)

RESULTS

Of the 60 subjects referred to the orthoptic department, 57% (34 of the 60; Figure 1) completed the assessment. Eleven subjects declined the orthoptic assessment (symptoms had improved and subjects considered further assessment unnecessary [*n* = 8]; unable to attend appointment because of childcare or work commitments [*n* = 3]) and 15 subjects, including 11 who had withdrawn from the original study, did not reply to the invitation.

Objective binocular vision abnormalities were found in 8 of the 34 (23.5%; Figure 1) subjects, who were further classified as to which causative ocular motility abnormality was present (Table 3). Statistical analysis of baseline data for all subjects (*n* = 34) showed no significant differences between those with (*n* = 8) versus those without (*n* = 26) a binocular vision abnormality (Table 4) and no significant between-group differences were noted for age, sex, or symptom duration (Table 1). Thirty of the 34 subjects who had the orthoptic assessment completed the vestibular rehabilitation program, including the 8 subjects found to have abnormal binocular visual function (Figure 1). A significant between-group difference in baseline data was noted *only* for depression symptom scores

Table 4. Mean (Standard Deviation) of Outcome Measures

Measure	Group A ^a		Group B ^b	
	Baseline n = 26	Posttreatment n = 22	Baseline n = 8	Posttreatment n = 8
SCQ	2.2 (1.0)	1.4 (0.9) ^c	2.5 (1.1)	2.2 (1.1) ^d
VSS-V	1.0 (0.7)	0.6 (0.5) ^c	1.5 (1.0)	1.2 (1.0) ^e
VSS-A	1.3 (0.7)	0.9 (0.6) ^c	1.6 (0.8)	1.3 (0.8)
BDI	10.7 (6.8)	6.2 (4.0) ^c	9.4 (6.7)	7.1 (4.7)
BAI	17.2 (9.3)	10.8 (6.9) ^c	18.4 (9.5)	15.8 (7.2)
FGA	19.0 (5.9)	25.1 (5.4) ^c	17.5 (7.7)	25.1 (5.4) ^c
Posturography	51.7 (21.8)	64.6 (16.2) ^c	47.8 (24.1)	68.4 (11.4) ^c

Abbreviations: BAI, Beck's Anxiety Inventory; BDI, Beck's Depression Inventory; FGA, Functional Gait Assessment; SCQ, Situational Characteristics Questionnaire; VSS-A, Vertigo Symptom Scale (autonomic and somatic anxiety symptoms); VSS-V, Vertigo Symptom Scale (global vertigo symptoms).

^aParticipants with refractory vestibular symptoms but no binocular vision abnormality.

^bParticipants with refractory vestibular symptoms plus the presence of a binocular vision abnormality.

^c*P* ≤ 0.01 indicates a significant within-group improvement compared with baseline.

^d*P* = 0.01 indicates a significant between-group difference.

^e*P* ≤ 0.05.

($U = 17.5$, $Z = -2.28$, $P = 0.02$), with higher (ie, worse) mean scores in individuals who did not complete the vestibular rehabilitation program (mean = 17.25; SD, 5.56) compared with those who did complete the vestibular rehabilitation program (mean = 9.47; SD, 6.63).

Questionnaires

A significant between-group difference for the SCQ symptom score change ($U = 33.0$, $Z = -2.58$, $P = 0.01$, $r = 0.47$; Figure 2) was noted, with a significant 39% improvement for Group A ($Z = -4.47$, $P < 0.0005$; 91% of subjects improved, 9% showed no change) compared with a nonsignificant 12% (75% improved, 25% increased, ie, worse symptoms posttreatment) change for Group P. A modified intention-to-treat analysis of the between-group difference for the SCQ symptom score change approached significance ($U = 57.0$, $Z = -1.92$, $P = 0.056$, $r = 0.33$).

No significant between-group differences were noted with either complete case or modified intention-to-treat analysis for vestibular (Vertigo Symptom Scale [VSS-V]), autonomic/somatic anxiety (VSS-A), or Beck's Anxiety and Depression Scale scores. Both groups showed significant within-group improvements for the VSS-V (Group A: $Z = -3.75$, $P < 0.0005$, 73% of subjects improved; Group P: $Z = -2.21$, $P = 0.027$, 75%). However, only Group A showed significant within-group improvements for VSS-A ($Z = -3.34$, $P = 0.001$, 77% of subjects improved), depression ($Z = -3.72$, $P < 0.005$, 73%), and anxiety scores ($Z = -2.60$, $P = 0.009$, 64%). Modified intention-to-treat analysis showed similar within-group findings for Group A: SCQ ($Z = -4.01$, $P < 0.0005$), VSS-V ($Z = -3.14$, $P = 0.002$), VSS-A ($Z = -2.89$, $P = 0.004$), depression ($Z = -3.42$, $P = 0.001$), and anxiety scores ($Z = -2.63$, $P = 0.009$). Descriptive data and statistics are displayed in Table 4.

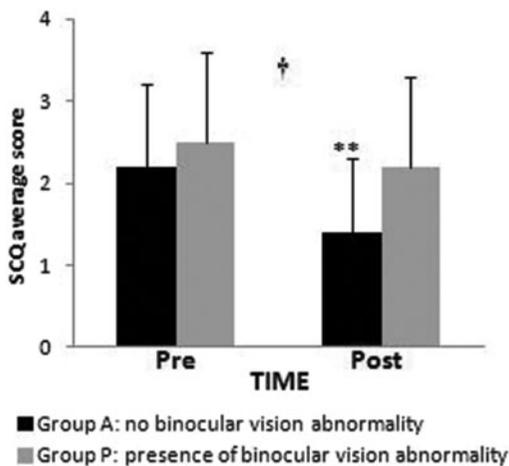


Figure 2. Mean and standard deviation of SCQ scores for assessment of visual vertigo symptoms pre- and posttreatment for subjects with peripheral vestibular dysfunction without and with a binocular vision abnormality. SCQ, Situational Characteristic Questionnaire. †A significant difference between groups ($P = 0.01$); **a significant within-group improvement ($P < 0.01$).

Baseline Beck's Anxiety and Depression Scale scores indicated mild depression for 11 subjects in Group A and 2 subjects in Group P. Scores indicative of moderate depression were reported by 5 subjects in Group A and 1 subject in Group P. Individual scores for subjects in Groups A and P, respectively, denoted (a) mild anxiety for 14 and 1 subjects, (b) moderate for 6 and 2 subjects, and (c) severe for 4 and 3 subjects.

When collapsing all subjects' scores independent of group, depression score improvements correlated with VSS-V improvement ($r = 0.38$, $P < 0.05$). SCQ improvements significantly correlated with the presence of a binocular vision abnormality ($r = -0.78$, $P < 0.01$), wherein those with binocular vision abnormalities improved less.

Balance and Gait Measures

Posturography

Baseline posturography scores were abnormal (ie, composite score $<70/100\%$)⁵¹ for 79.4% of subjects (Group A, $n = 20$; Group P, $n = 7$). Within-group improvements were noted for both Groups A (complete case: $Z = -5.09$, $P < 0.0005$; modified intention-to-treat: $Z = -2.71$, $P < 0.01$) and P ($Z = -2.53$, $P = 0.01$) (Table 4). For Group A, 81% of subjects improved, and for Group P, 100% of subjects improved. No significant between-group differences were noted either with complete case or intention-to-treat analysis (Table 4). Subjects with bilateral vestibular hypofunction were unable to maintain balance in conditions 5 (eyes closed, sway referenced surface) and 6 (eyes open, sway referenced surface, and visual surround) where vestibular cues play a major role; they did however show improvements and were included in the analysis because of their removal did not significantly alter findings.

Functional Gait Assessment

A significant within-group improvement in the FGA was observed in both Groups A (complete case: $Z = -4.31$, $P < 0.0005$; modified intention-to-treat: $Z = -3.54$, $P < 0.01$) and P ($Z = -2.52$, $P = 0.01$) (Table 4). No significant between-group differences were noted either with complete case or intention-to-treat analysis (Table 4). All Group P subjects and 91% of Group A subjects improved from baseline.

DISCUSSION

This study compared the effect of binocular vision abnormalities on vestibular rehabilitation outcomes, with changes in pre-/posttreatment VV symptom scores as the primary outcome measure of interest. Baseline VV scores did not differ significantly between subjects with and without a binocular vision abnormality; however, pre-/post-VV scores improved significantly only for the latter group. Within-group analysis showed FGA, posturography, and common vestibular symptoms significantly improved for both groups, with no significant between-group differences. Although no significant between-group differences were noted for psychological state and autonomic symptoms, within-group improvements were noted only for those without a binocular vision abnormality. Sex, age, and symptom duration did not affect outcome.

Subjective Symptoms

As in previous work,⁹ current findings indicate an overrepresentation of manifest deviations, other abnormalities of ocular ductions or binocular function in our small cohort of subjects with a vestibular disorder compared with the general population.^{53,54} Binocular dysfunction did not influence the presence and/or severity of common vestibular (ie, lightheadedness, giddiness, and feeling of unsteadiness) or VV symptoms at baseline, with both groups reporting similar scores. However, the presence of a binocular vision abnormality did have an impact on pre-/post-VV symptom changes with significant improvements noted only for Group A, who reported a 69% greater improvement compared with Group P. Although no information is available regarding clinically meaningful change in SCQ scores, the authors believe that a 39% decrease (ie, improvement) in scores, as noted in Group A, indicates a clinically significant change. A modified intention-to-treat analysis, which accounts for subject dropout and thus provides a more realistic and often less biased estimate of the average treatment effects,⁵⁵ also approached significance for the between-group difference of pre-/post-VV symptom changes, despite the small sample size.

It is hypothesized that VV symptom improvements are based on neural adaptability and a decrease in the overreliance on visual input for perceptual and postural responses.¹⁰ Optokinetic stimulation induces adaptation of specific vestibular parameters, including postrotational vestibular sensation and vestibular ocular reflex gain in primates, people with chronic peripheral vestibular disorders, and healthy individuals.^{56–59} Significant improvements have also been noted in optokinetic nystagmus,⁶⁰ and/or postural stability,^{60,61} after treatment with optokinetic stimulation in people with peripheral vestibular disorders or mal de débarquement syndrome. Treatment with graded exposure to optokinetic stimuli aims to habituate and desensitize to visual motion and promote a more effective use of vestibulo-proprioceptive cues through sensory reweighting.⁸ The underlying mechanism is likely to relate to motion-induced changes in neuronal excitability in visual motion cortical areas (V5/MT).^{62–64}

Binocular vision abnormalities affect the ability to process 3-way sensory information. Studies have indicated that the visuo-postural loop is likely influenced by ocular proprioceptive function.^{65,66} Bronstein^{1,67} suggested that ocular proprioceptive signals are unreliable in the presence of an ocular misalignment, and thus are likely to be disregarded by the cortical areas subserving postural control. This loss of the regulatory control of eye proprioceptive signals on visuo-postural responses would adversely affect sensory reweighting, further decreasing the ability of persons with a binocular vision abnormality to suppress subjective symptomatic and postural responses to intense visual motion or visual-vestibular conflict stimuli.^{1,67} We hypothesize that the lack of VV symptom improvement noted in people with a vestibular disorder and a binocular vision abnormality may be due to ineffective sensory reweighting processes, whereby subjects with binocular vision abnormalities may experience greater difficulty in reducing the relative influence of vision upon balance.

Migraineurs experience increased symptoms during optokinetic exposure⁶⁸ and show increased visual cortical

excitability, which may be correlated with an upregulation of visual sensitivity.⁶⁹ A slightly higher incidence of subtle binocular vision abnormalities, including impaired stereopsis (binocular depth perception), heterophoria (a tendency of the eyes to deviate from the parallel), and higher degrees of astigmatism (an optical defect that occurs when the curvature of the cornea or lens is not perfectly round, causing blurred or distorted vision), has also been noted in migraineurs compared with healthy subjects.⁷⁰ These findings might suggest that the worst symptom levels and least improvement would be noted in people with migraine, a binocular vision abnormality, and vestibular disorder, as all 3 conditions may independently modulate visual sensitivity.^{9,69,71} Our study identified no relationship between migraine and visual function, baseline and posttreatment VV symptoms, or pre-/posttreatment VV symptom changes. The lack of association between these factors in the current study may be due to the small sample size. In Pavlou et al,¹⁰ a higher percentage of migraineurs reported VV improvements that were significantly greater compared with those for nonmigraineurs. Previous authors suggest that medication may help control VV symptoms in migraineurs, enabling better exercise tolerance.⁷² All subjects with more than 3 migraines per month had been treated with prophylactic medication before commencing vestibular rehabilitation involving exposure to optokinetic stimulation. Medication was not controlled for, but no subjects changed medication during the study, and therefore, its role in VV improvements could not be clarified.

Postural Stability and Gait

Binocular dysfunction may contribute to abnormal postural control either due to an incorrect sense of direction or altered ocular proprioceptive signals.^{1,73} Bronstein¹ noted binocular abnormalities including diplopia (double vision), strabismus (abnormal eye alignment), and ocular motor weakness in 4 of the 5 patients who, in addition to VV, showed increased postural sway in response to visual motion stimuli. Thus, although strabismic pathology is not necessary to develop VV, it seems to contribute to the additional visuo-postural reactions that some people with VV have. In the current study, baseline VV symptoms were similar for both groups and abnormal binocularity did not impact on either baseline or pre-/posttreatment change in posturography or FGA scores. Furthermore, a clinically significant change, which is necessary in determining an intervention's efficacy,⁷⁴ was achieved by both groups for posturography^{75,76} and FGA⁷⁷ scores. However, the visual stimulus employed in the study by Bronstein¹ is more intense than the sway-referenced surround on posturography,⁵ and the lack of any detectable between-group differences for current findings may be due to the nature of the visual stimulus. The sway-referenced visual surround in posturography is specifically designed to provide inaccurate visual cues about the position of the body in space; however, it follows a person's center of gravity sway and thus, when sway is small, the movement of the surround may be insufficient to produce an abnormal reaction and a more dynamic stimulus may be required.⁵

There is a growing interest in the functional impact of binocular dysfunction on various visuomotor tasks including

ambulation. People with binocular dysfunction show impairments in crucial aspects of motor control, including movement speed and/or accuracy.⁷⁸ These individuals adapt a more cautious strategy during obstacle crossing, which may indicate increased fall risk during everyday ambulation.⁷⁹ It is well documented that people with vestibular dysfunction may also experience increased fall risk;^{80,81} in our sample this was indicated by the absence of significant between-group differences in mean baseline FGA scores for both participant groups. Findings therefore suggest that the presence of both refractory vestibular symptoms and a binocular vision abnormality does not have an additive effect on postural and gait responses, which can improve in these individuals.

Psychological State

Previous studies^{10,11} report a significant positive correlation between VV, anxiety, depression, and/or autonomic score improvements, wherein greater VV improvements are associated with a greater reduction in psychological and autonomic symptoms. This association was not noted in the current study, however, which may partly relate to lower participant numbers compared with previous work and to the observation that anxiety symptoms and psychiatric problems often exist before the onset of a vestibular disorder.^{82,83}

Baseline data indicated significantly worse depression symptom scores for those individuals who did not complete the vestibular rehabilitation program compared with those who did. This is not surprising as depression has been identified as a barrier to treatment adherence.⁸² However, Pavlou et al¹⁰ reported that vestibular rehabilitation adherence correlated with treatment group allocation, but not depression scores, wherein those allocated to an unsupervised, home-based exercise program had a significantly higher dropout rate compared with those receiving weekly, supervised sessions. In the current study, three-quarters of the noncompleters had been allocated to an unsupervised treatment group. The authors believe that mild depression scores, as indicated by the Becks Depression Inventory, and allocation to an unsupervised treatment group contributed to nonadherence in the current study. However, findings must be interpreted with caution, as the noncompleter group included only 4 subjects.

Limitations

The primary limitation of this study is the small sample size and limited number of people in the sample with binocular visual abnormalities. Overall, these are preliminary findings and should be interpreted with caution. Larger cohort trials should further investigate the short- and long-term treatment effects and interactions that may improve or inhibit response to intervention and specifically VV symptom improvement.

Clinical Implications

These findings may have important clinical implications for the management of refractory vestibular symptoms and a binocular vision abnormality in persons who experience VV symptoms. It is important for clinicians to be aware of the possible negative effect of this type of binocular abnormality on VV treatment outcome, in order to manage their own and the client's expectations from treatment. Furthermore, a

simple cover test for distant and near objects, as well as the Frisby stereoacuity test (Table 2), would have identified all study subjects with ocular motility abnormalities. These tests are easy to learn and quick to perform with little specialized equipment necessary. Therefore, physical therapists could assess and screen for the presence of binocular abnormalities in this population. However, if an abnormality is found, a formal orthoptic and ophthalmic assessment is recommended.

CONCLUSIONS

Overall, the presence of a binocular vision abnormality in people with refractory vestibular symptoms does not influence treatment efficacy for common vestibular symptoms, postural and gait stability nor does it appear to impact on the presence of VV symptoms. However, it does appear to directly influence VV symptom improvement, with significant improvements noted only for those subjects without a binocular vision abnormality.

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