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Visual-Vestibular Habituation and Balance Training for Motion Sickness

Background and Purpose. This case report describes physical therapy for motion sickness in a 34-year-old woman. The purpose of the report is twofold: (1) to provide an overview of the literature regarding motion sickness syndrome, causal factors, and rationale for treatment and (2) to describe the evaluation and treatment of a patient with motion sickness. **Case Description and Outcomes.** The patient initially had moderate to severe visually induced motion sickness, which affected her functional abilities and prevented her from working. Following 10 weeks of a primarily home-based program of visual-vestibular habituation and balance training, her symptoms were alleviated and she could resume all work-related activities. **Discussion.** Although motion sickness affects nearly one third of all people who travel by land, sea, or air, little documentation exists regarding prevention or management. [Rine RM, Schubert MC, Balkany TJ. Visual-vestibular habituation and balance training for motion sickness. *Phys Ther.* 1999;79:949–957.]

Key Words: *Habituation, Motion sickness, Physical therapy, Sensory conflict theory.*

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Descriptions of motion sickness date back to Hippocrates, who noted that "sailing on the sea shows that motion disorders the body" (Hippocrates, *The Nature of Man*). Investigations that have examined the symptoms, predictors, and causes of motion sickness and the underlying mechanisms involved in motion sickness have revealed that a conflict of visual and vestibular information, as it relates to postural control and visual stabilization, is a critical factor.¹⁻⁸ Despite these reports and recent interest in postural control and clinical intervention for individuals with dizziness or vertigo,^{5,7,8} little information exists about evaluation or effective treatment to ameliorate the symptoms of motion sickness, except as it relates to astronauts and pilots.^{3,6,9-13} This dearth of information may be due, in part, to a lack of evidence of vestibular deficit in people with motion sickness, as well as a limited operational definition of motion sickness. The restrictive definition (eg, onset of vomiting, nausea) and lack of clear diagnostic testing may result in false negative identification and an underestimation of the incidence of motion sickness.^{14(pp38-81),15,16} Additionally, most individuals can avoid circumstances that cause them motion sickness. For those individuals who cannot avoid these circumstances, however, the problem is of major consequence. If current theories of motion sickness are correct, then the principles of habituation that have been applied with varying success to reduce or prevent motion sickness in pilots and astronauts⁹⁻¹³ might be applicable to the development of evaluation and treatment methods for individuals with motion sickness that interferes with daily function.

The traditional operational definition of motion sickness has been the onset of vomiting or nausea experienced by the land, air, sea, or space traveler that results in impaired function.^{1,4,7,14(pp38-81)} Nearly 60% of astronauts report experiencing motion sickness,¹³ as do approximately 30% of ocean liner passengers¹⁵ and nearly 40% of flight trainees in the Royal Air Force.¹⁰ Because impaired function when piloting a plane or ship is not only debilitating but also potentially dangerous,

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interest in motion sickness has been a focus primarily of the military and aeronautical industries.^{13,15} Motion sickness can be induced, however, by either physical motion or stimuli that result in perceived motion (optokinetic stimuli), such as computer displays.^{6,8,9,15} Therefore, employees of airline and cruise ship companies, as well as those who work at computer displays or in other visually provocative situations, are exposed to conditions known to induce motion sickness. The limited

operational definition of motion sickness may preclude identification of the problem. In an attempt to provide a comprehensive, reliable, and less restrictive operational definition, scales were developed to be completed by people exposed to stimuli known to provoke complaints of motion sickness.^{15,16} Using this type of scale, investigators^{6,7,13,14(pp174-209),17,18} reported that symptoms and precipitating factors of motion sickness vary among individuals. Manifestations of motion sickness may include visual and postural instability, pallor, diaphoresis, excess salivation, headaches and anxiety, and nausea and vomiting.^{4,6,7,10,14(pp38-81),17} Precipitating environmental conditions include vertical- or frontal-axis movements (up-down or roll movements, respectively), movement in the anterior-posterior direction (pitch movements), rotational (yaw) movements, and optokinetic stimuli.^{4,6-8,13,18} In spite of these variations, most investigators agree that it is not solely the movement or movement stimulus that results in motion sickness, but rather a conflict in movement information detected by different sensory modalities.

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The sensory conflict hypothesis implies that the symptoms of motion sickness result from incongruent sensory inputs regarding orientation and movement.^{4,6,16,17,18} To test this hypothesis, investigators have developed paradigms in which they induce motion sickness via the manipulation of movement or via the manipulation of the visual or vestibular stimuli that affect the perception of movement. To date, most research has focused on acceleration, weightlessness, and increased gravitational force as causal factors of motion sickness and on training to achieve habituation and thus minimize the effects of these factors.^{15,16} Although most investigators agree that it is primarily an incongruence of visual and vestibular sensory information regarding movement and orientation that results in motion sickness,^{4,6,16} incongruence between canalithic and otolithic vestibular input has been implicated as the provocative stimulus in seasickness and in the onset of motion sickness associated with weightlessness.⁷ Visual stimuli have been shown to be most provocative of motion sickness symptoms.^{4,8} Other factors, however, have been identified that confound these findings. The potency of the provocative stimulus is dependent on head position and the demands placed on the postural control system,^{6,17} and instability has been attributed to a disruption of the activation of neuromuscular responses for postural control.^{6,20,21}

Daunton and Fox⁴ examined the contributions of the various sensory modalities (ie, visual, vestibular) to motion sickness and found that although moving visual stimuli were most provocative at slower speeds (ie, 60°/s), vestibular stimuli (eg, movements of the head or entire body) were most provocative at higher speeds (ie, 150°/s), which more closely resembles the demands during activities of daily living. Furthermore, combined incongruent visual-vestibular stimuli (eg, one stimulus indicating movement and the other stimulus not indicating movement) were more provocative (ie, symptom onset sooner and more severe) than either in isolation, or if combined and complementary (ie, both indicate movement).⁴ Fox et al²⁰ demonstrated that the effect of visual-vestibular conflicts was dependent on simultaneous demands or requirements placed on the postural control system. Subjects experienced motion sickness during visual-vestibular conflicts only if they were required to maintain posture, as opposed to being restrained or supported. Although Eyeson-Annan et al⁸ reported that visual stimuli were more provocative of motion sickness symptoms than either vestibular stimuli alone or a combination of visual and vestibular stimuli, all experiments were performed at slow speed only (ie, 60°/s), subjects were seated and fully supported, and the combined incongruent condition was not examined.

Lackner and Graybiel⁶ investigated the effects of the direction of head movement (eg, yaw, roll, pitch) and

reported that all movements increased susceptibility to motion sickness. The eyes-open condition was more provocative than the eyes-closed condition, pitch movements were most stressful, and acceleration and frequency of movement were important factors. Although increased speed led to increased motion sickness, increased frequency of oscillations resulted in a decrease in motion sickness. Lackner and Graybiel concluded that space motion sickness was, at least in part, due to exposure to a novel background force level, not just weightlessness. It is a consequence of being in an acceleration condition that differs from that to which the body's sensorimotor and postural control mechanisms are adapted.

Investigations^{5,17} that indicate the typical postural responses to motion stimuli are altered in people experiencing motion sickness support the deficient adaptation hypothesis. Reschke et al¹⁷ found that the overall gain of the soleus muscle motoneuron pool (eg, Hoffmann reflex or H-reflex) was modulated by statolith stimulation (eg, linear acceleration, static y-axis tilt [pitch plane], brief z-axis vertical drop). Specifically, with statolith stimulation, there was a delay in H-reflex potentiation in response to being tipped. Therefore, the vestibular stimulation modified the centrally activated response. Clement et al²¹ reported that, in the 2 space flight crew members tested, there was altered postural alignment with 13 degrees of forward body lean at the initiation of weightlessness. Adaptation occurred in several days under normal vision conditions. When vision was occluded or restricted, the forward lean was increased by an additional 4 degrees and persisted for the duration of weightlessness. Clement et al concluded that the physiological mechanisms underlying postural control were modified in weightlessness and that vision was critical for the recalibration of vestibular and somatosensory postural cues affected by weightlessness. These conclusions led to the hypothesis that, because adaptation can alter responses to stimuli, the application of stimuli that result in adaptation might be helpful in alleviating the symptoms of motion sickness. Motion sickness is, however, a normal, protective response that alerts the individual to impending trouble with equilibrium.⁷ Individuals who experience motion sickness typically have normal vestibular and visual system function. Thus, to address the condition is not to provide intervention for dysfunction, but to improve functional and adaptive responses.

Studies of animals and humans with motion sickness and interventions to reduce it have indicated that habituation, a reduction or modification in response to the provoking stimulus, can be achieved with repetitive visual and vestibular stimuli.^{2-4,9-11,22-23} Furthermore, although research on habituation training has focused

on the use of visual or vestibular stimuli, the results support the concept that habituation is stimulus-specific.^{4,6,22} Evidence exists that with vestibular stimulation, either caloric or via movement in the dark, there is habituation of the nystagmus response, as well as the perception of movement.¹⁴(pp174–209) The habituation is most dramatic if visual stimulation is also used. Tomura et al²² examined the effect of optokinetic training on nystagmus, spinal reflexes, and vertigo. Following 7 weeks of training, subjects had a decrease in stepping deviation and increased tolerance for optokinetic vertigo, and thus adaptation to optokinetic stimulation. Miles and Braitman²⁴ examined activity in cranial nerve VIII and reported that the changes are not due to adaptation at the peripheral level, but rather to habituation that involves central nervous system changes. In spite of these reports of successful habituation, we could find only one report of clinical application.

Gillilan and Todd²⁵ described a person for whom visual training was used to ameliorate the symptoms of motion sickness, which was visually induced by gazing at a computer terminal. The patient was a 33-year-old woman who complained of dizziness, headaches, and nausea when working at her computer and of mild car sickness, which became severe if she attempted to read. She did eye motility exercises, which included visually tracking a ball in all directions, 30 minutes daily for 17 days. Initially, she performed the exercises with her head stationary, but the exercises were later modified to include rotation and lateral flexion head movements as symptoms decreased. Although the patient reported nausea and eye fatigue during the first week of exercises, these symptoms disappeared by the end of the 3-week treatment. She was able to return to work at the computer and no longer had car sickness. On follow-up 2 years after treatment, she had no motion sickness symptoms. Vestibular test results either were not obtained or were not reported for this patient.

In summary, a conflict between visual and vestibular information regarding spatial orientation has been identified as the primary causal factor for motion sickness, and visual stimuli alone have been shown to induce motion sickness symptoms. Repeated vestibular and visual stimulation activities have been shown to be successful in achieving habituation, with optimal results attained when vision and balance training are provided. These reports led to the hypothesis that patients with motion sickness can be helped by visual-vestibular habituation balance training aimed at the primary cause of the motion sickness. The intervention presented in this case report was developed based on these reports and implemented for a patient with vision-induced motion sickness.

Case Description

Patient

The patient was a 34-year-old marine biologist referred for treatment of motion sickness. During the past 5 years, she experienced 3 severe episodes of vertigo, which lasted several days and were increasingly more severe. Initially, the only symptom was a feeling of light-headedness. Symptoms during the third episode included nausea, vertigo, and limited ability to function, and these symptoms persisted. Her primary care physician referred her to a neurologist (TJB), who diagnosed her as having vertigo and motion sickness and referred her for physical therapy. The medical examination done by the neurologist included rotary chair testing with electronystagmography and posturography, both of which were negative for central and peripheral vestibular deficits or other deficits. She had no other medical problems (eg, hearing loss, migraines) that could lead to motion sickness, and she had no precipitating illnesses or conditions (eg, trauma, inner ear infections) that could contribute to her current problem. The patient took no medications and had normal vision without corrective lenses. She had no family history of vertigo, vestibular dysfunction, or motion sickness.

Initial interview. During the initial interview conducted by the primary author (RMR), the patient reported (1) increasing episodes of visually induced vertigo over the past 5 years; (2) provoking conditions of driving (particularly pulling into and out of parking spaces or driving on on-ramps and off-ramps), riding in an elevator, flickering lights, or observation of any movement; (3) symptoms of feeling “light-headed,” nausea, cold sweating, and headaches; (4) loss of tolerance of air travel due to severe nausea, vomiting, and vertigo; and (5) severely limited ability to work because she could not tolerate standing on a floating dock or ocean diving, both of which were required in her work. When asked whether any activity or treatment minimized the symptoms, the patient reported that she would close her eyes and “get control of the situation,” which was minimally successful within 10 to 15 minutes. Her basic activities of daily living were affected only when she had to sit and wait for her motion sickness symptoms to subside. Rising from or turning in bed did not provoke symptoms. She had no evidence of panic or anxiety disorder.

Physical examination. A physical examination was performed by a physical therapist (RMR). The patient was a normocephalic woman of typical stature, height, weight, and general conditioning. Tests of balance, coordination, vision, vestibular system status, posturography, and general functional ability were completed (Table) to examine the patient’s vestibular and visual system func-

Table.
Evaluation and Results

Test Category and Item	Initial Results	Results 10 Weeks After Treatment
Balance		
Single-leg stance, eyes open	15 seconds, no difficulty	Same as initial
Single-leg stance, eyes closed	15 seconds, no difficulty	Same as initial
Tandem stance	15 seconds, no difficulty	Same as initial
Tandem walk (3 m [10 ft])	No difficulty, no side step	Same as initial
Walk on 8.9-cm [3.5-in] balance beam (1.8 m [6 ft])	Normal; no step off	Same as initial
Stand on dense foam mat, walk 1.8 m (6 ft)	Unable; side stepping (with head movement replicated motion sickness on dock or boat or in water)	Independent; no side step or symptoms ^a
Visual-vestibular		
Vision test (Snellen chart)	20/20	20/20
With head movement (2 Hz)	20/80; sitting; symptoms last 2 minutes	20/20
Vision stability; 1.3-cm (0.5-in) letters on index card at arm's length:		
Card moved side to side	Symptoms within 30 seconds, lasting 10 minutes; 0.5-Hz movement	Tolerated movement at 2 Hz, no symptoms, vision stable ^a
Card moved up and down	Symptoms within 30 seconds, lasting 10 minutes; 0.5-Hz movement	Tolerated movement at 2 Hz, no symptoms, vision stable ^a
Head moved side to side	Symptoms within 30 seconds, lasting 10 minutes; 0.5-Hz movement, but symptoms mild	Tolerated movement at 2 Hz, no symptoms, vision stable ^a
Head moved up and down	Symptoms within 30 seconds, lasting 10 minutes; 0.5-Hz movement, but symptoms mild	Tolerated movement at 2 Hz, no symptoms, vision stable ^a
Fukuda test ^{26,27}	Normal; movement 15.2 cm (6 in) forward, none rotary or sideways	
Hallpike test	Negative	
Posturography	Sensory organization test and dynamic perturbation test in normative range	
Function		
Walk on floating dock	Unable	Done without difficulty ^a
Scuba dive	Unable	Tolerated for 4.0 h ^a

^aFunctional improvement noted.

tion and to quantify her baseline status. Fukuda testing,^{26,27} posturography testing (including both sensory organization and dynamic perturbation testing), heel-to-shin testing, and finger-to-nose testing were negative. To test for positional vertigo, the Hallpike test²⁸ was performed. There was no nystagmus or onset of motion sickness symptoms. These negative results for tests of vestibular function (both vestibulo-ocular and vestibulo-spinal), in conjunction with negative results on rotary chair testing, indicated to the therapist that the peripheral vestibular system was intact. Examination of static and dynamic visual acuity²⁹ revealed visually evoked motion sickness, which was exacerbated with head movement. Specifically, the clinical test of dynamic visual acuity²⁹ revealed that although the patient had 20/20 vision with the head stable (static test), visual acuity was reduced (20/80 vision) on the dynamic component (head moved side to side or up and down), and the patient had to sit during this component. In addition,

when attempting to read 1.27-cm (0.5-in) letters on a card held at arm's length as the card was moved either left to right or up and down at a movement speed of 0.5 Hz, the patient reported moderate symptoms within 30 seconds. She became flushed and had to use her hands to maintain sitting, and she reported vertigo. The examination was stopped for 10 minutes, at which time the patient indicated that the sensations had stopped. Her pallor was normal. No nystagmus was noted. A similar response, but to a lesser degree, was noted when the card was held stable and the head was moved in the same directions. The patient indicated that the symptoms experienced thus far in the examination did not truly replicate those that most severely impaired her function.

To further clarify and identify the provocative stimuli, the patient was asked to repeat the dynamic visual acuity test with somatosensory information compromised. This was done by asking the patient to stand and march on a

dense foam mat, with eyes open, while she turned her head to the left and right and attempted to focus on 2.54-cm (1-in) letters 3 m (10 ft) away. When attempting this, the patient had to side step, required assistance to prevent a fall, and indicated experiencing severe light-headedness and nausea. She became diaphoretic. Her primary impairments included poor balance on unstable surfaces and impaired visual stability with head or object movement. Based on these examination results (ie, no symptoms with head movement alone, negative vestibular tests except for dynamic visual acuity, most severe symptoms experienced in response to observing movement, and replication of symptoms in the clinic achieved primarily with moving visual stimuli while standing on foam), visually evoked motion sickness with somatosensory preference was diagnosed.

Intervention

An exercise regimen was developed by the therapist (RMR) to increase the patient's tolerance to visual stimulation, decrease somatosensory preference and dependence, and improve postural control. This regimen consisted of the use of visual-vestibular habituation exercises and balance training, with a gradual increase in duration, speed, and difficulty of the activities (Appendix). The visual-vestibular exercises progressed from the use and stimulation of one sense (either the card moved and thus vision was challenged, or the head moved and vestibular input was altered) to activities in which both were challenged (both the card and the head moved). This was done to facilitate habituation in a stage-like fashion in increasingly provocative situations. Because the patient's goals included being able to drive and return to her job-related activities, and because dependence on somatosensory cues resulted in an exacerbation of symptoms when these cues were compromised, balance activities on a compliant surface were included. Exercises were reviewed and demonstrated, and provided to the patient in writing.

The patient demonstrated an ability to safely complete stage 1 visual-vestibular exercises 1 through 5 and stage 1 balance exercises 1 and 2. She was instructed to monitor her reactions to the exercises (eg, an increase or decrease in symptoms) and to note them in a daily log. She was to proceed to stage 2 activities as instructed in the handout when she could complete all stage 1 exercises without symptoms. If she experienced any severe reactions, she was told to stop the exercises and contact the therapist. Exercises were to be performed daily. We emphasized the time and duration of the visual-vestibular exercises as opposed to repetitions. This emphasis was important because, in the beginning, the patient could only move her head or the card slowly and the symptoms began after 30 seconds. Because the objective of treatment was to encourage maximal use of

the visual-vestibular systems to facilitate change (implying working the system at its limits), the objective of the activity was to move the head or card as rapidly as possible while maintaining a stable image. The patient was encouraged to increase the time that she did each activity until she could spend the full 90 seconds on each activity without rest. The patient was told to return to the therapy clinic in 2 weeks.

Outcomes

Week 2 follow-up. The patient reported that she was able to progress to completing all activities at stages 1 and 2 of the visual-vestibular component after 10 days and had just proceeded to stage 2 of the balance exercises the day before (stage 2 activities 1 and 2 only). She reported, and the daily log indicated, that she had completed the exercises on 12 of the past 14 days. Furthermore, although completion of the program initially required 45 minutes to 1 hour, she could now complete the activities in 20 to 30 minutes. The patient attributed this improvement to the reduction in time required for symptoms to subside between activities (or no symptoms occurred). In addition, she reported some reduction in car sickness, which she described as not feeling ill as she parked the car or moved out of a parking space. Only the visual stability exercises (stage 1 activity 1) continued to evoke symptoms, but they lasted only 10 seconds. All other activities in stage 1 of both categories of exercises could be completed without symptoms. The exercises were reviewed, and the patient was instructed to continue working at stage 2 of both types of exercise for another 2 weeks. To facilitate recovery and maximize somatosensory preference, we added balance training, which forced the use of visual and vestibular systems and minimized the use of somatosensory information (Appendix). This activity was balancing and walking with the use of foam "boots" (Figure), which were cut out of 8.9-cm (3.5-in) high-density foam and strapped over shoes with Velcro.* We felt that the patient's adherence could be improved because she could complete her daily tasks while "exercising," as this activity could be done as she prepared dinner and performed other daily tasks. All exercises were to be done at least 5 days per week.

Week 4 follow-up. The patient no longer experienced motion sickness while driving, and she was able to tolerate riding in elevators with minimal distress. She experienced vertigo and nausea, but the duration and severity were reduced. Although she could complete all stage 2 activities, she preferred standby assistance when marching on the cushion and turning her head. She said that she never experienced a loss of balance, but she did not feel secure alone. Two days prior to this visit, the patient was able to stand on the floating dock at work for

* Velcro USA Inc, 406 Brown Ave, Manchester, NH 03108.

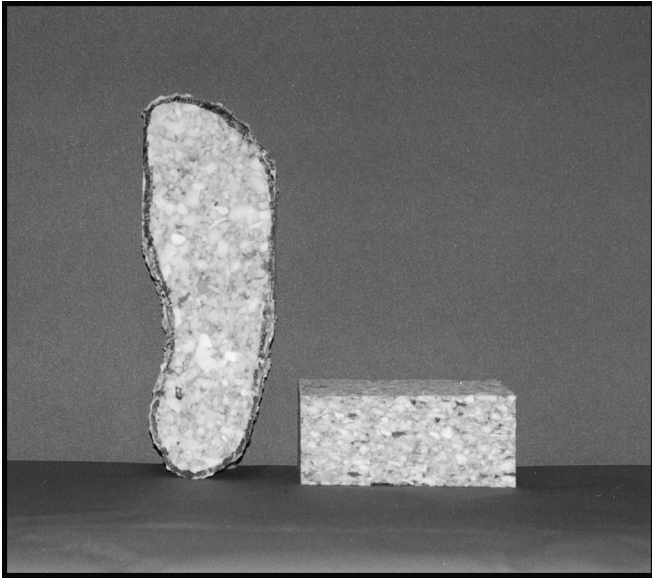


Figure. Dense foam cutout (8.9-cm [3.5-in] thickness) of shoe form used to simulate standing on a floating platform and thus replicate the motion sickness symptoms. The foam was attached to the bottom of the patient's shoes with Velcro straps.

approximately 3 minutes before the onset of motion sickness. She could not yet go out on the boats or dive. She said that, during the past 2 weeks, she completed the activities only 3 days of each week and did not do all activities. This verbal report was validated by log entries. The importance of doing the exercises was explained to the patient, and she was encouraged to perform all activities at least 5 days per week.

Week 7 follow-up. The patient reported that she had been doing her exercise program, and her log indicated that she exercised 15 of the last 20 days. The day before the visit, she was able to stand and work on the floating dock without symptoms, and she had scuba dived for 2 hours before the onset of motion sickness symptoms (nausea, headache, and diaphoresis), which prevented her from continuing. Due to the requirements of the trip, however, she remained on the boat (anchored) for 1 hour before returning to dock. Symptoms persisted for 1 hour after docking. All exercises were performed without difficulty except for mild symptoms during walking with the "boots" and during the visual-vestibular exercises in which the arm and head move in opposite directions (Appendix). She was instructed to continue only with these exercises and to return in 3 weeks.

Week 10 follow-up. The patient could complete all exercises without difficulty, had resumed all work activities, and experienced no dizziness at home or when driving (Table). She experienced only mild motion sickness (mild light-headedness, but no nausea, dizziness, or sweating) after scuba diving for 3 hours, but could

continue if necessary. Once out of the water (but still on the boat), all symptoms subsided within 15 to 20 minutes. The patient was instructed to continue with the exercises twice weekly to maintain her status and was discharged from therapy. Upon telephone follow-up 10 months later, she reported that she had stopped the exercises and was maintaining her ability to function at work and at home.

Discussion

Habituation therapy, which focused on the use of provocative visual and vestibular stimuli and balance training with gradual increase in difficulty, was followed by reduction of symptoms and improvement of function for this patient with debilitating motion sickness. Although she was not completely free of symptoms in the most provocative conditions, her ability to function in these situations was no longer limited and the symptoms were mild. The patient's outcome was similar to outcomes reported by Gillilan and Todd²⁵ and supports the idea that patients with motion sickness can benefit from intervention that is provided in a home exercise format. Furthermore, the case suggests that patient follow-through with the exercise program is important. During the time that the patient reduced the amount of exercise, her progress was minimal. With an increase in the amount of exercise and level of difficulty, improvement increased.

Although this case report cannot explain how improvement was achieved, the treatment was based on the sensory conflict theory and the observation that the effectiveness of habituation is stimuli-specific.^{4,16,17,19,22,24} As predicted by the results of the study by Daunton and Fox,⁴ the most provocative situations for this patient were those in which she could not rely on somatosensory cues and visual and vestibular cues were incongruent. In addition, as suggested by Fox et al,²⁰ the effect of the provocative stimuli was greatest when demands were placed on the patient to balance (eg, standing on dense foam). The work of Tomura et al²² showed that habituation was most effective when training included those activities or situations that most closely resembled the provocative stimuli. This was true for our patient as well. Resumption of activities such as driving and standing on a floating dock occurred after the exercises included the use of the foam "boots." The boots required the patient to be less dependent on somatosensory information and more dependent on visual and vestibular information, the activity that most closely replicated her symptoms. The outcomes are congruent with the systems theory of motor control and approach to rehabilitation.³⁰

Given the multisensory and multisystem demands of the tasks that were difficult for this patient, we believe that appropriate intervention required analysis of each of the sensory system's contribution to and effectiveness in the task. The inability of the system to adapt and function under varying sensory situations resulted in motion

sickness. Our intervention was focused first on the habituation and training for vision and vestibular function, which were the least effective inputs and whose combined conflicting input was provocative of motion sickness symptoms. Gradually, activities were added that forced the use of these information systems in the absence of meaningful somatosensory cues.

Although time alone may have resulted in a reduction of symptoms, this case indicates a gradual and continuous worsening of the symptoms, which did not stabilize or reduce until the exercise regimen began. In addition, because the patient was able to resume activities without a return to the initial level of symptoms after 10 months provides an argument for habituation via central mechanisms as suggested by Miles and Braitman.²⁴ Adaptation at the peripheral level would require continual stimulation to maintain the outcomes. Full support for this theory, however, requires an experimental design.

Although this case cannot be generalized to all individuals with motion sickness, it describes a treatment option for patients with this syndrome, which should be tested for effectiveness. The case also supports the need for further investigation of the mechanisms involved in motion sickness and appropriate interventions.

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Appendix.

Habituation Program

Exercises are to be carried out daily.

Items needed: Kitchen timer, sofa cushions, index card with 0.5-in^a letters (provided by therapist), and an 8- × 11-in sheet of paper with a horizontal line edge to edge on one side, and 2 words printed in 0.75-in letters on the other side.

I. Visual-Vestibular Exercises

Begin at stage 1. Proceed to stage 2 when all activities can be completed with no, or minimal, symptoms.

A. Stage 1

- 1. Seated in chair, hold index card with letters at arm's length in front of you at eye level. Move the card from left to right repeatedly as you maintain fixation on the letters. Identify maximum speed: Move the card slowly, counting in seconds, (one, one thousand) as the card is moved left to right repeatedly. Continue for 10 seconds. If you experience no motion sickness and can maintain a clear image of the letters at this speed, repeat for 10 seconds, moving the card more rapidly. Continue increasing speed until you identify the speed that results in mild symptoms. This is your maximum speed. Continue at maximum speed for 30 seconds. When all symptoms stop, repeat at the maximum level for 30 seconds 4 times. As you repeat this daily, you should attempt to increase your maximum speed level.
- 2. Repeat the same activity, except you are to move the arm and card in the up and down directions, centered in front of you (approximately 8 in up and down from center).
- 3. Seated, repeat step A1, but turn your head from left to right, keeping your arm and card steady and centered in front of you, focusing to keep a clear image of the letters. Establish maximum speed, as above, and continue for 30 seconds. Repeat 4 times.
- 4. Repeat step A3, except move your head in the up-down direction.
- 5. Repeat step A4, except tilt your head side to side (bring right ear toward the right shoulder and then the left ear toward the left shoulder as you visually fixate on the letters on the card, held centered in front of you). Achieve maximum speed as above, and continue for 30 seconds. Repeat 4 times.

B. Stage 2

- 1. Repeat steps A1 through A5 in the standing position.
- 2. Seated, with card held straight out in front as above, move both your head and the card simultaneously from left to right as you fixate on the letters on the card. Establish maximum speed as above, and continue for 30 seconds. Repeat 4 times.
- 3. Repeat step B2, but move the arm and head in the up and down directions.
- 4. Repeat step B2, but move the arm and head in opposite directions (eg, as the arm and card move to the right, your head is turned toward the left, and vice versa).
- 5. As above, move arm and head in opposite directions, but in the up and down directions.

II. Balance Training Exercises

A. Stage 1

Here you will work on balance. Begin at stage 1. Once you can complete all activities without support, proceed to stage 2.

- 1. Stand with hand on kitchen counter or other firm support object with eyes closed. March in place, counting to 50. Try to use the hand minimally. Gradually lift it off in an attempt to not use it. If you were able to march with eyes closed without use of the counter for support, advance to completing this activity with arm at your side.
- 2. Place thick sofa or foam cushion on floor 5 in from counter used above. Place sheet of paper with horizontal line on wall at eye level, 10 to 15 ft^b away. March in place on cushion as you look at the horizontal line, using the counter for support, as needed. Count to 50.

B. Stage 2

- 1. Stand in center of room, 20 ft from wall, with the paper with horizontal line taped to wall as above. Place several cushions on floor in a continuous line at least 15 ft long (between you and the wall with the paper). Have an assistant with you. With eyes open, walk across cushions as you look at the horizontal line, walking toward it. Assistant should be beside you to offer assistance in the event of a loss of balance. If you initially need to hold on to assistant, do so. On each consecutive day, attempt to use less and less support. Repeat 3 times.
- 2. Stand on cushion as in step B1, but put paper with letters on wall, 15 ft away. March in place as you look at the words and move your head from left to right. Be sure to have assistant beside you, or stand beside counter. Continue for 30 seconds. Repeat 3 times. Repeat, but move your head up and down.
- 3. Wearing foam "boots" provided, do daily activities in a standing position. This could be as you prepare a meal or work at a counter or workbench, as long as you are standing and moving periodically. This should be done 30 to 45 minutes per day.

^a 1 in=2.54 cm.

^b 1 ft=0.3048 m.