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Effects of Motion Sickness Severity on the Vestibular-Evoked Myogenic Potentials

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Abstract

Background—Motion sickness is a common debilitating condition associated with both actual and perceived motion. Despite the commonality, little is known about the underlying physiological mechanisms. One theory proposes that motion sickness arises from a mismatch between reality and past experience in vertical motions. Physiological tests of the vestibular system, however, have been inconclusive regarding the underlying pathogenesis. Cervical vestibular-evoked myogenic potentials (cVEMPs) arise from the saccule, which responds to vertical motion. If vertical motion elicits motion sickness, the cVEMP should be affected.

Purpose—The purpose of this investigation was to determine if cVEMP characteristics differ among individuals with a range of motion sickness susceptibility from negligible to severe. The hypothesis was that individuals with high susceptibility would have larger cVEMP amplitudes and shorter cVEMP latencies relative to those who are resistant to motion sickness.

Research Design—The study had two parts. The first was quasi-experimental in which participants comprised three groups based on susceptibility to motion sickness (low, mild-moderate, high) as identified on the short version of the Motion Sickness Susceptibility Questionnaire (MSSQ-S). The second part of the study was correlational and evaluated the specific relationships between the degree of motion sickness susceptibility and characteristics of the VEMPs.

Study Sample—A total of 24 healthy young adults (ages 20–24 yr) were recruited from the university and the community without regard to motion sickness severity.

Data Collection and Analysis—Participants took the MSSQ-S, which quantifies susceptibility to motion sickness. The participants had a range of motion sickness susceptibility with MSSQ raw scores from 0.0–36.6, which correspond to percent susceptibility from 0.0–99.3%. VEMPs were elicited by 500 Hz tone-bursts monaurally in both ears at 120 dB pSPL. MSSQ-S percent scores were used to divide the participants into low, mild-moderate, and high susceptibility groups. A fixed general linear model with repeated-measures analysis of variance tested cVEMP characteristics for the susceptibility groups (between participants) and ears (within participants). A

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univariate analysis of variance tested the cVEMP interaural amplitudes across groups. The second analysis was a regression of the severity of motion sickness in percent on cVEMP characteristics. Significance was defined as $p < 0.05$.

Results—Participants in the high susceptibility group had significantly higher cVEMP amplitudes than those in the low susceptibility group. cVEMP amplitudes did not differ between ears, and latencies did not differ between the two groups or between ears. Regression analysis on MSSQ-S percent susceptibility by VEMP amplitudes revealed a best-fit cubic function in both ears, with r^2 values of more than 42%. The interaural asymmetry ratio was negatively associated with motion sickness susceptibility ($r^2 = 0.389$).

Conclusions—The current study is the first to report that greater susceptibility to motion sickness is associated with larger cVEMP amplitudes and lower interaural cVEMP asymmetries. Larger interaural asymmetries in cVEMPs did not promote motion sickness susceptibility. The cVEMP findings implicate the saccule and its neural pathways in the production of motion sickness and are consistent with the theory that vertical motions elicit motion sickness. Motion sickness susceptibility may contribute to the variability in normative cVEMP amplitudes.

Keywords

Vestibular-evoked myogenic potential; motion sickness; saccule; vestibular nerve; inner ear; perception; humans; adults

INTRODUCTION

Motion sickness is characterized by autonomic and physiological signs of discomfort resulting from motion caused by many forms of transportation and by perceived motion. Susceptible people may experience nausea, emesis, cold sweats, excessive salivation, and headaches (Yates et al, 1998). People who are susceptible to motion sickness are also prone to experience nausea after surgery, during migraines, and in Meniere disease (Golding, 2006). Although motion sickness is a well-known debilitating condition, its pathogenesis is still unclear. The balance system includes the vestibular, visual, and somatosensory systems, which work together to provide orientation in space, stabilization of vision with movement, and maintenance of balance (Golding, 2006). A conflict among these systems theoretically can cause symptoms of motion sickness, but where or how the conflict occurs is not known (Reason, 1978).

Multiple theories about motion sickness exist, each explaining some but not all of the characteristics of motion sickness (for reviews, see Bles et al, 2000; Flanagan et al, 2004). The most common theory of motion sickness, the sensory rearrangement theory (Reason, 1978), has two tenets: first, eliciting motions must generate conflicting messages among the sensory, vestibular, and somatosensory systems; and, second, that the vestibular system must be involved in the generation of motion sickness.

With respect to the first tenet, specific conflicting messages have been identified by Reason (1978) and others. Reason reported two categories of possible conflicts: conflicts between the visual and vestibular systems and conflicts between the semicircular canals and the otolith organs. Two other potential conflicts include the interaural asymmetry (IAA)

between the otolithic organs (Diamond and Markham, 1992) and interaural asymmetries in the size of the otoconia in the utricle and/or saccule (Helling et al, 2003). Reason (1978) credited Held (1961) for the proposal that a conflict must occur between the current sensations and past experience in order to induce nausea.

The second of Reason's (1978) tenets stated that motion sickness emanates from the vestibular system, and vestibular stimulation requires motion to be variable rather than steady. The second tenet also requires the vestibular system to be functional for individuals to experience motion sickness. The necessity for vestibular viability includes studies in both animals and humans; animals no longer experience motion sickness after vestibular system ablations, and humans no longer experience motion sickness after pathological loss of vestibular function (Money, 1970; Cheung et al, 1991; Yates et al, 1998; Dai et al, 2007).

Several studies have focused on vertical perturbations in the genesis of motion sickness. O'Hanlon and McCauley (1974) proposed a model of motion sickness based on a variety of frequencies of vertical sinusoidal motion that induced motion sickness and vomiting. Lawther and Griffin (1986; 1987) corroborated the effects of vertical stimulation in motion sickness in a study of nearly 5,000 passengers on one ship over 17 voyages, each lasting up to 6 hr. Despite the many forces acting on a ship at sea, they noted that the vertical motion of the ship was most closely related to motion sickness. Subsequently, Bles et al (1998) produced a simplified model of motion sickness by using straight vertical forces and focusing on the vestibular system registering a conflict between the current and past perceptual experience of vertical motion.

Studies of individuals susceptible and nonsusceptible to motion sickness are inconclusive with respect to the underlying vestibular response that elicits symptoms. Motion sickness has been evaluated with electronystag-mography (ENG) and caloric responses (e.g., Lidvall, 1962), the vestibulo-ocular response (e.g., Gordon et al, 1996), and the balance platform (e.g., Flanagan et al, 2004). Preber (1958) reported that ENGs showed that individuals who were susceptible to motion sickness had faster slow-phase velocity (SPV) than motion sickness-resistant individuals when they were tested with caloric irrigation. Lidvall (1962) demonstrated faster SPV in caloric responses of participants who were sensitive to motion sickness, and concluded that motion sickness was caused by hyperactivity of the vestibular system. Gordon et al (1996) reported that the vestibular ocular reflex had higher gain and lower phase lead in persons with motion sickness compared with those who were resistant. On the other hand, Mallinson and Longridge (2002) found no relationships between motion sickness and caloric responses. All of their participants, however, both those with and without motion sickness, were patients in a dizziness clinic and presumably had some degree of vestibular disturbance.

One proposed mechanism for motion sickness lies in the response of the vestibular system to vertical movements (Bos and Bles, 1998; Tal et al, 2006; Buyuklu et al, 2009). Recent studies used cervical vestibular-evoked myogenic potentials (cVEMPs) to evaluate motion sickness due to its stimulation of the saccule, which responds to vertical acceleration. The afferent cVEMP pathway includes the saccule, inferior vestibular nerve (cranial nerve VIII), and the vestibular nucleus; responses are measured from the sternocleidomastoid muscle

where surface electrodes pick up the inhibitory motoneuron response (Colebatch et al, 1994; Kushiro et al, 1999; Colebatch and Rothwell, 2004; Welgampola and Colebatch, 2005). If motion sickness results from vertical stimulation, individuals with motion sickness sensitivity may be expected to have different cVEMP responses compared with responses in more resistant individuals. If increased vestibular activity occurs in individuals who are sensitive to motion sickness, then the cVEMP amplitude could be heightened in these individuals relative to cVEMPs in individuals who are more resistant to motion sickness.

The first studies evaluating motion sickness with the cVEMP, however, have not been conclusive. Tal et al (2006) reported that seasickness in sailors was associated with increased cVEMP thresholds and decreased cVEMP amplitudes. Sailors, however, may become adapted to motion with their frequent exposure to provocative stimuli, and their responses may not be representative of responses in the general population. Buyuklu et al (2009) tested the inferior vestibular nerve with cVEMPs and the superior vestibular nerve with the caloric tests in participants with and without susceptibility to motion sickness. The results of the physiological tests were not significantly different for the two groups, although the susceptible group did have larger cVEMP amplitudes.

The purpose of this investigation was to determine if cVEMP amplitudes and latencies differ between individuals who do and those who do not experience motion sickness. The hypothesis was that individuals susceptible to motion sickness have more robust responses from their vestibular systems defined by larger cVEMP amplitudes and shorter cVEMP latencies, compared with those who are resistant to motion sickness. This hypothesis developed from studies that indicate the vestibular system must be functional for motion sickness to occur (Yates et al, 1998; Dai et al, 2007), and studies indicating that vestibular function is heightened in individuals with motion sickness (Preber, 1958; Lidvall, 1962; Gordon et al, 1996). The research questions were as follows: (1) Do people with motion sickness have larger cVEMP amplitudes and shorter cVEMP latencies than those without motion sickness? (2) Do people with motion sickness have a left-right asymmetry in their vestibular systems as indicated by cVEMP amplitudes and latencies? (3) Do cVEMP amplitudes or latencies correlate with severity of motion sickness?

METHODS

Participants

A total of 24 healthy young adults were recruited from the local community, and all participants signed an informed consent before being entered into the study. A case history was taken to ensure that participants were not taking medications that could affect the vestibular system, were in good general health, and had not sustained head trauma. Otoscopy results revealed clear ear canals, and 226 Hz tympanometry was within normal limits (Roup et al, 1998). Air conduction thresholds were 20 dB HL or less at octave intervals from 250–8000 Hz; bone conduction thresholds were within 10 dB and interweaved with the air conduction thresholds.

Motion Sickness Susceptibility Questionnaire – Short Form (MSSQ-S)

Participants completed the MSSQ-S. Originally developed by Reason and Brand, the MSSQ was revised and shortened into the MSSQ-S by Golding (1998; 2006) and was shown to be reliable. The MSSQ-S comprises questions pertaining to individuals' susceptibility to motion sickness and the types of motion that are most provocative. Participants rated the frequency of their motion sickness [felt sick never (0 points), rarely (1 point), sometimes (2 points), or frequently (3 points)] for nine different types of transportation/motion in childhood and within the last 10 yr. Scores were determined by the following formula: total points for motion sickness frequency multiplied by the nine types of transportation divided by the number of types of transportation experienced. The result of the calculation for childhood experiences was designated MSA, and the calculation for adulthood experiences was designated MSB. The raw score was the addition of MSA and MSB, and could range from 0 (no experiences of motion sickness) to 54 (frequently experienced motion sickness for all types of transportation, both as a child and as an adult). On the basis of the raw scores obtained from the questionnaire results, participants were divided into three susceptibility groups based on the quartiles defined by Golding (2006) as follows: low susceptibility = first quartile, mild-moderate susceptibility = second and third quartiles, and high susceptibility = fourth quartile. A percentage ranking of motion sickness was expressed with the following fourth-order polynomial equation:

$$\begin{aligned} \text{Percent score} &= a * x + b * x^2 + c * x^3 + d * x^4 \text{ where } x' \text{ is the raw score, } a \\ &= 5.1160923; b \\ &= -0.055169904; c \\ &= -0.00067784495; \text{ and } d = 0.00001071452 \text{ (Golding, 2006).} \end{aligned} \quad (1)$$

cVEMPs

cVEMPs were conducted via Intelligent Hearing System instrumentation (version 4.14). Surface silver-silver chloride electrodes were placed on the sternocleidomastoid muscle one-third the way down from the mastoid muscle origin on both sides of the neck. The inverting input electrode was placed on the sternal notch, and the ground electrode was on the forehead. Electrode impedances were less than 5 kOhms, and interelectrode impedance differences were less than 2 kOhms. Physiological filters were set to pass activity from 30–1500 Hz, the artifact rejection was disabled, and responses were amplified 5,000 times. The time window was 100 msec, which included a 25 msec prestimulus baseline.

Stimuli were air-conducted 500 Hz rarefaction tone bursts with a 5 msec duration in a Blackman envelope. The tone-burst at 500 Hz was chosen because it has the largest amplitude in comparison with clicks and other tone-bursts (Akin et al, 2003). Stimuli were presented at 5.1/sec through insert earphones (ER-3A) monaurally at 120 dB pSPL. Ipsilateral cVEMPs were recorded for both the right and left ear stimulation, and the first ear to be tested was alternated between participants.

During the testing, the participants were seated in a reclining chair. Participants were instructed to keep their baseline electromyogram activity within a range of 50–100 μ V by

monitoring the level displayed by the IHS software on the computer monitor. Before the stimuli were presented, the tester instructed the participant to turn the head to one side, lift it slightly, and hold that position for the duration of each run (128 sweeps). Each condition was replicated. The tester was blind to the degree of motion sickness susceptibility of the participant until after the cVEMP amplitude and latency data were analyzed. MSSQ-S responses were kept in sealed envelopes until all waveform measures were completed.

Data Analysis

cVEMP amplitudes and latencies were measured for each ear. The latency of the initial positive component (P1) and the negative component (N1) of each cVEMP response were measured in milliseconds (msec). Amplitude was measured from the positive peak (P1) to the succeeding trough (N1) in microvolts (μ V). The IAA ratio was calculated with the following formula: $(A1 - A2) / (A1 + A2)$, where A1 is the larger amplitude and A2 is the smaller amplitude (Welgampola and Colebatch, 2005).

Statistical analyses (SPSS, version 20) were performed on the averaged values from the two waveform replications. The first set of analyses included a general linear model analysis of variance with repeated measures (RM-ANOVA) for the MSSQ-S group (between participants) and ear (within participants), with separate analyses for the amplitude of P1-N1, latencies of P1 and N1, and the P1-N1 interval. The Least Significant Difference test was used to determine significance among groups and interactions with ears. The IAA ratios were tested across groups with a univariate ANOVA. Significance was accepted at $p < 0.05$.

The second set of analyses included regressions with curve estimation to determine the best-fit function for the individual data for the amplitudes of P1-N1, IAA ratio, and latencies of P1 and N1 and their relationships with severity of motion sickness as defined by the MSSQ-S percentage scores. The model with the best fit determined by highest coefficient of determination (R^2) was used. Significance was accepted at $p < 0.05$.

RESULTS

Participants and Motion Sickness Susceptibility

Table 1 includes the characteristics of the 24 participants with their motion sickness susceptibility group, age, gender, and both raw and percentile MSSQ-S scores. Figure 1 shows MSSQ-S raw scores on the abscissa and percentile scores on the ordinate; the current data are represented by the open circles superimposed on the line and filled squares from Golding (2006). The 24 participants covered the range of motion sickness susceptibility with MSSQ raw scores ranging from 0.0–36.6, which corresponds to percentage susceptibility ranging from 0.0–99.3%.

cVEMP Comparisons across Motion Sickness Susceptibility Groups

cVEMPs were present for both ears in all participants and had the characteristic waveforms, as shown from a typical participant in Figure 2. Table 2 includes the mean data (with SDs) for the cVEMP amplitudes and IAA ratios for the three susceptibility groups. The RM-ANOVA for the cVEMP amplitudes of P1-N1 confirmed a significant main effect of group

$[F_{(2,22)} = 3.761, p = 0.040]$. The post hoc analysis localized the significance to the difference between the high- and low-susceptibility groups (least significant difference, $p = 0.014$), with the low-susceptibility group having the smaller amplitudes. Other group differences were not significant. The differences in cVEMP amplitudes between the ears and the interaction of group and ears also were not significant ($p > 0.05$). The univariate ANOVA for the cVEMP IAA ratios across susceptibility groups indicated that differences were not significant ($p > 0.05$).

Table 3 shows the latencies (and SDs) for P1, N1, and the P1-N1 interpeak latency difference. With respect to all latency measures, the RM-ANOVA indicated no significant differences for the main effects of the groups or ears or any interactions.

Relationships between Individual Data and Motion Sickness Susceptibility

The regression analysis with curve analysis modeled the distribution of the cVEMP P1-N1 amplitudes for the left and right ears for the participants relative to the MSSQ-S percent susceptibility scores. Figure 3 shows the individual data points for the left ear (x) and right ear (o) plotted for the MSSQ-S percentages (abscissa) and cVEMP amplitudes (ordinate). The cVEMP amplitudes as related to percent motion sickness susceptibility for both ears were modeled with a cubic function [left: $F_{(3,20)} = 4.771, p = 0.011$; right: $F_{(3,20)} = 5.665, p = 0.006$], and R^2 values indicated that 42% (left ear) and 46% (right ear) of the variability of the cVEMP amplitudes were explained by the MSSQ-S percent scores. Thus, the p -values confirm the significance of the source of the variance identified in the model. The function suggests that individuals who have virtually no susceptibility to motion sickness have the smallest cVEMPs, those with mild to moderate susceptibility have midrange cVEMP amplitudes, and those with the highest susceptibility to motion sickness have the highest cVEMP amplitudes. Latencies were not significantly related to MSSQ-S percentages.

Figure 4 shows the relationship between the individual IAA ratios (ordinate) and the MSSQ-S percentile scores (abscissa). A regression analysis with curve estimation showed a nonsignificant cubic relationship of IAA ratios with MSSQ-S percent score. Of the three MSSQ-S susceptibility groups, the low group had the most variability, with IAA ratios ranging from 0.04–0.45. The mild-moderate group was the most cohesive, with IAA ratios ranging from 0.00–0.19. The high-susceptibility group had an IAA ratio range of 0.16–0.60, which appears large. The ratio of 0.60 at the MSSQ-S score of 90%, however, is an outlier in the group (beyond ± 3 SD from the group mean; Marascuilo and Serlin, 1988, p. 73). Removal of the outlier reduces the range of IAA ratios for the high-susceptibility group to 0.01–0.15, which places the high-susceptibility group at the lowest variability of the three groups. Removal of the outlier also reduces the total ratio range to 0.00–0.45 and raises to significance the negative cubic regression of IAA ratios with MSSQ-S percent scores [$F_{(1,21)} = 9.251, p = 0.006$]. The R^2 now equals 0.389, which indicates that motion sickness susceptibility accounts for 39% of the variability of the IAA.

DISCUSSION

Greater susceptibility to motion sickness was positively associated with larger cVEMP amplitudes, suggesting that motion sickness susceptibility is associated with the saccule

and/or its neural pathways. By analogy, perception of the vertical motion, as coded by the saccular system, is implicated in the genesis of motion sickness as theorized by Bos and Bles (1998). The precise location along the cVEMP pathways at which the hyperactivity is generated, however, could not be determined by this study, and the study does not eliminate from contention other vestibular mechanisms that may also contribute to the susceptibility to motion sickness. Further research is needed to confirm the current results and to identify the source of the higher levels of along the cVEMP pathway in individuals who are most affected by motion sickness compared with those who are most resistant.

The cVEMP amplitudes, but not the latencies, in the current study corresponded with susceptibility to motion sickness. These results, however, are in direct contrast to the results of the only three previous studies that used cVEMPs (Tal et al, 2006; 2007; Buyuklu et al, 2009) to evaluate susceptibility to motion sickness. Buyuklu and colleagues had 20 participants who were susceptible and 20 who were not susceptible to motion sickness. The cVEMP protocol was essentially the same as the protocol in the current study; however, they also included ENG to characterize their participants further. They found no differences in the results of either the cVEMPs or the ENG caloric responses relative to MSSQ-S scores. Some participants in their subject population, however, may have had subclinical vestibular pathologic conditions, which could have led to the negative findings. In their susceptible group, two participants had no cVEMP response in the left ear, whereas all of their participants in the nonsusceptible group had cVEMPs in both ears. In the caloric stimulation, four participants in the susceptible group and one in the nonsusceptible group had canal paresis. The absence of cVEMPs and caloric responses may suggest that some underlying vestibular pathologic condition altered the vestibular responses in affected individuals. All normal individuals younger than 60 yr old are expected to have cVEMPs (Welgampola and Colebatch, 2005), and the caloric weakness may suggest a peripheral vestibular pathologic condition (Ahmed et al, 2009).

Tal et al (2006), on the other hand, reported decreased amplitudes and increased thresholds of cVEMPs in sailors who were highly susceptible to motion sickness. Differences in the participants in the two studies may explain some of the differences. Although both studies used 500 Hz tone-bursts, the Tal and colleagues reported much larger mean amplitudes of 252.5 μ V in the susceptible group and 365.7 μ V in the nonsusceptible group. In the current study, mean amplitudes were 96.5 μ V in the highly susceptible group and 63.8 μ V in the low/moderately susceptible group. The reason for this amplitude difference between studies is not clear. The two studies also used different questionnaires to quantify susceptibility to motion sickness, so comparisons of the severity of motion sickness in the studies cannot be made. Tal and colleagues used a 7-point scale, and the difference in scores between their two participant groups was only 4 points. By contrast, the current study used a 54-point scale (the MSSQ-S), and the scores of the participants covered a range of 0–37 points or 0–99% susceptibility, which allowed for a better description of the susceptibility of the various individuals. Finally, the participants in the study by Tal and colleagues were sailors whose vestibular systems may respond differently compared with the vestibular systems of people who are not exposed as frequently to motion sickness-inducing stimuli. The sailors may have learned to suppress motion sickness because individuals who are frequently exposed to motion learn to habituate to it. Another study by Tal et al (2007) found very poor responses

in sailors with susceptibility to motion sickness: one had bilateral cVEMPs, two had unilateral cVEMPs, and seven had no responses. In Tal's 14 nonsusceptible participants, who were also recruited from the navy, 7 had bilateral cVEMPs, 3 had unilateral cVEMPs, and 4 had no responses. The low-amplitude cVEMPs (all between 5.6 and 7.2 μ V) and absent cVEMPs were attributed to adaptation to the sea environment. Adaptation of sailors to seasickness was demonstrated with cVEMPs by Tal et al (2013). Newly recruited sailors who habituated to motion sickness during 6 mo at sea had lower cVEMP thresholds and a larger dynamic range for cVEMP amplitude compared with those who did not habituate.

The current study showed that only the participants most sensitive to motion had increased cVEMP amplitudes, whereas those with little to no susceptibility had the smallest cVEMPs. These findings are consistent with previous studies that have indicated a viable vestibular system is necessary for motion sickness to be experienced (e.g., Yates et al, 1998; Dai et al, 2007). These findings are also consistent with Lidvall (1962), who reported higher SPV in the caloric responses of people who were susceptible to motion sickness compared with those who were more resistant. The fact that many medications to relieve or prevent motion sickness reduce vestibular responsiveness (Schmäl, 2013) is also consistent with the larger responses in the susceptible individuals.

The current study also showed no evidence of interaural vestibular asymmetry. The largest range of IAA ratios was in the low-susceptibility individuals, and the size of the IAA ratios decreased as percent susceptibility to motion sickness increased. Furthermore, the analysis of cVEMP amplitudes from right and left ears found no significant differences in any of the susceptibility groups. These results are consistent with those of Buyuklu et al (2009), who also did not find IAAs in their study.

One limitation of the current study was that the cVEMPs stimulate only a limited portion of the balance system including the saccule, the inferior vestibular nerve, and related neural pathways, and thus can reveal only some of the mechanisms involved in susceptibility to motion sickness. ENG can assess the caloric responses, and the associated superior vestibular nerve and pathways; the ocular VEMP assesses the utricle, which responds to horizontal acceleration, and stimulates the superior vestibular nerve (Rosengren and Kingma, 2013). A battery of tests, therefore, ultimately may be necessary to characterize the contributions of the various vestibular structures in motion sickness. If the mechanism responsible for motion sickness is determined through future research, an appropriate testing protocol could be established for these patients. A standardized assessment protocol may allow clinicians to evaluate severity and monitor rehabilitation for individuals with motion sickness. A better understanding of this disorder may lead to new rehabilitative therapies for those affected.

The current study had a small number of participants with a limited demographic. A larger sample size would increase the validity of the study and allow stronger inferences about the general population. The optimal sample would be larger overall, span a wider age range, and include more severely affected individuals. If the current study is correct that higher levels of vestibular responsivity are associated with more susceptibility to motion sickness, and cVEMP amplitudes decrease with age (Su et al, 2004; Zapala and Brey, 2004; Basta et al,

2005), then older adults should gradually become less susceptible to motion sickness as their vestibular systems become less responsive with age.

In summary, the current study is the first to report larger cVEMP amplitudes in individuals who are highly susceptible to motion sickness compared with those who are most resistant to motion sickness. Because cVEMPs primarily indicate saccular function, the saccule and its neural pathways specifically are implicated and, by analogy, perception of vertical motion. The study, therefore, supports theories related to the perception of vertical motion in the genesis of motion sickness. This study, however, does not identify where sensory conflicts might occur, and further studies will be necessary to resolve that issue. A relevant clinical issue arising from the results is that susceptibility to motion sickness, either high or low, may contribute to the high variability of cVEMP amplitudes found in healthy populations. More studies are required to reach a consensus on the tests that can best evaluate motion sickness.

Abbreviations

cVEMPs	cervical vestibular-evoked myogenic potentials
ENG	electronystagmography
IAA	interaural asymmetry
MSA	motion sickness experienced in childhood
MSB	motion sickness experienced in the past 10 years
MSSQ-S	short version of the Motion Sickness Susceptibility Questionnaire
N1	first negative vestibular-evoked potential
P1	first positive vestibular-evoked potential
RM-ANOVA	repeated-measures analysis of variance
SPV	slow-phase velocity

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Figure 1.

Individual data points (open circles) for the MSSQ-S raw scores (abscissa) and MSSQ-S percent susceptibility scores (ordinate). The filled squares and lines are from normative data of Golding (2006). Current data encompass a wide range of susceptibility for both raw scores (0–37) and percentile scores (0–99%).

Figure 2.
VEMP P1-N1 wave from a typical participant, with peaks of interest labeled.

Figure 3.

The amplitudes of the right ear (o) and left ear (x) VEMP amplitudes as a function of MSSQ-S percent scores. The lines through the data show the cubic function that best fit the data. R^2 was 0.438 for the right ear and 0.406 for the left ear, indicating that the MSSQ-S score accounted for 46% and 41%, respectively, of the variability of the right and left ear VEMP amplitudes.

Figure 4.

Individual IAA ratios for all participants (ordinate) plotted against MSSQ-S percent scores (abscissa). The line through the data shows the cubic function that best fits the data. The diamond symbol in the upper-right corner of the graph is an outlier. With the outlier included, the coefficient of determination R^2 is 0.168 ($p > 0.05$); with the outlier removed, the R^2 is 0.389 ($p = 0.022$). Individuals with the least susceptibility to motion sickness have the greatest range of IAA ratios.

Table 1

Participant and Motion Sickness Characteristics of the Groups with Low, Mild-Moderate, and High Susceptibility to Motion Sickness

Susceptibility Group	Gender (M/F)	Age (yr) *	MSSQ-S Raw Scores *	MSSQ-S Percent Scores (%) *
Low	3 M	23.5 (1.3)	1.9 (1.7)	9.2 (8.2)
	6 F	21–25	0.0–4.0	0.0–19.5
Mild-Moderate	2 M	23.0 (2.04)	12.1 (3.7)	52.0 (13.2)
	5 F	23–25	6.8–17.0	32.0–69.0
High		24.2 (0.99)	28.0 (5.2)	90.3 (7.3)
	8 F	23–25	19.9–36.6	76.4–99.3

* Values are expressed as means (SDs) and ranges.

Table 2

Means (SDs) and Ranges for the VEMP P1-N1 Amplitudes and IAA Ratios for Each MSSQ-S Susceptibility Group and Both Ears

MSSQ-S Group	P1-N1 Amplitude (μ V)		IAA Ratio
	Right Ear	Left Ear	
Low (n = 9)	62.11 (22.08)	53.11 (29.57)	0.23 (0.14)
	35–100	13–90	0.04–0.45
Mild/Moderate (n = 7)	67.57 (13.66)	71.14 (23.13)	0.10 (0.07)
	41–84	32–94	0.00–0.19
High (n = 8)	98.13 (34.29)	95.25 (47.75)	0.15 (0.10)
	65–163	17–167	0.01–0.60
Total (N = 24)	75.71 (29.04)	72.42 (38.28)	0.16 (0.15)
	35–163	13–167	0.00–0.60

Table 3
Means (SDs) and Ranges of VEMP Latencies for the Right and Left Ears for the Three MSSQ-S Susceptibility Groups

MSSQ-S Group	Latencies (msec)					
	Right Ear			Left Ear		
	P1	N1	P1-N1	P1	N1	P1-N1
Low	14.6 (1.5)	20.9 (1.4)	6.8 (1.7)	15.0 (1.7)	20.4 (2.1)	5.2 (1.6)
	13.1–15.4	18.5–23.4	4.1–7.8	13.0–17.0	17.4–23.0	1.8–7.3
Moderate	14.7 (2.1)	20.6 (4.4)	6.0 (3.0)	14.0 (2.3)	20.7 (4.8)	6.9 (2.8)
	12.2–17.1	16.9–29.4	4.1–8.9	11.8–18.1	16.5–29.8	4.5–11.7
High	13.9 (1.1)	19.8 (2.4)	6.0 (1.6)	14.1 (2.5)	20.9 (3.4)	6.5 (1.9)
	13.0–16.5	16.2–24.4	2.8–7.9	10.4–17.0	14.4–23.5	4.0–9.2
Total	14.4 (1.6)	20.4 (2.8)	6.3 (2.1)	14.4 (2.1)	20.7 (3.3)	6.1 (2.1)
	12.2–17.1	16.9–29.4	2.8–8.9	10.4–18.1	14.4–23.0	1.6–11.7