THE VESTIBULO-OCULAR REFLEX AND SEASICKNESS SUSCEPTIBILITY

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Abstract — VOR parameters were compared in subjects at the extremes of the seasickness susceptibility scale. Thirty-nine subjects highly susceptible to seasickness and 30 nonsusceptible subjects participated in the study. The VOR was evaluated by the Sinusoidal Harmonic Acceleration (SHA) test at frequencies of 0.01, 0.02, 0.04, 0.08, and 0.16 Hz. In subjects susceptible to seasickness, VOR gain was significantly higher at 0.02 and 0.04 Hz, and phase lead was significantly lower at 0.01, 0.02, 0.04, and 0.08 Hz, than in nonsusceptible subjects. Our findings are in agreement with the notion that the vestibular response will be more intense in subjects susceptible to motion sickness. The present results support the contention that a natural insusceptibility, or increased resistance to seasickness produced by adaptive responses to repeated sea exposures, may be reflected by lower VOR gain and higher phase lead.

Keywords — motion sickness, seasickness susceptibility; vestibulo-ocular reflex; sinusoidal harmonic acceleration; vestibular habituation; adaptation.

Introduction

Because a functional vestibular system is essential for the development of motion or seasickness, it was speculated early on that vestibular function, as measured by different vestibular tests, could be employed in the evaluation of motion sickness susceptibility. Earlier studies reported longer duration of a turning, rotation-induced sensation, or a steeper sensation cupulogram and a lower perception threshold, in subjects susceptible to motion sickness compared to nonsusceptible subjects (1–5). Studies using rotatory or caloric electronystagmography (ENG) also reported differences between susceptible and nonsusceptible subjects (6–8). These findings were explained in terms of higher labyrinthine sensitivity in subjects susceptible to motion sickness. However, other studies failed to confirm the above findings and explained the reported differences in terms of reduced vigilance, flaws in the experiment, or technical drawbacks (9,10). Earlier rotatory tests and caloric ENG have been found to be highly variable, in particular due to the inability to control and quantify the stimulus (acceleration) and the inaccurate (naked eye) analysis of the response. The advent of sophisticated and reliable torque–motor drive systems has made possible the precise control of acceleration, which is the fundamental vestibular stimulus. The addition of a computer, which digitizes and statistically analyses eye movement data in response to angular acceleration, provides a constant test protocol and highly accurate analysis.

In a previous study conducted in our laboratory using the low frequency Sinusoidal Harmonic Acceleration (SHA) test, Shupak and colleagues (11) studied vestibulo-ocular reflex (VOR) changes related to habituation in a heterogeneous group of candidates for future sea service. Lower VOR gain values at 0.01 to 0.08 Hz
correlated with less susceptibility to seasickness, as reported after 1 month of regular sailing. A significant increase in phase lead at 0.01 to 0.04 Hz after 6 months of sailing was related to the process of habituation to sea conditions. On the basis of these results, we postulated that differences might exist between the VOR gain and phase lead of subjects susceptible and non-susceptible to seasickness.

The purpose of the present study was to evaluate VOR measurements in seamen at the extremes of the seasickness susceptibility scale.

**Materials and Methods**

A group of 39 subjects highly susceptible to seasickness and a group of 30 nonsusceptible subjects were selected from a naval crew population. All subjects were healthy males aged 18 to 23 y, with at least 3 months’ sailing experience. They generally sailed 1 to 3 times a week, each voyage lasting 5 to 10 h. A detailed neurootological examination was normal in all subjects. All were unpaid volunteers; their informed consent was obtained, and the study was approved by the local Helsinki committee.

Susceptibility to seasickness was determined using a questionnaire concerning past and present history of motion sickness (12) and a seasickness questionnaire adopted from Wiker and colleagues (13,14) concerning actual seasickness severity during sailing. The validity and reliability of this questionnaire were proved to be high in a seasickness survey conducted by the United States Coast Guard (14). Based on self-reported symptomatology, seasickness is rated on a scale from 0 to 7. Our susceptible group included 39 highly susceptible subjects who had a positive past and present history of other forms of motion sickness according to the motion sickness questionnaire and who scored 7 points on the categorization scale (subjects who regularly vomited during sailing). The nonsusceptible group included 30 nonsusceptible subjects who had a negative past and present history of other forms of motion sickness and who scored from 0 to 2 points on the categorization scale (subjects with no signs or symptoms of motion sickness during sailing, or sometimes suffering only slightly from minor symptoms). This categorization was further validated using a peer questionnaire. Subjects were completely drug and alcohol free for at least 72 h before the study.

The VOR was evaluated by the SHA test, as described by Wolfe and colleagues (15,16), using the Contraves Goerz DP-300 computerized rotatory chair system. Each subject was exposed to a series of yaw accelerations ranging from 3.8°/s² to 60°/s², at a maximum velocity of 50°/s and at frequencies of 0.01, 0.02, 0.04, 0.08, and 0.16 Hz. The trials included 4 sinusoidal cycles at 0.01 Hz and 8 cycles at each of the other frequencies. Subjects sat with their heads tilted forward 30° in order to achieve maximum stimulation of the horizontal semicircular canals. The test was conducted in total darkness. Alertness was assured by continuous conversation and questions. Standard electronystagmographic techniques were employed to record the eye movements. VOR gain, phase lead, and asymmetry were calculated as described previously (11,17). Only if spectral purity is greater than 75% can the gain and phase lead be considered accurate descriptors of the VOR. The results were compared using unpaired Student t test.

**Results**

Table 1 summarizes the mean values of VOR gain, phase lead, and asymmetry for the 2 study groups. Individual gain and phase lead values are shown in Figure 1 panels A and B, respectively. VOR gain was higher and phase lead lower in the susceptible group. Significant differences in gain values were found between the groups at 0.02 and 0.04 Hz ($P < 0.05$). At 0.08 and 0.16 Hz, differences bordered on statistical significance ($P = 0.056$ and $0.055$, respectively). Phase lead was significantly different between the groups at 0.01, 0.02, 0.04, and 0.08 Hz. There were no significant differences in asymmetry between the groups. All individual VOR parameters were within the normal range in both groups. Spectral purity in all test trials was greater than 80%. None of the subjects reported any discomfort or symptoms of motion sickness during the test.
Table 1. Gain, Phase Lead, and Asymmetry in Subjects Highly Susceptible (H S) and Nonsusceptible (N S) to Seasickness

<table>
<thead>
<tr>
<th></th>
<th>0.01 Hz</th>
<th>0.02 Hz</th>
<th>0.04 Hz</th>
<th>0.08 Hz</th>
<th>0.16 Hz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gain</td>
<td>HS</td>
<td>0.60 ± 0.18</td>
<td>0.93 ± 0.19</td>
<td>0.91 ± 0.19</td>
<td>0.97 ± 0.22</td>
</tr>
<tr>
<td></td>
<td>NS</td>
<td>0.54 ± 0.14</td>
<td>0.83 ± 0.20</td>
<td>0.80 ± 0.20</td>
<td>0.87 ± 0.19</td>
</tr>
<tr>
<td>Phase lead (degrees)</td>
<td>HS</td>
<td>37.6 ± 6.65</td>
<td>21.3 ± 5.33</td>
<td>11.9 ± 5.10</td>
<td>5.19 ± 6.57</td>
</tr>
<tr>
<td></td>
<td>NS</td>
<td>42.7 ± 7.36</td>
<td>24.4 ± 5.04</td>
<td>15.1 ± 4.48</td>
<td>8.18 ± 4.37</td>
</tr>
<tr>
<td>Asymmetry</td>
<td>HS</td>
<td>-2.1 ± 9.7</td>
<td>-2.4 ± 7.8</td>
<td>-2.7 ± 7.4</td>
<td>-2.1 ± 9.7</td>
</tr>
<tr>
<td></td>
<td>NS</td>
<td>-6.2 ± 10.2</td>
<td>-4.0 ± 8.8</td>
<td>-3.2 ± 7.7</td>
<td>-3.3 ± 8.7</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation.
*p < 0.05.

Discussion

The present results demonstrate a significant difference in VOR gain and phase lead, as measured by the SHA test, in subjects at the extremes of the seasickness susceptibility scale. VOR gain was significantly higher at 0.02 and 0.04 Hz, and phase lead significantly lower at 0.01, 0.02, 0.04 and 0.08 Hz, in subjects highly susceptible to seasickness compared to nonsusceptible subjects.

Our findings are in general agreement with most early studies, that found that subjects susceptible to motion sickness have a more intense vestibular reaction than nonsusceptible subjects, as measured by a reported turning sensation, cupulometry, or a caloric ENG test (1–8). Despite the differences described, no vestibular test or any other physiological measurement has been employed to categorize individual motion sickness susceptibility. As Graybiel (18) pointed out, the intensity of the vestibular response might not necessarily be related to motion sickness symptomatology that depends on complex central nervous system integration of physiological and psychological cues. As can be seen from Figure 1, even when a significant difference was found between the groups, there was broad overlapping of individual values, with no clearly defined point at which one can differentiate between susceptible and nonsusceptible subjects.

The present results are also in agreement with findings previously reported by us in a population of crew members examined at the beginning of their naval service and 6 months later. Lower VOR gain values at 0.01 to 0.08 Hz were associated with less susceptibility to seasickness as reported after 1 month of regular
sailing. A significant decrease was found in VOR gain at 0.01 and 0.02 Hz, as well as a significant increase in phase lead at 0.01 to 0.04 Hz, after 6 months of regular service at sea (11).

Inasmuch as all of the subjects in the present study had at least 3 months experience at sea, sailing 1 to 3 times a week, the findings could be explained in terms of a more adequate process of adaptation to sea conditions in subjects categorized as nonsusceptible to seasickness, rather than by differences in primary or natural susceptibility. It may also be argued that a more appropriate process of adaptation to the unnatural motion patterns produced by a ship at sea took place in the nonsusceptible subjects, thus making it quite impossible to differentiate between primary and acquired insusceptibility. The term “adaptive process” is used to indicate the neurophysiological responses that improve the interaction between the subject and the unnatural motion environment at sea. They include both vestibular habituation and the adaptive VOR changes induced by a mismatch of visual and vestibular inputs (19). It is quite probable that during repeated sea voyages, subjects are exposed both to unnatural, low frequency motion stimulation, resulting in vestibular habituation, and to visual–vestibular mismatch, which produces adaptive VOR modifications.

Vestibular habituation to different kinds of stimulation has been studied in animals and humans (20). Habituation to low-frequency sinusoidal oscillations has been well documented in the monkey and in man; phase lead increased and gain decreased with the development of habituation (21–23). In a recent study, a decrease in the caloric nystagmus response was reported in subjects exposed to the open sea for 72 h. This was explained in terms of vestibular habituation (24). Numerous studies have documented adaptive VOR gain and phase modifications in response to induced visual–vestibular mismatch (25). For example, subjects wearing prisms that invert the seen world laterally had impressive adaptive gain and phase changes that actually reversed their VOR. While these changes were taking place, the subjects reported symptoms of motion sickness, reflecting the mismatch between visual and vestibular inputs (26).

It has been found that two of the most commonly used anti-motion sickness drugs, dimenhydrinate and scopolamine, reduce the gain of the vestibular and optokinetic response. This effect was accompanied by a concomitant diminution of experimental nausea and vertigo (27,28). We recently reported the effectiveness of the antihistaminergic agent cinnarizine in preventing seasickness and its effect in reducing VOR gain as measured by the SHA test (29,30).

The present results and the studies mentioned above support the contention that increased resistance to seasickness as a result of primary insusceptibility to motion sickness, adaptive responses to repeated sea exposures, or the administration of anti-motion sickness drugs may be reflected in lower gain and higher phase lead. Whether habituation to low frequency sinusoidal stimulation, a nonprovocative motion sickness stimulus, is accompanied by an increase in resistance to sea and motion sickness is still a matter for investigation.

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