

Presence of Vestibular Dysfunction in Individuals with Motion Sickness

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Abstract

Aim and Objective: To study if there is vestibular dysfunction in individuals with motion sickness.

Type of Study: Correlation study.

Study population: Motion sickness Individuals from age 20-40 years and fulfilling the inclusion criteria were approached from the community for the study. Interested individuals were asked to fill the motion sickness questionnaire for the information about the symptoms. Vestibular function clinical tests like Dynamic Visual Acuity [DVA], Modified clinical test of sensory interaction on balance (MCTSIB) and Head thrust test were used to assess vestibular dysfunction in these individuals.

Results were obtained using spearman's correlation analysis.

Results and Conclusion: There is a strong correlation between motion sickness and vestibular dysfunction. ($r=0.951, p<0.0001$)

Keywords: Motion sickness; Vestibular dysfunction; MCTSIB; DVA; Head thrust

Introduction

Motion sickness (MS) is a syndrome which is produced by the response to a perceived motion which involves various autonomic nervous system symptoms of nausea, cold, sweating, paleness, and vomiting in susceptible persons.

Other frequently experienced symptoms include headache, drowsiness, sleepiness, apathy, depression, and a reduction in cognitive function, reflected by reduced performance on various psychomotor tasks.

Most common types of Motion Sickness are:

- Car sickness
- Sea sickness
- Vehicle simulator sickness, and space sickness [1]

As the time spent on transport systems occupies a considerable part of daily life, travellers normally perform a variety of activities while being transported, leading to various active head movements during passive motion. However, motion sickness can be provoked or aggravated by active head movements in the presence of passive motion, considerably hindering the quality of travel [2]. Normal activities of daily life (such as running) can have head velocities of up to 550°/s, head accelerations of up to 6,000°/s², and frequency content of head motion from 0 to 2 Hz. Only the vestibular system can detect head motion over this range of velocity, acceleration, and frequency. Additionally, the latency of the vestibulo-ocular reflex (VOR) has been reported to be as short as 5 to 7 milliseconds. In contrast, ocular following mechanisms such as smooth pursuit, generate slower eye velocities ($\sim 60^\circ/s$) and have relatively long latencies (up to 100 milliseconds) [3].

The various physiological correlates of Motion sickness that have been investigated in the past, are alterations in heart rate and respiratory rate variability [4,5] plasma hormonal changes [6], salivary secretion and personality factors [7], changes in gastric motility [8], and the saccular reflex [9]. However, neither physiological nor functional variables have been found to be of sufficient sensitivity and specificity to act as a single diagnostic measurement for motion sickness susceptibility. In absence of the motion-sensing organs of the inner ear, motion sickness

does not occur. This condition suggests that the inner ear is critical for development of motion sickness. The reactions of dogs to emetic drugs is greatly reduced or absent after they have been labyrinthectomized compared with their baseline responses.

There are large individual differences with respect to susceptibility to motion sickness. The incidence of motion sickness varies, depending on the intensity of the stimulus and the susceptibility of the individual. The cause of motion sickness is unknown, but MS is generally produced by environmental challenges such as linear and or angular accelerations. Many studies have been done to establish the cause and objective criteria for diagnosing and evaluating motion sickness [1].

Prevalence of motion sickness among north east Indians was 28% and 26% among northwest Indians. Generally speaking, females 27.3% were more susceptible than males 16.8%. According to the national travel survey of 1000 men and women, 63% report of motion sickness. Nearly 60% of astronauts report experiencing motion sickness, approximately, 30% of ocean liner passengers and nearly 40% of flight.

The range of vulnerability in the normal population varies about 10,000 to 1.

Ears and eyes are the most potent receptors of provocative motion that causes sickness [10].

Various theories on motion sickness have been put forward but none of them justifies the exact cause for motion sickness. The theories include the evolutionary theory holds that motion sickness is essentially a response to poisoning. The notion is that when a noxious substance is ingested (e.g. rotting flesh) if nausea and vomiting result, inactivity will be induced and symptoms will be attenuated because of reduced levels

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of toxins circulated in the blood stream. Decreased activity enhances the possibility of recovery.

The second suggestion given by the theory was in motion sickness many systems are involved in controlling movement and determining location of body in space and continually in action and highly susceptible to even a minor disruption they cause an early warning system for detecting early central effects of neurotoxins as defence mechanism in form of emesis [10].

The sensory conflict theory of motion sickness proposed by (Reason 1969 and Brand 1975) was developed into a quantitative model by Oman [11].

Until a century ago physicians commonly referred motion sickness because of reduced cerebral blood flow or mechanical stimulation of abdominal afferents owing to motion of the viscera.

These ideas were termed as “blood and guts” theories which were rejected as individuals who lack inner ear vestibular function are immune to motion. It was widely accepted that MS is produced by the overstimulation of the vestibular organs by “unnatural” body motions. Provocative stimuli can be visual or vestibular in origin which contradicted the hypothesis of previous theories. Sensory conflict theory provided broader etiological perspective on syndrome. This conflict theory is now widely accepted. This theory is being termed as basis for seasickness, car sickness, air sickness, flight simulator sickness etc.

Sensory conflict hypothesis was put forth on the discrepancy between the information given to an individual by one set of sensations with that of other, and rather their occurrence in synchronised combination.

For e.g. when travelling by bus the visual system gives us the information for being stationary and other system gives an input about motion.

First the theory was assumed to be directly as a result of conflict between the signals provided by different sensory modalities (e.g. the signals from the eye, ear do not agree). However further in 1978 it was rejected. The sensory information coming to central nervous system has normal behaviour and coding in which conflict can be dependent on context and previous experiences. Thus, inter modality conflict was rejected. Reafference principle or neural mismatch hypotheses which explained that brain mostly evaluates the sensory information which gets stored in the form of memory and then updates on the basis of experiences interacting with environment. Whenever a movement is commanded, CNS fetch from the neural store associated normally anticipated sensory input which corresponds to “efference copy”.

Actual sensory input and retrieved sensory memories traces are continuously compared leading to sensory conflict. What remains perplexing, however, is why some conflicts are provocative and others are not. But overall in all the theories, the major part is played by the vestibular system which leads to the conflict with visual system. So there is a need to find out if the function of the vestibular system is affected or the reflex pathway [11].

The ecological theory of motion sickness is based on the hypothesis that motion sickness is caused by postural instability, a loss of postural control. They defined postural stability as “the state in which uncontrolled movements of the perception and action systems are minimized. Postural instability might be a situation in which person tries to control the posture in presence of oscillations. Studies of the relationship between postural instability and onset of motion sickness also show little support for the ecological theory of motion sickness

[23]. It is likely that the conditions in which postural instability is before “motion sickness” are actually conditions in which postural hypotension is being elicited, hence symptoms such as “can’t see straight, feeling dizzy,” “hard to focus, everything is gray,” and occasionally nausea can occur. This study was conducted to study if vestibular dysfunction is present in individuals with motion sickness.

Motion sickness questionnaire

This questionnaire is primarily concerned with:

- Susceptibility to motion sickness and,
- The types of motion are most effective in causing this sickness.

It is reliable and validated tool for assessing susceptibility of motion sickness with values of $r=0.8$ and test re-test reliability= 0.8 [12].

Tests performed to evaluate vestibular dysfunction

Dynamic Visual Acuity test (DVA): The dynamic visual acuity test assesses gaze stability during sinusoidal, examiner mediated head rotations relative to head stationary visual acuity.

To administer this test visual acuity is first assessed under static head movement condition i.e. (no head movement).

The patient is instructed to wear prescription lenses if needed for distant viewing then seated at distance of 20 feet.

The patient is instructed to read the lowest line recognisable and to keep reading until he or she can no longer identify all letters on a given line. The examiner should take notes of last line where all letters were recognisable for the dynamic component of the test, the examiner should stand behind the patient and firmly grasps the patient’s head with both hand just above the ears. For testing in the yaw plane, the patient’s head should be flexed forward 30 degrees to bring horizontal semi-circular canals in plane of testing. Next the examiner will oscillate head at 20-30 degree from midline. Cervical ranges were measured and corrective lenses if present. Frequency of rotation 2 Hz i.e. (2 cycles per second) was maintained by metronome. The patient will read the letters on the lowest line of eye chart possible until he or she no longer identifies them. Examiner should note the line. A loss of 3 or more lines of visual acuity in relation to one’s static visual acuity is regarded as clinically significant and suggestive of vestibular dysfunction. Dynamic visual acuity test has excellent test-retest and inter-tester reliability (ICC=0.94 and ICC 0.84) for the horizontal dynamic visual acuity test. Sensitivity, specificity positive and negative predictive values were 100% to identify vestibular dysfunction [13].

Head thrust test: (Head-Trust or Halmagyi test) Patients head has been pitched 30° down to move the subject’s head in the plane of the lateral semi-circular canals. Patient was asked to fixate on the top of the examiner’s nose and the examiner moved the head quite rapidly to the left. Then the head was moved rapidly to the right. In healthy subject, eyes remained fixed on their target. Then the manoeuvre was repeated to the right side. If any labyrinth did not work properly, the eyes performed a catch-up saccade to the right or left to fixate the target again. This presence or absence of saccades is noted. Presences of saccades denote vestibular dysfunction. Head impulse thrust specificity is 82% and sensitivity is 71% to identify vestibular dysfunction [14].

Modified clinical test of sensory interaction on balance: The mCTSIB is a modification of the CTSIB that eliminates the use of the visual conflict dome. It includes conditions 1,2,4,5 of the original CTSIB. To perform the test, the patient stands with their hands at their side and performs the following 4 conditions:

- (CTSIB condition 1) Stand on firm surface with the eyes open.
- (CTSIB condition 2) Stand on firm surface with the eyes closed.
- (CTSIB condition 4) Stand on compliant surface (foam) with the eyes open
- (CTSIB condition 5) Stand on compliant surface (foam) with the eyes closed.

Condition 4 will be done to assess vestibular dysfunction. Patient performance is timed for 30 seconds. If a patient is unable to maintain the position for 30 seconds i.e. if they sway it denotes vestibular dysfunction. They are provided with 2 additional attempts. The scores of the 3 trials are averaged. The mCTSIB condition 4, standing on foam with the eyes closed has sensitivity of 95%, and specificity of 90% compared to SOT condition 5 in identifying vestibular dysfunction [15].

Materials and Methods

Type of study

Correlation study.

Study population

Motion sickness individuals.

Inclusion criteria

- People from age 20-40 years
- Both males and females
- Having motion sickness according to motion sickness questionnaire
- People having corrected vision 6/6

Exclusion criteria

- People having any balance, anxiety disorders
- People having any history of neurological conditions, visual disorders
- People having any musculoskeletal problems

Materials

- Snellen's Chart
- Measuring tape.
- Goniometer to measure cervical ranges
- Medium density foam
- Stop watch
- Metronome (with 120 bpm)

Outcome measures

- Motion sickness questionnaire
- Dynamic visual acuity test
- Head impulse thrust
- Modified Clinical test of sensory interaction on balance

Study settings: Community

Sampling technique: Purposive sampling (as we have selected samples who have reported to have motion sickness)

Sample size: Sample Size=167

Procedure

Ethics clearance was obtained from the institutional ethical committee. Individuals from age 20-40 years and fulfilling the inclusion criteria were approached from the community for the study. Interested individuals were asked to fill the motion sickness questionnaire for the information about the symptoms. Written consent was taken from these individuals (Figure 1).

Vestibular function clinical tests were used to assess vestibular dysfunction in these individuals these tests consist of:

- Dynamic visual acuity [DVA]
- Modified clinical test of sensory interaction on balance (MCTSIB)
- Head thrust test Results were obtained and descriptive statistical analysis was done.

Results

For carrying out the following study, statistical analysis was performed for sample size estimation.

In this study about 167 Individuals from age 20-40 years from community were approached interested individuals were asked to fill the motion sickness questionnaire for the information about the symptoms.

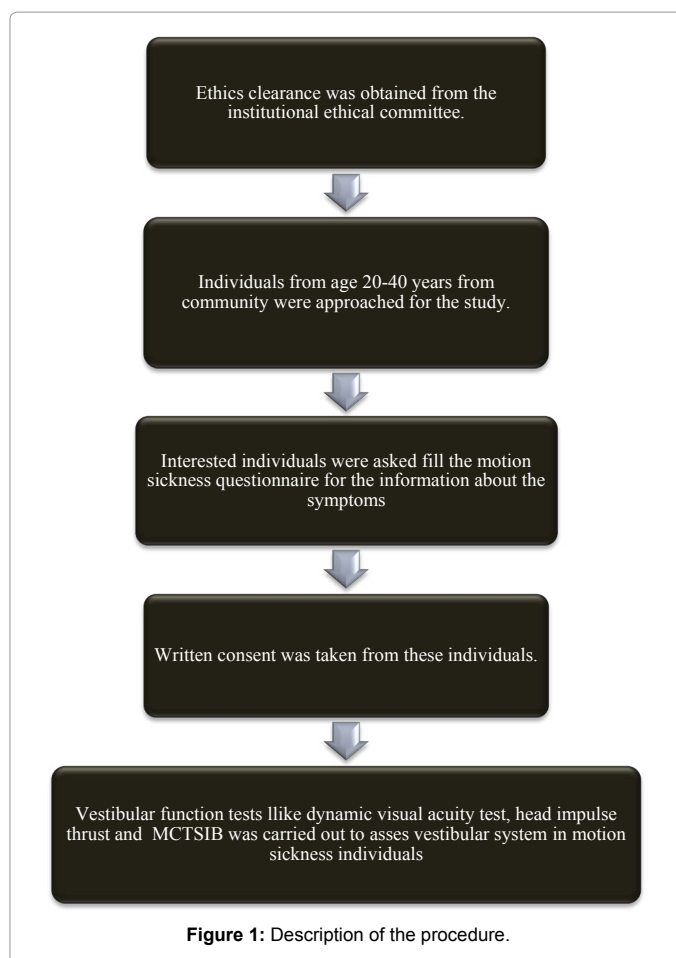


Figure 1: Description of the procedure.

Vestibular function tests were carried out on these individuals which included dynamic visual acuity, head impulse thrust, modified CTSIB.

Data coding was done depending on the severity of motion sickness. The average motion sickness was given number 1, more than average was given number 2 and much more than average were given number 3.

For the Head impulse thrust if the saccadic movement was seen it was scored as 1, if no saccadic movement was seen, it was scored as 0. In case of DVA if Individual could read all 3 lines, score was given 0, if could not complete 3 lines, then score was 1 and if can't complete the test then it was given score 2.

In case of modified CTSIB, if the time the individual could stand is within normal range (29.8 or more than), it was denoted as 0; if individual could stand for 20-29.7 seconds, then it was given score 1, if could stand for 11-20, it was denoted by 2 and could stand less than 10 sec, it was given rating 3.

The scores of all 3 vestibular dysfunction were added.

Correlation between motion sickness severity scores and summated vestibular dysfunction score was established using spearman's correlation test.

Discussion

Motion sickness is caused by unfamiliar (non-adapted) motion stimuli and particularly, by perceptual incongruences among the visual, vestibular, and somatosensory systems. Individual differences in motion sickness susceptibility and physiological responses to motion stimulus make predicting motion sickness susceptibility difficult [9]. An attempt was made in order to find out the presence of vestibular dysfunction in individuals with motion sickness from age 20-40 years from the community.

Interested individuals having motion sickness were asked to fill the motion sickness questionnaire for the information about the symptoms in them vestibular function tests were carried out which included dynamic visual acuity, head impulse thrust and modified CTSIB.

Table 1 describes the age distribution among the motion sickness individuals showing no. of cases of motion sickness goes on decreasing as the age increases. The reason for that may be because of the habituation of vestibular system, it may also be related with ageing of the vestibular system, or avoidance of motion producing environment [16]. Table 2 describes that women showed higher incidence of motion sickness i.e. about 93.4% and men 6.6% which clearly states that women are more susceptible to motion sickness which may be associated with 2 reasons, Female Hormonal cycle [16,17]. Or it may be also related to the difference in anthropometric measurements in men and women.

Age (in years)	No. of cases (in %)
< 25	77
30	49
35	16
40	25

Table 1: Age wise distribution of the subjects.

Gender	Percentage
Male	93.40%
Female	6.60%

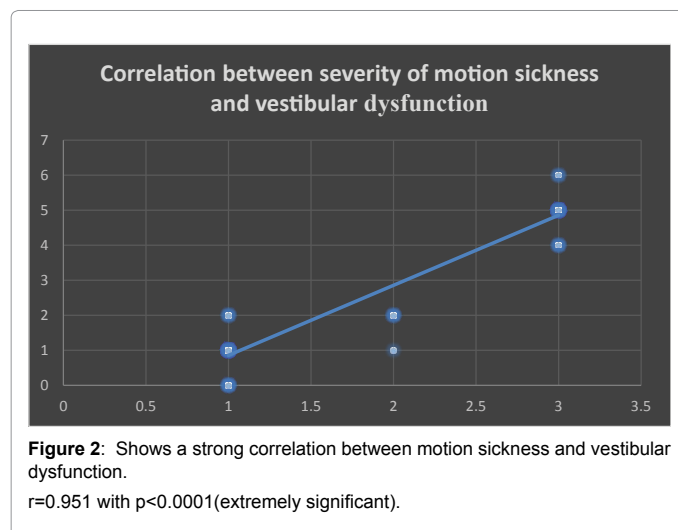
Table 2: Genderwise distribution of cases.

Koslucher et al. (2016) also found incidence of motion sickness in women was greater than in men in which motion sickness was correlated with bodily anthropometric measurements. Motion sickness was found negatively correlated with weight, height, BMI [18]. Figure 2 state that there is a strong correlation between motion sickness and vestibular dysfunction.

The vestibular system detects motion of the head and maintains stability of images on the fovea of the retina as well as postural control during head motion. Signals representing angular and translational motion of the head as well as the tilt of the head relative to gravity are transduced by the vestibular end organs in the inner ear [3]. This sensory information is then used to control reflexes responsible for maintaining the stability of images on the fovea (the central area of the retina where visual acuity is best) during head movements [3]. If the vestibular system is not generating an adequate compensatory eye velocity during head rotation, there would be a deficit in the VOR, leading to motion of images on the fovea [3].

Similarly, in this study, individuals with motion sickness were not able to generate adequate eye velocity to read the letters from Snellen's chart when head was rotated at the frequency of 2 Hz/s. This decline in visual acuity suggests sub-clinical vestibular dysfunction [3]. In accordance to this study, Webb NA, concluded that subjects with poor visual acuity experienced greater sickness and there is an association between visual acuity and motion sickness [19]. In accordance to this study Gordan demonstrated that vestibular ocular reflex and sea sickness susceptibility concluded that vestibular responses will be more intense in subjects susceptible to motion sickness and the VOR gain was significantly higher at 0.02 Hz and 0.04 Hz [7]. In the present study, head impulse thrust was not found to be positive i.e. Saccades were not seen on assessment. Most of subjects could not tolerate the continuous head rotation in the following test even after the rest period was given in between, and started getting symptoms of nausea and dizziness. Therefore, the test was not carried out further.

Motion sickness individuals on an average were able to maintain balance for less than 29.8 which is normal which suggests that vestibular contribution for maintaining postural control was affected in these individuals having motion sickness value [20]. Postural instability was in the form of body sway in condition 4 of MCTSIB. Since the test was



non instrumental MCTSIB quantification of sway was not possible [21]. Motor behaviours are highly dependent on specific labyrinthine signals, vision and somatosensory system [22,23]. Stabilizing reactions at the neck and ankle, for example, are altered when peripheral vestibular inputs are reduced, but the remaining responses are still sufficient to maintain an upright position [22]. In situations where other sensory information is also unreliable (e.g. conflicting visual inputs and somatosensory inputs in ships or buses), peripheral vestibular deficits create greater instability leading to postural instability in conflicting environment [23].

This experimental relation between spontaneous postural sway, imposed on motion sickness was predicted, previously, by a new theory of motion sickness. Riccio and Stoffregen hypothesized that motion sickness is caused by instability in the control of the posture of the body and or its segments. There can be variation in the magnitude of instability, and instability can persist over long periods of time without necessarily leading to loss of control [24].

Motion sickness is syndrome which includes autonomic nervous system symptoms of nausea, cold, sweating, paleness, and vomiting in susceptible persons. Studies have shown that there is anatomical and physiological relation between vestibular and autonomic nervous system. Any head rotation and changes in body orientation can have consequences on autonomic nervous system. The central circuits explain the strong association of vestibular stimulation and the autonomic motor responses through descending effects on brain stem autonomic regions. There is convergence of vestibular information on nucleus solitary tract and rostral ventro lateral medullary reticular system. So the vestibular dysfunction can cause effect on these pathways ultimately leading to the symptoms of motion sickness. In accordance to this study Yates et al. in his study has showed that most critical signals required for the generation of motion sickness comes from vestibular system. In addition, conflicting sensory information from different vestibular end organs can induce motion sickness. For e.g. bilateral galvanic stimulation of vestibular nerves which produce signal indicating that the head is simultaneously moving in many directions can elicit emesis in cats.

Since, the vestibular dysfunction is present in individuals with motion sickness it can have other psychological effects such as changes in perceptual- motor strategies. As individuals unconsciously learn to adopt altered modes of postural control or make different use of the perceptual information for orientation, there can be extensive reliance on visual system for postural control leading to recurring disorientation and imbalance in situations where visual information regarding the orientation is misleading or absent. There may also be deliberate alterations in behaviour, such as avoidance of head movements (which stimulate the vestibular system) or disorienting environments. This vicious cycle of self-imposed inactivity which may be caused by fear of movements and environments that might provoke dizziness, in turn will deprive the individual of the opportunity to adapt to these provocative movements and environments. This cycle will make the condition more chronic affecting the quality of life [25]. Thus vestibular dysfunction is predominantly present in individuals with motion sickness.

Clinical Implication

In this study we have found that vestibular system may be compromised in motion sickness individuals. Since vestibular system affection can have effect on vestibular ocular reflex and balance, vestibular rehabilitation can be done in these individuals to reduce their symptoms and improve their balance and VOR gain.

Conclusion

From the study it has been concluded that vestibular dysfunction is predominantly present in individuals with motion Sickness. Thus, vestibular dysfunction and motion sickness are highly associated.

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