



Prevalence and Correlates of Susceptibility to Motion Sickness

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Abstract. Data on susceptibility to motion sickness were collected on a sample of 535 individuals divided into eight groups. The prevalence of motion sickness among Tibetans and Northeast Indians (28%) was slightly higher than Northwest Indians (26%). Generally speaking, females (27.3%) were more susceptible than males (16.8%). Among different groups, the highest incidence of susceptibility to motion sickness (SMS) was recorded in schizophrenic patients (30%), while the lowest in rowers (zero percent). Ears and eyes are the most potent receptors of provocative motion that causes sickness. Individuals with greater spatial and motor control, reflected in sports like rowing, athletics and professions like armed and paramilitary forces, were less susceptible to motion sickness.

The SMS was significantly higher in individuals who suffered from spatial disorientation (35.05%), migraine (26.31%), gastrointestinal disorders (26.82%) and those who were more sensitive to unpleasant odours (24.64%) and preferred sweet flavours (24.48%) than their counterparts. These correlates have been utilized to explain the genesis of sickness using threshold model. Genetic and environmental pathways are strongly advocated. Past episodes of motion sickness acted as a strong psychological attribute in determining further episodes. The roadway buses and trucks proved more effective passive transportation types that caused sickness. The voluntary stabilization of the head and neck movements and gaze control proved very effective natural measures adopted by 38% subjects to avoid or limit motion sickness. About 50% of susceptible individuals became less susceptible or fully acclimatized to motion sickness due to habituation. The mean age at acclimatization was 21.98 ± 5.93 years.

Key words: Motion sickness (MS), Prevalence, Ethnic variation, Correlates.

INTRODUCTION

Motion sickness is a generic term that reflects body reaction to various kinds of motion stimuli caused during passive transportation or by other devices like swings, etc. This sickness may also be evoked by walking while wearing horizontally reversing goggles

both in adults and children [28]. The motion sickness is characterized by symptoms like nausea, vomiting, pallor and cold sweating. These symptoms must have been experienced at least since marine vessels have been invented. It is a syndrome with which perhaps everyone is familiar but amazingly even today its fundamental nature is not completely understood. In particular, it is not clear why it exists at all. It does not only exist in man but in hosts of other species too, e. g. horses, cows, monkeys, chimpanzees, seals, sheep, cats, etc. The literature on the topic is voluminous not because it is very common but because of its implications in space travel, military and aviation industry. A few detailed reviews and books have also been published on the topic [4, 7, 16, 21, 30].

Many hypotheses have been forwarded regarding the etiology of motion sickness. Following the observation made more than a century ago that deaf-mutes are immune to motion sickness [12], vestibular sensory system has been recognised to play the pivotal role in motion sickness causation. However, the concept that the condition is caused by vestibular overstimulation is not tenable for numerous reasons [16, 18].

It is now widely held that condition which may cause motion sickness should not be considered as an isolated vestibular phenomenon but rather as the response of the organism to discordant motion cues [18]. Irwin [11] who is believed to be the pioneer in the field of motion sickness, for the first time proposed the importance of sensory-conflict hypothesis as the principal etiological factor. The theory was further elaborated by Reason [19] in his neural mismatch or sensory rearrangement theory [21]. Reason [20] asserts that motion sickness is a self-inflicted symptom during the course of adaptation to unfamiliar stimulus conditions. Though widely accepted, this theory has been criticized for some of its limitations as it does not explain the biological significance of self infliction in motion sickness and why it occurs, but it does explain where and when it occurs. Watt [32] claims that Reason basically dismisses motion sickness as the coincidental result of technology outrunning evolution, making the question itself meaningless.

There are others for whom signs and symptoms of motion sickness are important features in formulating their hypotheses. Treisman [29] has proposed that vomiting associated with motion sickness actually reflects a mechanism for getting rid of ingested poisons. According to this theory, the basic underlying mechanism is that poison reaches the inner ear which is sensitive to it, hence evoking vomiting to expel whatever toxic material remains in the stomach. Motion is said to merely activate this mechanism. Money and Cheung [17] infer that since bilateral loss of labyrinthine function no longer evokes vomiting in response to injected emetics, the vestibular system is involved in response to poison.

There are number of problems with Treisman's theory [23, 32]. For example, it is probably a bit late to get rid of toxins through vomiting by the time poison reaches the inner ear. Vomiting is the ultimate state of motion sickness and this may be absent in many susceptible individuals. The other features of motion sickness like anorexia, headache, giddiness, etc. do not explain how the toxins are expelled. Some species (e.g. rats) do not vomit as a part of motion sickness though they still become sick. Since they cannot vomit, then why should poisons and motion cause other symptoms [32]?

The above passages indicate wide gaps in our knowledge to fully understand why motion sickness occurs, and why this syndrome affects a wide variety of species, right from pisces to mammals and its absence in many other species. Within species, there

are population differences and some individuals are more susceptibles while some others are immune. There are age and sex differences. Some of these features can certainly be explained with hypothesis of genetic susceptibility to this disorder [22]. In one of the recent reports on teleology of motion sickness, Takahashi et al. [27] observe that motion sickness is an alarm against loss of spatial orientation. Once the spatial orientation is lost, ataxia progresses to a dangerous level unless uncomfortable symptoms appear.

The above review has raised many issues which might be confronted if a large number of studies were conducted on different human populations, on individuals from different kinds of life and professions and on persons suffering from some selected categories of diseases related to brain and sense organs. Keeping these things in mind, the present study has been undertaken.

RESEARCH OBJECTIVES

The following are the main objectives which the present study intends to pursue:

1. to study racial/population differences in susceptibility to motion sickness; to investigate the relative effectiveness of various types of transport/motion in producing sickness and to record relative occurrence of some of the signs and symptoms of motion sickness;
2. in our previous report [22], it was suggested that culture may play some definite role in relative susceptibility to motion sickness. For example, if culture involved extensive participation in sports and sailing in small boats then, some of these populations might present a decreased susceptibility to this syndrome due to their sports and life habits. On this basis, a hypothesis has been framed: the individuals who, by personal choice or cultural reasons, have practised those activities/professions which involve extensive physical and motor activities, may be less susceptible to motion sickness than general population;
3. to confirm whether deaf mutes are immune to motion sickness as revealed by older studies [12]. Since then, revolutionary changes in types of transportation/motion have occurred;
4. to study the incidence of motion sickness in patients/individuals suffering from mental disorders. It is hypothesized that such individuals may be more susceptible to motion sickness because they have poor neural/ sensory control resulting in mismatch of afferent and efferent impulses;
5. to study the relationship of motion sickness susceptibility with variables related to sense of smell, food habits, etc....;
6. since movements of visual field without movement of body are said to cause some of the signs and symptoms of motion sickness [16], blind persons should be less susceptible to motion sickness. This hypothesis is also intended to be tested in this study.

MATERIAL AND METHODS

Study sample

Data were collected on a sample of 535 individuals ranging from 10 to 55 years of age. The sample also included the following eight categories chosen according to the needs of the study design:

1. the control group consisted of 200 individuals (100 males and 100 females). They were normal, healthy, school/college/university students from Chandigarh;
2. the mongoloid group sample consisted of 50 normal healthy students from Tibet/Northeast India. These individuals had mongoloid features like high cheek bone, epicanthic eye fold and straight head hair;
3. the sample of athletes consisted of 50 college/university students. They had participated in various athletic competitions at college/university/state level;
4. the sample of rowers consisted of 35 individuals who were either students or professional rowers of international standing. They belonged to various Asian countries like Korea, Japan, China besides maritime Indian states, like West Bengal and Kerala. They were in Chandigarh in connection with an international rowing competition at Sukhna lake;
5. paramilitary forces have to travel across the whole country for maintenance of law and order. Since India is a vast country, their job requirement includes rough and tough life style and lot of travelling. A sample of 50 individuals was collected from such a police force, Central Reserve Police Force (CRPF);
6. a sample of 50 deaf-mutes was collected from an institute for deaf and dumb at Chandigarh. They all had been suffering from this disease since birth;
7. a sample of 50 blind subjects was collected from their institute at Chandigarh. Some of them were blind since birth, while others lost their sight at the age of 4-5 years due to some accident or disease. Since there were no significant differences in these two groups, they all were treated as a single category;
8. a sample of 50 patients suffering from schizophrenia and/or mental depression was collected from the psychiatry wing of Nehru Hospital, Post-Graduate Institute of Medical Sciences, Chandigarh. These individuals were under medical treatment.

METHODS

Data on susceptibility to motion sickness were collected by retrospective method using standard questionnaires and/or interviews schedules. This method has certain advantages over the other techniques that involve exposing the subjects to fairly rapid changes in motion/acceleration. The former technique covers a wide variety of types and conditions of motion than does the exposure technique. There are several standard questionnaire forms that are available [13, 15, 21]. However, the basic structure of these remains the same. The subject is required to indicate: a) the types of motion that have made him/her

sick, if any; b) the frequency of sickness; c) the severity of symptoms, i.e. nausea alone or nausea and vomiting. This information is used to assign the subject to the broad category of sick. In the present study, this information was recorded by administering interviews to the subjects personally. Deaf-mutes and blind individuals were assisted by their teachers to record the information required.

The recorded data were codified and statistically treated at the Panjab University Computer Centre. Chi-square Test, Spearman rank correlation and maximum likelihood logistic regression analyses [2, 3, 8, 24, 33] were employed for comparison and to study interrelationship between motion sickness and various other variables.

Maximum likelihood logistic regression analysis

It may be assumed that probability of a dichotomous event Y occurring depends upon a vector X of independent variables and a vector β of the unknown parameters. Each Y takes conventionally either 1 or 0 value for occurring or non-occurring of motion sickness event with probability of P and $(1-P)$ respectively. The following log-odds paradigm was estimated using maximum likelihood procedure.

$$\text{Log } (P/1-P) = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \dots + \beta_n X_n$$

The term $\log (P/1-P)$, called logit of P , is odds that Y is one rather than zero, i.e., the term logit of P indicates the log of odds in favour of occurring an event.

The coefficients of linear regression models usually indicate or quantify the marginal change in dependent variable with a unit change in an independent variable. On the contrary, the coefficients in the logistic regression indicate the change in log-odds ratio of the probability of occurring an event upon a unit increase in an independent variable. Thus, the interpretation of logistic coefficients is somewhat cumbersome and perhaps less appealing intuitively because one is not used to think in terms of change in log-odds. Consequently, the raw logistic coefficients have been transformed into changes in probability (ΔP) and elasticity (E) coefficients. ΔP transformation is analogous to an unstandardised regression coefficient. It would indicate the change in the probability of the event occurring as a result of a unit change in the independent variable. Elasticity coefficient (E) would indicate the percentage of change in the probability of an event occurring as a result of one percent increase in the independent variables.

To assess the adequacy of the fitted maximum likelihood (ML) logistic model, three diagnostic tests were used in the present study. For details of these tools, please refer to Anderson [3]; Sivapulle [24]; Lesaffre and Albert [14]. However, a bare minimum of details are presented to facilitate interpretation of the results.

The first diagnostic test used was the likelihood ratio (G^2). It follows chi-square distribution with K (number of functionally independent unknown parameters) degrees of freedom, under the null hypothesis $H_0 = \beta_1 = \beta_2 = \dots = \beta_K = 0$. If the value of G^2 is larger than the table value of chi-square (K) at $\alpha\%$ critical level, then the null hypothesis stands rejected; otherwise it leads to acceptance of null hypothesis.

The second diagnostic test used was the pseudo R^2 that varies between zero and one and its higher value would suggest that the model fits to the observed data.

The third diagnostic test employed was the predictive accuracy or the probability of correct classification (PCC). This statistical test is frequently used in discriminant analy-

sis to test the overall fit of the logit model. Higher values of this test would indicate higher predictive accuracy of the logit model. It is expressed as the percentage of cases accurately classified by the model.

RESULTS

The results on prevalence rates in different categories of individuals are presented in Table 1. The main features of these results are analyzed under the following categories:

Sex differences

Data in Table 1 under category one and two show different results. Northwest Indian females are more susceptible to motion sickness than their male counterparts. This finding is in accordance with the previous reports on SMS [21, 22]. However, it is interesting to note that the category composed by Tibetans and Northeast Indians shows different results. In the latter category, males are more susceptible than females. This finding is contrary to the known features of motion sickness as well as various hypotheses forwarded to explain higher susceptibility among females. For example, women may relatively be less adapted or may show greater sensory response to sickness inducing aspects of motion [21]. However, the rejection of these hypotheses should await more detailed studies on the prevalence of motion sickness in various human populations. Moreover, in the pooled sample, females are significantly more susceptible (27.3%) than males (16.8%); the chi-square value being 8.49 ($P < 0.01$) rejects the null hypothesis of no sex differences in SMS.

Table 1 - Prevalence of motion sickness (MS) in different groups of individuals

Category	N	MS Incidence	Chi-Square
1. Normal (NW Indians)	200	52 (26)	
a) Males	100	20 (20)	8.49*
b) Females	100	32 (20)	
2. Tibetans & NE Indians (Mongoloid-Normal)	50	14 (28)	0.82
a) Males	25	8 (32)	0.40
b) Females	25	6 (24)	
3. Rowers	35	0 (0)	11.58*
4. Athletes	50	9 (18)	1.36
5. Police personnel	50	9 (18)	1.36
6. Blind	50	8 (16)	2.18
7. Deaf-mutes	50	6 (12)	4.38*
8. Schizophrenia / mental depression	50	15 (30)	0.30

* $P < 0.05$; Figures in parentheses are percentages.

Racial or population differences

The results do indicate slightly higher prevalence rate of SMS in Tibetan & Northeast Indian sample compared to Northwest Indian sample. These differences become more acute if we consider both sexes separately. It may be worth mentioning here that under racial classification scheme, Northwest Indian populations are grouped under the broad and composite caucasoid group; while Tibetans and Northeast Indian populations predominantly have mongoloid (Asiatic) features. Stern et al. [26] have pointed out that Asiatic people are more susceptible to motion sickness than American whites and negroids. To tease apart genetic and cultural components, they compared Asian Americans, i.e. the students who were born and brought up in the USA compared to the African and European Americans. But still they found that Asiatic Americans, felt sick faster than the other two groups. They suggested that genes were to be blamed for this higher SMS.

However, the genetic basis of susceptibility to motion sickness had been worked out much earlier than the above report [1, 22]. The results presented in Table 2 also indicate higher prevalence of SMS in relatives of susceptible individuals as compared to non-susceptible subjects. The details on mode of inheritance of genetic SMS are presented elsewhere on the basis of twin and family study [22]. So there is no point in stretching this argument further.

Prevalence of motion sickness in some special categories of individuals

The pivotal role of vestibular sensory system has been recognised since long. Early in 1882, it was reported [12] that none of the 15 deaf-mutes, who had been exposed to rough weather at sea, was found sick. There are a few more scattered reports on deaf-mutes but some of these are not first-hand reports [16]. Consequently we thought of studying the SMS in deaf-mutes and surprisingly the results are contrary to our expectations (Table 1). Of the 50 deaf-mutes, 6 children reported that they had suffered from motion sickness. Though the prevalence rate is just one-half of that in the general population, it is not zero as expected. Perhaps in these individuals the sickness was caused in response to moving visual fields and other perceptions.

If we assume that moving visual field is the only underlying cause of SMS, then its

Table 2 - Family-history of susceptibility to motion sickness in the sample

Relationship with the subject	Number of affected family members among	
	susceptible subjects	non-susceptible subjects
Mother	26 (23)	83 (19.66)
Father	7 (6.2)	29 (6.87)
Sister	16 (14.15)	39 (9.24)
Brother	7 (6.2)	12 (2.84)

Figures in parentheses are percentages.

prevalence should be zero in blind subjects. However, as can be seen in Table 1 blind subjects show different results. The prevalence rate is 16%, though definitely lower than that in the general population.

The prevalence rate of SMS in patients suffering from schizophrenia/mental depression has been found to be slightly higher than the general population, as hypothesized in the present study.

The last group of special categories includes individuals, whose life style/daily routine activities involved tremendous physical activities. This group consists of three categories. The first one is that of rowers. These individuals belonged to maritime states of India and Southeast Asia. Their culture involved a lot of swimming and sailing in small boats and these individuals were exposed to such culture right from their early childhood. Surprisingly, none of thirty five such rowers ever suffered from motion sickness.

The other two categories included in the above group are athletes and police personnel from Central Reserve Police Force (CRPF). In these two groups, the prevalence rate of SMS is definitely lower than that in general population. None of the police personnel ever suffered from motion sickness in buses or cars etc. But some of them suffered when the motion was very rough and done in trucks on difficult and zig-zag terrain and involved long drives, while few others suffered during their journey in steamers.

Some select determinants of motion sickness

A large number of constitutional, behavioural and environmental factors, both normal and abnormal, may influence susceptibility in the presence of provocative motion. The results on the association of such select factors with motion sickness are presented in Table 3. The chi-square test is significant for the following: migraine, spatial disorientation, acrophobia, sensitivity to unpleasant odours and choice of food taste. The results on the rank correlation analysis are presented in Table 4. The correlation coefficients are significant at 5% level of probability for all the traits/conditions listed in the table except eating habits and going up/down the hill.

The relative effectiveness of the type of transport/motion that causes motion sickness

The results on relative effectiveness of the type of transport/motion are presented in Table 5. The most effective producers of nausea and vomiting appear to be buses, trucks and ships. The least effective producers are small boats and trains.

The relative incidence of nausea and particularly vomiting depends to a large extent upon whether or not the motion stopped at will. Motion sickness in a car as compared to roadways buses can be reduced or cured by getting the former stopped at will among mild and moderate susceptibles. This seems to be the reason for relatively lower incidence of motion sickness during car journeys.

The passive vehicular motion usually causes movements of the head relative to the body, so that the head and vehicle move differently with respect to inertial space.

Effect of hilly terrain and food habits on motion sickness

Travelling on hilly terrain is bound to cause more anxiety to the susceptible individual as the motion involves sharp turns, going up or down the hill. The results of these investi-

Table 3 - Relative incidence of motion sickness in relation to presence or absence of some specific disorders/traits

Condition/Factor	N	Motion sickness Incidence	Chi-Square
<i>Migraine</i>			
a) Present	57	15 (26.31)	3.81*
b) Absent	478	90 (20.50)	
<i>Spatial disorientation</i>			
a) Present	16	7 (43.73)	5.07*
b) Absent	519	106 (20.42)	
<i>Acrophobia</i>			
a) Present	129	46 (35.05)	22.13*
b) Absent	363	58 (15.90)	
c) Unknown	43	9 (20.90)	
<i>Handedness</i>			
a) Right	520	111 (21.35)	0.56
b) Left	15	2 (13.34)	
<i>ABO blood group</i>			
a) A	61	14 (22.90)	1.39
b) B	123	30 (24.30)	
c) AB	14	3 (21.40)	
d) O	118	23 (19.47)	
e) Not known	219	43 (19.63)	
<i>Vision defect</i>			
a) Present	140	32 (22.80)	0.34
b) Absent	395	81 (20.50)	
<i>Gastro-intestinal disorder</i>			
a) Present	82	22 (26.82)	1.89
b) Absent	453	91 (20.08)	
<i>Sensitivity to smell/unpleasant odour</i>			
a) Present	426	105 (24.64)	5.60*
b) Absent	109	8 (7.33)	
<i>Sensitivity to petrol/diesel smoke/odour</i>			
a) Present	369	89 (24.11)	6.44*
b) Absent	166	24 (14.45)	
<i>Choice of food taste</i>			
a) Sweet	237	58 (24.48)	6.41*
b) Salty	246	48 (19.50)	
c) Both	52	7 (13.46)	

* P < 0.05; Figures in parentheses are percentages.

Table 4 - Rank correlation coefficients (r_s) between susceptibility to motion sickness and its probable determinants

Correlate/variable	r_s
Sex	0.30*
Occupation	0.47*
Categories	-0.39*
Handedness	0.65*
Blood group	0.22*
Vision defect	0.40*
Hearing defect	0.55*
Gastro-intestinal disorder	0.54*
Sensitive to smell/unpleasant odour	0.54*
Sensitive to petrol or diesel smoke/odour	0.50*
Choice of food taste	0.35*
Eating habits	0.08
Migraine	0.46*
Spatial disorientation	0.64*
Acrophobia (fear of heights)	0.43*
Family history	0.33*
Type of transport	0.57*
Going up or down the hill	0.04
Past episodes of motion sickness	0.78*

* $P < 0.05$.**Table 5 - The relationship between type of transport/motion device and incidence of motion sickness in the susceptible individuals**

Kind of transport / motion device	No. of exposures			Sickness feeling only			How often vomited	
	0	1	2	3	4	5	4	5
Bus	0	9	104	12	6	4	48	43
Car	10	8	95	71	3	4	12	13
Train	5	16	92	100	5	1	2	0
Merry go round	34	56	23	58	5	3	6	7
Small open boat	58	36	19	52	1	0	2	0
Roller skating	12	32	9	27	7	4	1	2
Aeroplane	105	8	0	4	2	1	0	1
Truck	107	0	6	—	2	1	1	2
Steamer / ship	110	2	1	0	0	0	2	1

0 = No experience; 1 = Less than 10 trips but more than 1 trip; 2 = More than 10 trips;
 3 = Never; 4 = Sometimes; 5 = Frequently / Always.

gations are presented in Table 6. In susceptible individuals, severity of symptoms increases in 8% of the individuals while going up the hill, in about 18% while going down and in majority of individuals the condition is grave both ways. Of the 113 susceptible individuals, 28 individuals suffered from motion sickness only when the motion was rough, on hilly terrain or long journeys. If these cases are excluded then the overall prevalence rate of SMS is reduced to 15.9% from 21.1%.

Eating habits have also been reported to play some role in precipitating vomiting (See Table 6). Heavy, fried foods have been listed as the greatest precipitating agents by the majority of susceptible individuals.

Signs and symptoms of motion sickness

Table 7 lists the relative frequency of various signs and symptoms of motion sickness. Pallor, cold sweating, giddiness, nausea and vomiting are the most common signs. Cold sweating and pallor generally precede nausea and nausea always precedes vomiting.

Besides these, there are many other signs and symptoms related to cardio vascular, respiratory and gastrointestinal systems. The relative proportions of these as told by the subjects are shown in Table 7.

The susceptible individuals also manifest differential variability in the time taken to recover from the ill effects of motion sickness (Table 7). About 45% of the individuals recovered within ten minutes of removal of motion stimulus. But there are a few individuals who even took more than a day to recover fully.

Treatment and adaptation

Since avoiding motion disorders is not a practical proposition in many situations, the susceptible individuals resort to many ways to improve and avoid sickness. Table 7 lists some of such measures, besides data on adaptation status and age at acclimatization in the susceptible individuals.

There are three types of voluntary motor control measures, such as, postural changes,

Table 6 - Effect of eating habits and type of hilly terrain in triggering motion sickness

	N	%
A. <i>Eating habits before travel</i>		
Heavy and fried food	71	62.83
Empty stomach	30	26.50
Both empty and full stomach	12	10.61
B. <i>Type of hilly terrain</i>		
While descending the hill	9	7.96
While ascending the hill	43	38.05
Both while ascending and descending the hills	20	17.69
No experience	41	36.28

Table 7 - Distribution of signs and symptoms of motion sickness and time taken to recover

Sign / Symptoms	N	Percentage
A. <i>General signs</i>		
Cold, sweating & pallor	56	49.56
Dizziness or giddiness	42	37.17
Nausea only	14	12.39
Vomiting	99	87.61
B. <i>Cardiovascular and respiratory signs</i>		
Increase of pulse rate	10	8.84
Low pulse rate	8	7.07
Increased ventilation	5	4.42
Slow respiration rate and shallow breathing	3	2.65
No change noticed or uncertain	87	77.00
C. <i>Gastrointestinal signs</i>		
Gas or belching	17	15.03
Salivation	74	65.50
Swallowing problems	2	1.76
No effect	20	17.69
D. <i>Recovery time</i>		
Less than 10 minutes or at cessation of stimulus	51	45.12
Less than 1 hour	60	53.18
More than 1 hour	26	23.00
More than 1 day	2	1.76
Never noticed	25	22.12

stabilization of head and neck and gaze control by closing eyes, that have been reported to help control/limit the symptoms. These measures have been adopted perhaps through experience or cognition. These adaptive responses helped 38% of susceptible individuals to control sickness.

It is a known feature of motion sickness that habituation or continued exposure to motion stimuli result in declining sickness response in most individuals [16]. The present study also corroborates these results (Table 8). About 50% of the individuals have become acclimatized or less susceptible over the years. There is no change in another 40% of individuals. But surprisingly 10% of them became more sick. The mean age at acclimatization is 21.98 years with one standard deviation of 5.93 years.

Logit analysis

The results of logit analysis are presented in Table 9. The estimated coefficients are significant only for five out of eleven correlates: sex, sensitive to unpleasant odour, spatial disorientation, acrophobia and category. Females are definitely more susceptible than males. The negative signs indicate that with decrease in sensitivity to unpleasant odour,

Table 8 - Treatment and adaptation

	N	Percentage
<i>A. Effective steps to control motion sickness</i>		
Posture	10	8.84
Restriction of head and neck movements	14	12.38
Gaze control (by closing eyes)	19	16.81
Medicine	43	38.05
Diet Control	11	9.73
No step taken	16	14.15
<i>B. Adaptation status</i>		
No change	45	39.82
Less susceptible	32	28.32
More susceptible	12	10.62
Immune / Acclimatized	24	21.24
<i>C. Age at acclimatization</i>		
Before 12 years	2	1.77
Between 12-19 years	12	10.62
After 19 years	10	8.85
Not acclimatized	89	78.76
Mean age at acclimatization	24	21.98 \pm 5.93

spatial disorientation, presence of acrophobia, the susceptibility to motion sickness also decreases. The relationship between category and motion sickness indicates that as you depart from normal population to special categories like the blind, deaf-mutes, rowers, and athletes, etc., the susceptibility to motion sickness (SMS) decreases because these factors are negatively correlated with SMS. The diagnostic statistics like G^2 clearly indicate that contribution of each independent variable is not equal. The other statistics like Pseudo R^2 and PCC confirm that the model used is perfectly valid to explain the underlying variability of motion sickness.

DISCUSSION

These results present new challenges to biomedical anthropologists and human biologists since the field has not received hitherto their attention. Some of these results are interesting but look intriguing in the light of existing knowledge. For example, it has been observed here for the first time that right handed individuals are more susceptible to motion sickness than their left handed counterparts. This situation is curious in the light of observations made by Dupont et al. [9]. They have reported that many areas (regions) responsive to motion are situated in the right hemisphere of the human brain. These two incompatible situations can be explained if we assume that the hypothesis of cerebral laterality may not be true as far as handedness is concerned. This is in fact so as many other reports have challenged cerebral lateralization hypothesis in determining handedness [5,

Table 9 - Determinants of motion sickness: Logit analysis

Variable	Estimated Coefficient	Standard error	Delta P	Elasticity	Diagnostic statistics		
					G ²	Pseudo R ²	PCC
<i>Set 1</i>							
Sex	0.59*	0.23	0.11	16.35			
Vision defect	-0.22	0.41	-0.05	-7.29			
Hearing defect	0.80	0.46	0.14	21.05			
Gastro-intestinal disorder	-0.07	0.29	-0.02	-2.22			
Sensitive to smell/unpleasant odour	-1.39*	0.44	-0.33	-48.81			
Sensitive to petrol/diesel smoke/vapour	-0.03	0.29	-0.01	-1.04			
Migraine	0.11	0.36	0.02	3.40			
Spatial disorientation	-1.30*	0.57	-0.31	-45.80			
Constant	0.40	1.78	0.08	0.13			
					662.27*	0.56	78.88
<i>Set 2</i>							
Choice of food taste	-0.30	0.17	-0.04	-0.39			
Acrophobia	-0.71*	0.20	-0.09	-1.04			
Constant	0.42	0.44	0.08	0.34			
					644.24*	0.55	78.88
<i>Set 3</i>							
Category	-0.09	0.05	-0.01	-0.24			
Constant	-1.02	0.18	-0.12	-0.81			
					631.67*	0.54	78.88

* P < 0.05.

10]. They argue that hand preference may be determined by many factors such as intrauterine environment, in particular testosterone and estrogen levels in conjunction with their receptors in the brain. It is also likely that a strong genetic component and post-natal factors, including social pressures, exercise powerful modifying influences on several expressions of cerebral lateralization [6, 10]. However, this association of right handedness with motion sickness should await confirmation till more data on this aspect are reported.

Factors which are critically associated with motion sickness *inter alia* include loss of spatial orientation, mental sickness and acrophobia. From these results, it may be hypothesized that those individuals whose sensory control systems fail to initiate adaptive steps in response to stress caused by provocative motion in the form of mismatch signal are more susceptible to motion sickness.

In another situation under this hypothesis, we would expect lower SMS in individuals whose sensory and motor controls are very efficient. For example, athletes including gymnasts and rowers are expected to be less susceptible under the model, and our results are compatible with this hypothesis. This greater motor control may be genetic and/or

environmental including cultural component. Moreover non-susceptible individuals rarely suffer from spatial disorientation or acrophobia. Since cerebellum and brain stem control above sensory organs, we can assume that these organs play critical role in determining motion sickness. These observations draw strength from the report of Stephan et al. [25] and other referred physiological studies which have shown that human brain controls sensory-motor function and parietal areas are associated with spatial aspects of motor planning. The comparative data on prevalence of motor sickness in deaf-mutes and blind vis-a-vis their normal counterpart indicate that ears and eyes act as key receptors of provocative motion.

The above account clearly points out that the impairment of spatial equilibrium is an important pathways in the genesis of sickness. Similar conclusions have been drawn by Takahashi et al. [27]. They have even gone to the extent of defining motion sickness as, "mixture of autonomic nervous symptoms, ataxia and dizziness or vertigo resulting from impaired spatial orientation". They further said that once spatial orientation was moderately impaired, subjects could no longer walk and complained of sickness, perceived dizziness and exhibited ataxia. This attribute also explains the higher incidence of motion sickness while travelling on hills. In hilly terrains, occupants are exposed to a complex pattern of linear and angular movements resulting in spatial disorientation much faster than plains.

There are many other factors which can trigger the process of motion sickness. Motion sickness would occur in susceptible individuals much faster when an attempt is made to maintain postural stability in sitting upright position and head and neck movements are not restrained. Conversely, motion sickness can be avoided by lying in supine position and/or restraining the head and neck movements. Greater head movements result in greater vestibular stimulation and hence greater susceptibility. Another interesting feature is that any attempt to read newspaper during vehicular journey by a susceptible individual would trigger sickness symptoms and consequently affected individuals find it difficult to read because of spatial disorientation. The mismatch in this situation probably lies in the disparity of oculomotor efference with afferent information from the retina and ocular muscles [18].

The results have further revealed many other precipitating factors, like anxiety due to past episodes of motion sickness, gastro-intestinal disorders, food habits, sensitivity to unpleasant odours. These various determinants of motion sickness have been summarized in Figure 1 which shows various pathways of motion sickness genesis. The neural mismatch theory provides a conceptual framework for the classification of different types of sensory conflicts associated with nauseogenic motion stimuli in vehicles and laboratory devices. Detailed tabulations of mismatching visual, vestibular and proprioceptive sensory cues have been provided by several authors [18]. In the above model, it is necessary to introduce a threshold level in the pathway to account for individual variation in susceptibility to motion sickness. Hereditary and environmental factors are also important determinants of motion sickness. Many studies have confirmed the role of hereditary factors in sensory variables like audition, perception of certain tastes, perhaps smell and pain perception [31].

We have discussed in detail how and when motion sickness strikes. But why do we get motion sickness? Moreover, this is a phenomenon that has considerable evolutionary significance.

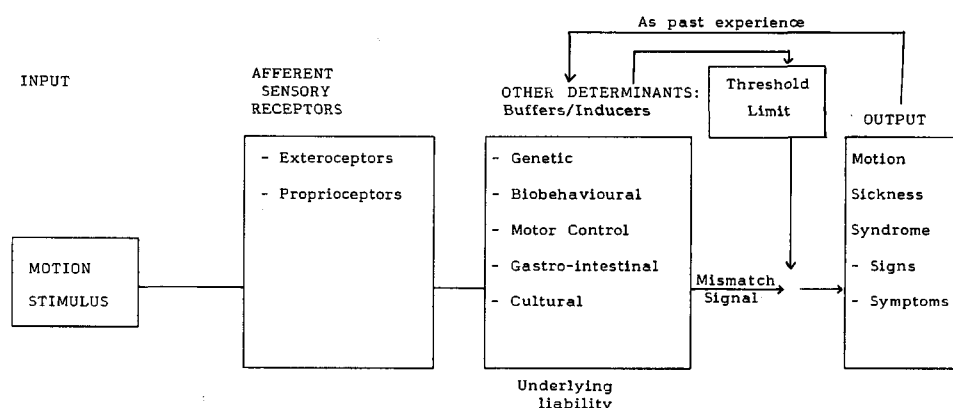


Fig. 1 - Genesis of motion sickness syndrome

According to Watt et al. [32], motion sickness serves as a warning against inappropriate motor strategies which are causing undesired changes in vestibular function, and subsequent disruption of normal sensori-motor integration. Thus, motion sickness may have definite purpose. Dwelling on this aspect, Takahashi et al. [27] state that this warning system not only evokes appropriate autonomic nervous symptoms, but also freezes regulation of action. The various symptoms of the motion sickness serves as a mechanism to warn animals including man that a situation is becoming dangerous with the loss of spatial orientation. And the present study equivocally supports these contentions, besides offering new ideas relevant to the etiology and variability of motion sickness.

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