Chapter 8 – Seasickness: Guidelines for All Operators of Marine Vessels, Marine Helicopters and Offshore Oil Installations

by

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This review is written for the NATO lecture series: NATO RTA "Survival at Sea for Mariners, Aviators and Personnel Involved in Search and Rescue". It provides a brief historical perspective, updated scientific information on the phenomenon, susceptibility and management of seasickness. It is designed to provide the latest data on seasickness for all those who earn their living working on or flying over the water.

INTRODUCTION

Many different forms of transport, from surface vehicles (land and sea) to air and space vehicles, cause motion discomfort with symptoms ranging from nausea to vomiting and/or retching in susceptible individuals. These symptoms are collectively known as motion sickness. The most dreaded kind of motion discomfort occurs on long duration voyages where the susceptible individuals often feel that they are effectively imprisoned in the nauseogenic environment. Seasickness is the most widely experienced form of this oppressive motion

sickness. Reports of seasickness, also known as "kinetosis" and "naupathia" date back as far as the authors of Greek mythology, who were familiar with the discomforts associated with seasickness. It seems likely that humans suffered from seasickness well before they could make written records of it. Lord Nelson was a chronic sufferer of seasickness, even on his final voyage to fight at Trafalgar. Sir Charles Darwin never missed a chance to get off the boat during his famous voyage on the Beagle because he too succumbed to seasickness.

Irwin [68] recorded the term "motion sickness" as follows:

> Seasickness, or motion sickness, as it might be more correctly named – for not only does it occur on lakes and even on rivers, but, as is well known, a sickness identical in kind may be induced by various other motions than that of turbulent water – is essentially a disturbance of the "organs of equilibration"

Usage of the term "motion sickness" was popularized by Sir Frederick Banting during the Second World War when seasickness and airsickness were studied together. However, with the advance in knowledge and technology, the term "motion" is a misnomer as the symptom characteristics can be evoked as much by the absence of expected motion as by the presence of unfamiliar or apparent conflicting motion. For example: simulator sickness and cyber sickness (sickness induced by computer generated virtual displays) are examples of conditions where the evocative stimulus is the absence of physical motion stimuli and the presence of visually induced apparent sensation of self motion. The term "sickness" is also a misnomer as it carries a connotation of "(affected with) disease". It obscures the fact that motion sickness or seasickness is a normal physiological response of a healthy individual without organic or functional disorder, when exposed to unfamiliar or conflicting motion of sufficient severity for a sufficient period of time. **Hence, seasickness and other associated forms of motion sickness (airsickness, carsickness, simulator sickness and space sickness) can now be defined as a maladaptive response to real and apparent motion**. However, it should be noted that visually induced sickness comprises a number of motion sickness-like signs and symptoms, with slightly different profiles from true motion sickness. Visually induced sickness is generally less severe, but the after-effects (flashback from cyber sickness) can appear much later after the initial exposure. Therefore it is important to distinguish the stimuli that were used when evaluating the results of laboratory studies on the effects and countermeasures of motion sickness.

SIGNS AND SYMPTOMS

The cardinal signs of seasickness are pallor and/or flushing in the facial area, cold sweating, vomiting or retching. Facial pallor arises from the constriction of surface blood vessels. Sweating often occurs even when the thermal conditions would not make this necessary. The cardinal symptom of motion sickness is nausea; often, it is a precedent to vomiting. Vomiting can sometimes occur without nausea. The physiological mechanism of vomiting and retching is identical except that vomiting involves the forced expulsion of stomach contents and psychologically it is more gratifying afterwards, as it usually provides a rapid relief from nausea. However, retching is unproductive (no expulsion of stomach contents) and usually the feelings of malaise linger for a while. There are other signs and symptoms associated with motion sickness.

They commonly occur in an orderly sequence as follows:

- Stomach (epigastric) awareness;
- Stomach discomfort;
- Pallor;

- Cold sweating;
- Drowsiness;
- Yawning;
- Feeling of bodily warmth;
- Increased salivation;
- Nausea; and
- Vomiting/retching.

The common after-effects with motion of long duration [99] are:

- Persistent headache (especially frontal);
- Apathy;
- Lethargy;
- Anorexia;
- General malaise;
- Persistent dizziness;
- Light-headedness or disorientation;
- Belching/flatulence; and
- Feeling miserable or depressed.

In addition, there is a symptom complex known as Sopite Syndrome that is centering on irresistible drowsiness. The typical symptoms of Sopite include:

- Frequent yawning;
- Drowsiness;
- Disinclination for work, both physically and mentally; and
- Avoidance of participation in group activities.

Generally, the symptoms characterizing this syndrome are interwoven with other symptoms, but under two circumstances this syndrome may become the sole overt manifestation of motion sickness:

1) When the intensity of the eliciting stimuli is closely matched to a person's susceptibility and the syndrome is evoked either before other symptoms of motion sickness or in their absence; and
2) During prolonged exposure in a motion environment when adaptation results in the disappearance of motion sickness symptoms except for responses characterizing the sopite syndrome.

In general, the time scale for the development of motion sickness symptoms is determined primarily by the intensity of the stimulus and the susceptibility of the individual. Therefore, individuals vary in their response: for instance, certain individuals may experience many of the above effects, feeling ill for a considerable amount of time, but they may not vomit; others may have a relatively short warning period (few signs and symptoms), vomit and feel better almost immediately. The rapid relief is partially attributable to the fact that salivation, stomach disturbance, respiratory and heart rate changes are also part of the organized chain of events that comprise the act of vomiting. If exposure to the motion continues, nausea increases in intensity and

results in vomiting or retching. In more susceptible individuals, the cyclical pattern may last for several hours or in extreme cases, for days. Dehydration and disturbance of electrolyte balances in the body brought about by repeated vomiting compounds the disability.

The pathognomonic signs of vomiting and retching in visually induced sickness (non-motion or limited motion based simulator) are rare [15], while other overt signs such as pallor; sweating and salivation are more common. However, vomiting appears to have a sudden and sometimes unexpected onset, and occurs often without accompanying prodromal symptoms. Postural changes have also been observed immediately after simulator flights. As briefly earlier, the more serious problems associated with simulator sickness are residual after-effects which have been documented by a number of studies [5, 36, 74, 91, 129] including illusory sensations of climbing and turning, perceived inversions of the visual field and disturbed motor control.

AETIOLOGY OF MOTION SICKNESS

The combination of sensorimotor systems involved in bringing about the onset of motion sickness and in the maintenance of spatial orientation awareness is identical. It involves the visual, vestibular (organ of balance) and the somatosensory receptors (tactile cues and non-vestibular proprioception such as joint receptors, Golgi tendon organs and muscle spindles). The vestibular apparatus is incriminated in motion sickness and visually induced sickness. Labyrinthine-defective individuals are immune to motion sickness [51, 75, 116, 117] and visually-induced sickness [15]. Sudden unilateral loss of vestibular function gives rise to symptoms of motion sickness including vomiting. More than a century ago, Irwin [68] suggested that sensory conflict (where sensory signals from the eyes and the organ of balance do not agree) was the principal cause of motion sickness. However, the prescribed conflict is not limited to signals from the visual system, the vestibular system and somatosensory receptors. These signals are also at variance with the information that the central nervous system expects to receive [31, 112]. Therefore, the conflict theory of motion sickness holds that in an environment conducive to motion sickness, the pattern of sensory inputs concerning orientation and motion is in conflict with the pattern of inputs anticipated on the basis of past experience.

This theory of a simple conflict causing sickness is insufficient, as it does not explain habituation to provocative stimuli or the after-effects of exposure to such stimuli. It does not explain why such a conflict should produce vomiting. Nevertheless, the sensory conflict theory is satisfactory as all known causes of sickness can be accommodated by this theory and it suggests some useful preventive measures. A further modification suggested that conflicting sensory inputs are interpreted centrally as neurophysiological dysfunction caused by poisoning [124] and that some evidence concerning the basic validity of this “poison” theory was provided by Money and Cheung [98] and Ossenkopp, et al. [106]. Watt [132] suggested that motion sickness is significant as it serves as a warning against inappropriate motor strategies that are causing undesired changes in vestibular function, and the subsequent disruption of normal sensorimotor integration. The ability of the human sensory system to resolve the motion experienced depends on the frequency of oscillation because the different senses do not all respond to the imposed acceleration. However, the severity of the signs and symptoms of motion sickness increases as a function of exposure time and acceleration intensity.

FREQUENCY OF MOTION AND SUSCEPTIBILITY

Early empirical and experimental observations suggested that vertical motion (heave) is the predominantly nauseogenic stimulus in the normal gravity (1G = 9.8 m/s^2) environment. Data concerning the frequency of motion and motion sickness are derived from early surveys conducted at sea relating passenger seasickness questionnaire responses to their exposure to linear and angular motion. It concluded that vertical oscillation

(heave motion) was the best predictor of motion sickness and that other linear (horizontal) or angular motions (roll, pitch and yaw) were less significant [82]. Because of the high inter-correlation between various types of ship motion, these surveys could not distinguish the separate contributions of each type of motion. As a result, early controlled laboratory studies employed largely vertical linear oscillation as the primary stimulus to provoke motion sickness and there were no controlled data relating frequency of motion to the nauseogenicity of horizontal motion until much later by Golding and Markey [47].

For example, laboratory studies using vertical oscillation showed that sickness increases with decreasing frequency to at least about 0.2 Hz. Alexander [3] used a modified elevator to expose seated, blindfolded subjects to motion at frequencies of 0.22, 0.27, 0.37 and 0.53 Hz (magnitudes ranging from 1.96 to 5.47 m/s^2) for 20 minutes; there was a significant increase in nauseogenicity as frequency decreased. Their results also suggested that increases in motion magnitude did not necessarily increase the incidence of vomiting. O'Hanlon and McCauley [104] and McCauley, et al. [89] investigated the responses of over 500 subjects seated with their heads against a backrest with eyes opened in an enclosed cabin that oscillated vertically. Subjects were exposed to five frequencies 0.167, 0.25, 0.33, 0.5 and 0.6 Hz and various magnitudes from 0.278 to 5.5 m/s^2 RMS for a maximum of 2 hours. The highest percentage of vomiting was at 0.167 Hz and the incidence of vomiting decreased gradually towards 0.3 Hz and a more rapid decrease with higher frequency. There was limited evidence suggesting that motion sickness incidence further decreased at frequencies below 0.167 Hz. Lawther and Griffin [81] conducted a similar study, measuring the motion of car ferries operating in the English Channel and the consequent sickness among passengers. Data were analyzed for 17 voyages of up to 6 hours in duration, involving over 4900 passengers. The results were similar to those of O'Hanlon and McCauley in that the strongest correlations between motion sickness incidence and motion were in the vertical (heave) direction, both in magnitude and duration of exposure. In addition, position aboard a vessel is a significant factor in how the subjects perceive a given motion. Data collected by Lawther and Griffin [81] were from very large passenger ships that typically have relatively small pitch and roll movements. An incident wave tends to excite vertical oscillation (heave) of the vessel at its natural frequencies of buoyancy. It will excite the hull over a range of frequencies in heave, roll and pitch in which the vessel is compliant. In most vessels this happens most often at vessel motion frequencies of 0.1 to 1 Hz, which is particularly nauseogenic to humans.

It is not surprising that the traditional view that vertical motion is the principal stimulus for vibration induced motion sickness has been challenged. Wertheim, et al. [133] suggested that pitch and roll when combined with small heave motion, which in themselves are not sickness provoking, produce more motion sickness than claimed by the classic model. The motion parameters were: heave frequency at 0.1 Hz (with RMS between 25 and 32 cm; G between 0.02 and 0.035) pitch frequency at 0.08Hz (with RMS from 4.9° to 9.9°, G between 0.01 and 0,022), roll frequency between 0.05 and 0.07 Hz (with RMS between 7.1 and 9.9°; G between 0.003 and 0.014). Frostberg [42] investigated motion sickness occurrence in a group of 40 subjects exposed to 7 different combinations of lateral and roll oscillation and reported that combined roll and lateral oscillation caused greater sickness than either roll oscillation or lateral oscillation alone.

In general, the larger the vessels, the less likely seasickness will afflict the ship's complement at a given sea state and condition [135]. However, large mobile drilling platforms and super-tankers of immense displacement and dimensions with high structural flexibility and low inherent structural damping can exhibit vibrations frequencies below 2 Hz. Naval vessels such as light cruisers and destroyers tend to heave, pitch and roll at frequencies of 0.13 to 0.33 Hz that are particularly nauseogenic. Because of the heave component of the composite motion of the vessel, susceptibility to seasickness can be shown to increase monotonically as a simple geometric function of the lateral distance of the subject from the effective centres of rotation of the vessel [11]. Smaller vessels such as Coast Guard patrol boats, passenger and pleasure craft can experience

violent motion that include abrupt yawing and large amplitude roll, pitch and heave in severe weather that will provoke motion sickness as well as shipboard injury.

PERFORMANCE

There are three general aspects of major importance to the problem of human performance degradation in a moving environment:

1) Motion sickness incidence (MSI);
2) Motion induced interruptions (MII); and
3) Motion induced fatigue (MIF).

MSI and MII will interfere with task performance due to sickness symptoms and the loss of balance. MIF caused by added muscular effort to maintain balance can interfere with cognition or perception, especially in long duration tasks.

As eluded previously, true manifestations of seasickness share an underlying physiological mechanism and definable frequency range of oscillatory motion that is provocative. In addition to signs and symptoms of motion sickness, there are also documented changes in behaviour and performance such as: loss of well-being, distraction from task, decreased spontaneity, inactivity, being subdued, decreased readiness to perform and decreased muscular and eye-hand coordination. Other serious related problems have been documented, sometimes without overt sickness. For example, spatial disorientation, sleep disturbance, postural disequilibrium, mal de débarquement and altered gaze reflex that will affect visual acuity [45, 49, 59, 60, 77, 80]. Perhaps the greatest impact of seasickness in the operational environment is maintaining effective watch-keeping. The functional ability of all marine vessels is degraded in severe weather conditions, primarily due to the adverse effects of ship motion on crew performance. From the crew's perspective, loss of well being interferes with the ability to perform task and can become a liability to others as well. Seasicknesss can also affect the ability of passengers (troops) to carry out duties immediately after landing. The sight and smell of vomitus in a confined space can affect morale. It has been reported that severe seasickness erodes the will to survive and the affected individuals are less able and less willing to take positive action to aid survival.

Commercially, FPSO (Floating Production Storage and Offloading) vessels are increasingly being used to operate in deep water where the operating environment can be very extreme. The crew on these vessels must often work under extreme weather conditions, in shifts throughout the day and night for up to three weeks at a time, or even longer if the weather prevents crew changes. Seasickness and its after-effects, motion-induced fatigue and motion-induced interruptions are a potential problem for the safety and health of crewmembers at sea. A questionnaire-based survey [22, 25] based on 2255 returned questionnaires revealed that the crew complained of a variety of problems including:

- Sleep disturbance;
- Task completion;
- Task performance;
- Loss-of-concentration;
- Decision-making; and
- Memory disorders.

Specifically, the correlation between sleep disturbance and ship motion was relatively high. There were relatively few complaints of seasickness. These results are consistent with the findings by Colwell [33] in a NATO sea trial. The results suggested that significant correlation between fatigue and cognitive performance is at least partly influenced by ship motion effects on sleeping and low level of motion sickness. There appears to be no apparent habituation among subjects who participated in more than 2 shifts offshore. In general, it is apparent that the number of safety, health and performance issues increases with the deterioration of weather conditions.

MOTION SICKNESS AND INDIVIDUAL SUSCEPTIBILITY

Reason and Brand [112] suggested three relevant characteristics of an individual that might affect susceptibility to motion sickness: receptivity, adaptability, and retentiveness. Receptivity is defined as the initial reactivity, the internal amplification or the range of motion stimuli that produce a response. In other words, motion sickness is more of a problem to a receptive individual. Adaptability is the ability to adapt to the motion and to reduce sickness symptoms. It is suggested that those who report a greater history of problems with motion sickness tend to adapt more slowly to novel motion. Retentiveness is the ability to retain the adaptation during abstinence periods, and ability to reinstate adaptive responses upon re-exposure to the motion. It is the ability to retain the internal model of motion and to adapt to the same stimulus in successive exposures. The greater the retentiveness the less chances of sickness in subsequent exposure to the same motion. Genetically, susceptibility to motion sickness was reported to be one of the significant differences in concordance between monozygotic and dizygotic twins [2]. A single nucleotide polymorphism of the α 2-adrenergic receptor increases autonomic responsiveness to stress induced by off-axis rotation at increasing velocity [41]. Volunteers with the 6.3-kb allele had greater signs and symptoms of motion sickness mediated by the autonomic nervous system. However, it is unclear whether this is a marker for motion sickness susceptibility, per se, or a general marker for autonomic sensitivity. A recent postal survey conducted in an age-matched sample of monozygotic and dizygotic adult female twins [113] indicated that 40% of respondents reported moderate susceptibility to motion sickness. The pattern of responses among twins suggested a significant genetic contribution.

AGE AND SUSCEPTIBILITY

Early scientific literature suggested that susceptibility fluctuates with age [30, 97, 128]. Infants below the age of 2 are generally immune. Susceptibility appears to be at its highest level between 2 and 12. There is a significant decline between the ages of 12 and 21 [111]. However, a longitudinal study in the squirrel monkey (with a typical life span of 13 – 15 years) indicated that there were no significant differences in the susceptibility level (as measured by latency to vomiting and retching and cumulative sickness scores) throughout a 10-year period [16].

GENDER AND SUSCEPTIBILITY

There were a number of survey questionnaires and subjective reports that involves large sample of population suggested that females are more susceptible to seasickness [1, 34, 56, 81, 83, 102, 127]; airsickness [84]; short haul flights [127]; trains [71]; carnival devices, performing gymnastics [85, 111]; and military flight simulators [76]. However, other reports suggested that there were no difference between gender in seasickness susceptibility [87], during coach journey [125], visually induced sickness [69, 107] and vestibular Coriolis cross-coupling induced sickness [23]. It was explained that female are more susceptible to motion sickness

because of the hormonal fluctuation especially during the menstrual cycle. The origin of this explanation can be traced to an early observation made by Schwab on one female. Schwab [114] described "… a nurse in the army medical corps who successfully crossed the Atlantic on a small vessel during rough weather without being ill, but who became nauseated and vomited in calm sea of the Mediterranean when her menstrual period began". Grunfeld and Gresty [58] observed a slight increase in the number of sickness events reported during premenstrual and permenstrual phases. Cheung, et al. [21] reported that the menstrual cycle appears to have no influence on subjective symptoms and cutaneous blood flow increases under controlled laboratory conditions. In addition there is a lack of commonality between the types and levels of hormones that are released during motion sickness and those that are involved in different menstrual phases. Clemens and Howarth [32] reported that susceptibility to virtual simulation sickness varies over the menstrual cycle as a consequence of hormone al variation. Using a relatively mild provocation of a video game, only the most susceptible part of the population would have experienced significant motion sickness. In addition, the stimulus dose of a video game is difficult to control as it is under the control of the subject to a great extent. A later study by Golding, et al. [48] using a staircase profile Coriolis cross-coupling stimulus suggested that there was a small but significant trend that motion sickness susceptibility was maximal at day 5 and decreasing through days 12 and 19 to a minimal at day 26 premenstrual of the menstruation period. A lengthy staircase profile of successive motion exposures may introduce a possible confounding habituation effects. The authors suggested that it is unlikely that hormonal fluctuation account for the greater susceptibility in women since the magnitude of the fluctuation is only about one-third of the overall difference between male and female susceptibility (based on previous surveys). The variance in these laboratory findings is similar to the different results obtained by surveys studies as stated above. It might be that there is no reliable result of the menstrual cycle on motion susceptibility and that it is a random observation between studies.

AEROBIC FITNESS AND SUSCEPTIBILITY

Whinnery and Parnell [134] reported that 2% of endurance trained subjects complained of motion sickness with 38% progressing to vomiting, while 23% of untrained subjects experienced motion sickness with 7% progressing to vomiting. A longitudinal study by Cheung, et al. [14] concluded that tolerance to vestibular stimuli decreased as the subject's aerobic capacity improved. Aerobic capacity has been reported to be specifically linked to signs and symptoms of motion sickness of vasomotor origin including stomach discomfort, nausea and vomiting [110]. Vasomotor symptoms (epigastric discomfort, nausea and vomiting) are significantly increased in aerobically fit individuals.

MOTION SICKNESS AND CORE TEMPERATURE CHANGES

Among the other signs of motion sickness, body temperature has received a renewed attention in recent investigations leading to the hypothesis that motion sickness can facilitate the development of hypothermia. The report on this phenomenon dates back to 1874 when Hess described that the body temperatures of seasick patients were found to be at least half a degree (Fahrenheit) lower than those of the same persons under normal conditions. In addition, early observation of seasickness also indicated that victims of seasickness showed marked pallor, cold and clammy extremities and a slightly subnormal temperature [9, 12, 73]. Other temperature signs reported include coldness of the extremities [9, 90] and lower skin and oral temperature [62, 63].

A case report by Golden [46] described that two occupants who had been shivering violently for 3 or 4 hours before capsizing, were both seasick and ultimately lost their lives. Investigation into the Alexander L. Kielland (an oilrig accommodation platform at sea) disaster (27 March, 1980) revealed that most survivors rescued from

the TEMPSC (totally-enclosed motor-propelled survival craft, i.e. lifeboat) suffered from hypothermia and seasickness at the time of rescue [105]. Recent studies by Mekjavic, et al. [92], Sweeney, et al. [123], and Nobel, et al. [103], all suggested that subjects exhibit potentiation of core cooling although some of these observations did not reach statistical significance. None of these studies consider the susceptibility of the individuals and the severity of the stimuli. The idea that all seasick survivors in a cool environment are particularly susceptible to hypothermia may be overly stated. A recent laboratory study suggested that the decrease in body temperature could be related to the level of susceptibility. The decrease in core temperature did not reach statistical significance once the subjects (of all level of susceptibility level) habituated to vestibular Coriolis cross coupling induced stimulus in 4 consecutive days [26, 28].

PREVENTION AND COUNTERMEASURES TO SEASICKNESS

Prevention of seasickness can take several forms, for example, elimination or reduction of the cause (the motion environment) which is not practical unless the affected individual withdraws from that type of occupation. The second possibility is the isolation of the body from the cause. For example, it has been suggested that in situations where the dominant stimulus is a changing linear acceleration in a defined axis, as in a heaving ship, the sickness is less severe when the stimulus axis is in the longitudinal (Z) axis of the head than in the antero-posterior (X) axis [130]. Finally, one can minimize the effects of the cause by pharmacological treatment and/or desensitization training.

It has been demonstrated in the laboratory and in the field that certain drugs can reduce the incidence and severity of motion sickness. Unfortunately, none can completely prevent motion sickness in the population at risk under all conditions of provocative stimulation. In addition, none of the drugs of proven efficacy in the treatment of motion sickness are entirely specific and all have side effects [138], which severely limit their utility in the working environment. There have been some attempts to study the impact of anti-motion sickness drugs on psychomotor performance. Paul, et al. [108] concluded that, among 25 mg promethazine, 50 mg meclizine, 50 mg dimenhydrinate, 25 mg promehazine plus 60 mg pseudoephedrine and 25 mg and 10 mg of d-amphetamine, only the last combination was free from having an effect on psychomotor performance and did not increase sleepiness. Unfortunately, the study was conducted without subjecting the participants to provoking motion. It is unknown which medication was in fact, effective in ameliorating motion sickness in those individuals and their correlation with psychomotor performance. In addition, it is well known that the pharmacokinetics (rate of absorption and metabolism) of medication varies under different stressful environments [88].

Those given drugs must be warned that the drugs may impair their ability to drive or operate machinery and that they should refrain from the consumption of alcohol as it will increase the sedating effect. The mechanisms of action of commonly used agents are poorly understood. It has been postulated that these agents suppress integration of sensory stimuli in the vestibular nuclei or the electrical activities in the vestibular nuclei. Some of these drugs (e.g. dimenhydrinate) in fact, were shown to suppress nystagmus during rotation.

There are four major classes of pharmaceuticals that have been used:

1) Antimuscarinics (Scopolamine/Hyosine);
2) Antihistamines (Cyclizine, Meclizine/Bonamine);
3) Anticholinergics – with antimuscarinic and antihistamines properties (Promethazines “Phenergan”, Diphenhydramine “Benadryl”, Dimenhydrinate “Dramamine”); and
4) Sympathomimetics (Amphetamine, Ephedrine).

The three relatively effective and commonly used drugs (promethazine, dimenhydrinate, and scopolamine) are central depressants that can affect brain activities and cause drowsiness or sleepiness and dizziness. They should not be taken by those in whom an impairment of skilled performance could jeopardize safety. There is a place, however, for the administration of anti-motion sickness drugs to crew members during the early stages of training. The possible performance decrement due to sickness must be weighed against side effects that may be produced by the drugs.

The commonly used dosage and duration of action are listed below:

Antimuscarinics

1) Scopolamine

- 0.3 – 0.6 mg Scopolamine HBr (+ 5 mg of Dexedrine).
- Oral dose, acts within 0.5 – 1.0 hour, lasts for 3 – 4 hours.
- Transdermal therapeutics system (TTS) patch applied 18 hr before lasts for 48 – 72 hours; it delivers a loading dose of 200 mg and a controlled release at 10 mg/hr.
- It exhibits high variability between subjects in both effectiveness and incidence of side effects.
- It is non-selective for 5 types of muscarinic receptors found in vivo.

Side effects of Scopolamine:

- Autonomic nervous system: Reduced salivation, bradycardia, blurred vision (reduced accommodation).
- Central nervous system: reduced short term memory, impaired attention, and lowered feelings of alertness.

2) Zamifenacin

It is an M3 and M5 muscarinic antagonist which was as effective as scopolamine in human subjects when tested using the rotating chair.

Antihistamines H_1 Receptor Antagonists

These are less efficacious than antimuscarinics and are more commonly used due to high safety and longer duration.

1) 50 mg Dimenhydrinate (Dramamine, 8-chlorotheophylline salt).
2) 50 mg Cyclizine HCl (Marzine) has been displaced in the US by the longer acting meclizine, oral dose, acts within 1 – 2 hours, lasts about 6 hours.
3) 50 mg Meclizine (Bonamine, Ancolan, Postafene), 1 – 24 hours before exposure to motion.
4) Cinnarizine (and its derivative, Flunarizine 10 or 30 mg single dose) with low incidence of sedation, but unavailable in the US [118]. Common side effects include: sedation, spatial disorientation, reduced hand-eye coordination, reaction time, psychomotor performance tasks such as digit substitution, critical flicker fusion threshold, etc. Those side effects may last 8 – 12 hours after ingestion.

Antimuscarinics/Antihistamines

These drugs are frequently described as antihistamines that also have anticholinergic effects. At therapeutic doses, they are highly anticholinergic. They exert antagonistic action on the parasympathetic system and relax voluntary muscle and they are short acting.

1) 25 mg Promethazine HCl (Phenergan) + 25 mg pseudo-Ephedrine, Promethazine is more anticholinergic than diphenhydramine (Benadryl).
2) Oral dose, acts within 1 – 2 hours, lasts for 8 – 12 hours.
3) Side effects include drowsiness and sleepiness.
4) Diphenhydramine is effective in humans during turbulent flights and in the laboratory.

Sympathomimetics

Amphetamine is effective in rotating chair [17, 78] and commercial transatlantic cruises [136, 137]. However, studies using vomiting as end points reported no benefits in swing sickness, acrobatic flight transatlantic troop-ships or small craft in heavy seas [119, 128]. The differences in findings could be a result of the different duration of testing, severity of stimuli, different end-points and different motivational factors.

SECOND GENERATION NON-DROWSY ANTIHISTAMINES

Due to the unwanted side effects of drowsiness in antihistamines, there is merit to the experimental testing of "second generation" antihistamines as they do not cross the blood brain barrier. They bind selectively to peripheral H_1 receptors and are less likely to cause drowsiness and have a relatively longer duration of action. For example, a single large dose (300 mg) of terfenadine (Seldane®) was shown to have a statistically significant therapeutic effect as an anti-motion sickness drug [79]. However, over the past few years there has been increasing evidence of cardiotoxicity with terfenadine and astemizole not related to their antihistaminic potency, but due to the blockade of the delayed rectifier potassium current leading to prolongation of QTc (QT interval corrected for heart rate) and with the possibility of ventricular arrhythmias [37]. Cheung, et al. [24] reported that cetirizine (Reactine®), at dosage of 10 mg/d and fexofenadine (Allegra®) at dosage 60 mg/d did not significantly influence the amount of vestibular cross-coupling stress that subjects could tolerate before reaching the symptom of definite nausea. Furthermore, no significant difference were noted in the total number and severity of symptoms displayed. It appears that the selective peripheral actions of cetirizine and fexofenadine are of no benefit in the prevention or treatment of laboratory induced motion sickness. These research findings raise additional questions regarding the relationship between the sedative action and the anti-emetic effectiveness of the H_1 receptor antagonist.

FIELD EVALUATION OF ANTI-MOTION SICKNESS DRUGS

During a voyage traversing the Drake Passage between Argentina and the Antarctic Peninsula that took 2 – 3 days at 11 knots with gale force winds and sea swells up to 9 m [43] conducted a sea trial evaluation of anti-motion sickness drugs. Ninety-eight percent (260/265) of the passengers participated in the study, with ages ranging from 15 – 87 years (115 males and 145 females). The number of individuals and the type of anti-motion sickness drugs that were used are listed as follows:

191 subjects used medications in the following proportion and types:

- 67 (35%) used meclizine
- 60 (31%) used transdermal scopolamine
- 28 (15%) used dimenhydrinate
- 16 (8%) used cinnarizine
- 20 (10%) used acupuncture wrist bands

Their finding in decreasing order of efficacy is: Scopolamine > Meclizine > Dimenhydrinate > Cinnarizine > Acupressure.

OTHER UNCONVENTIONAL TREATMENTS

Acupuncture has been used to treat gastrointestinal symptoms in China. A commonly used acupuncture point is P6, the Neiguan point on the pericardial meridian, located about 3 cm from the distant palmar crease, between the palmaris longus and flexor carpri radialis tendons. Various forms of acupuncture therapy are available as alternative treatment of motion sickness. There are a number of studies which suggest that acupressure is effectiveness in treating visually induced sickness [66, 67] and seasickness [10]. However, commercial devices such as wristbands, sea bands and other forms of acupressure therapy have been investigated under controlled scientific studies [13, 131] and found to be ineffective in reducing nausea and vomiting as induced by motion in humans. Furthermore, Miller and Muth [96] suggested that acupressure is ineffective in treating cross coupled induced sickness.

There are many other unproven treatments offered for ameliorating motion sickness. A variety of herbal (ginger-root), homeopathic (Cocculus, Nux Vomica, Petroleum, Tabacum, Kreosotum, Borax and Rhus Tox) remedies have been proposed. In particular, some studies on the effectiveness of ginger roots have been performed. Ginger root was reported by Mowry and Clayson [100] to have prophylactic effects. In a controlled sea trial of ginger root, fewer symptoms of nausea were reported after ginger root ingestion; however, the difference was not statistically significant [57]. However, Stott, et al. [121], Stewart, et al. [120] failed to substantiate the effectiveness of ginger roots. A controlled double-blind study revealed that powered ginger root (Zingiber officinale) had no influence on experimentally induced nystagmus [65]. Any reduction of motion sickness symptoms derived from ginger roots may be acting at the gastric system level. In general, most of these herbal remedies have not been found consistently effective and the various purported evidence is confusing at best. It is possible for the alternative remedies to appear beneficial by a combination of the placebo effect and habituation to the environment. It is, however, prudent to avoid any purported effective commercial devices until scientific validation is available.

BIOFEEDBACK AND RELAXATION THERAPY

Biofeedback uses operant conditioning to control autonomic responses. Physiological measures such as heart rate, blood pressure, body temperature, and galvanic skin resistance are commonly chosen as control parameters in “biofeedback” training. However, it is not clear whether a consistent and reliable relationship exists between motion sickness and these measures [35, 50, 52, 54, 55, 61, 97]. Moreover, individuals vary greatly in the extent to which they can benefit from biofeedback training. Training conducted in the laboratory situation may not transfer to operationally relevant situations, which involves active integration with other tasks [8, 93, 94, 95, 115]. It appears that biofeedback and other behavioural techniques can modify the physiological responses of some individuals and ameliorate the anxiety that accompanies certain noxious situations, but it remains to be

seen whether these responses bear a direct relationship to the symptoms of seasickness and other forms of motion sickness.

DESENSITISATION

The most suitable non-pharmacological intervention at least for airsickness in the military environment appears to be habituation to the nauseogenic stimuli. Various desensitisation schemes have been shown to be helpful for those who do not develop sufficient protective habituation during the course of normal flight training [4, 6, 20, 38, 40, 44, 53, 70, 86]. As mentioned earlier, motion stimuli tend to provoke sickness when the motion elicits patterns of sensory stimulation that do not conform to those expected on the basis of past experience. Therefore, exposure to the nauseogenic manoeuvre is essential. This exposure also provides the individual affected by motion sickness with the opportunity to improve their ability to predict the spatial sensory patterns that are generated by the spatial consequence of their actions. This ability is crucial to resolve the sensory conflicts or neural mismatch in an altered gravitoinertial environment and thus the stimulus is less able to provoke motion sickness.

Earlier studies suggested that habituation to slow (yaw) rotation to the right have been shown to result in suppression of responses to both right and left yaw rotation [50]. Rapid adapters to sudden stop visual-vestibular interaction also showed rapid adaptation to parabolic flight and rapid to average adaptation to cross-coupled stimulation in the slow rotation room [53]. Tolerance acquired using real motion (rotating chair that also tilted ± 40° in the roll and pitch planes) could transfer to circular- and linear-vection [39]. On the other hand, it was shown that habituation to vertical linear acceleration did not increase tolerance to Coriolis acceleration [109]. Similarly, tolerance acquired to the cross-coupled angular motion did not result in an increase in tolerance to vertical oscillation [4]. Although there has been no sea trial to determine the degree of improvement in tolerance to ship motion that results from tolerance gained from ground-based training, Cheung and Hofer [26] suggested that that desensitization to one provocative motion could be transferred to a less provocative motion stimulus.

SOME PRACTICAL RECOMMENDATIONS TO MINIMIZE THE OCCURRENCE AND/OR DELAYING THE ONSET OF SEASICKNESS

Behavioural Measures

1) Be well informed about the causes of seasickness; have a thorough understanding of what sensory systems are involved and be familiar with the signs and symptoms of motion sickness.

2) It is useful to be familiar with one's symptom development of motion sickness on the ground, beginning with mild Coriolis stimulation, and progressively moving to more provocative and specific stimuli.

3) Do not dwell on past experience of seasickness (motion sickness) or worry about the occurrence of seasickness because anxiety will only inhibit habituation to the provocative environment.

4) For those who have a choice, but this is often not the case. An individual should not sail unless he feels fit and well. Do not go out to sea when you are hung over or have an upset stomach. Recent illness and fatigue all cause debility and adversely affect an individual's general ability in the air. They also make one prone to seasickness especially during severe sea state.

5) Affected individuals should discuss their symptoms of seasickness, as early as possible, fully and frankly with the on-board medical staff. It will facilitate recovery and prevent misunderstanding when the effects of seasickness decrease an individual's performance. The ship captain or whoever is in charge of the vessel or helicopter, should be aware of the cause and symptoms of seasickness could make minor adjustments, when possible, to assist the individual to habituate quickly. The affected individual should be given every opportunity to be in at sea whenever there is an opportunity for habituation/desensitisation.

6) Affected individuals are likely to develop some degree of anxiety about their sickness problem. Minimizing the anxiety by introducing the personnel (such as pilots/aircrew) gradually to the type of motion that might be experienced using ground-based devices (e.g. six degrees-of-freedom motion platforms) might be useful. However, it is understood that mariners may not have such privilege. In those cases, a self-desensitisation procedure such the torso-rotation technique [26] may be introduced under the supervision of a physician or medical staff, who is familiar or have been taught about the procedure.

7) It is useful to be involved and to concentrate on the task at hand which can minimize the introspection and attention to bodily function. This is evident by the fact that the person least likely to be motion sick is the pilot of the aircraft. or driver of the car. However, when it is not possible to be in control of the vessel, involvement in some absorbing task is better than being preoccupied with the state of one's stomach.

8) Do not self-medicate with over-the-counter anti-motion sickness drugs. In certain operational personnel, the attending physician may decide to prescribe some form of medication. Be aware that all the current effective anti-motion sickness drugs have side effects. These side effects include dryness of the mouth, sleepiness, and dizziness. In some cases serious visual disturbance includes double vision.

9) Food Consumption: The fear of seasickness sometimes results in avoidance of food intake, but experimental studies have found no evidence that the time of day of motion exposure or its relation to meal times has any effect on the incidence of motion sickness. Excessive consumption of food may be best avoided since it may increase the volume of vomitus, and therefore, both the fear of sickness and the extent of any subsequent inconvenience. It is generally agreed that vomiting is less unpleasant when the stomach contains something to vomit than an empty stomach. It is recommended that personnel should maintain a normal light consumption of food and drink.

10) Avoidance of Alcohol: It is well known that the after-effects of alcohol (hangover) adversely affect an individual's general ability. Alcohol can cause semicircular canal conflict by developing a density gradient with the membranous canal. It has also been shown that alcohol affects the visual feedback of target position during voluntary and involuntary head movement [7]. After the blood alcohol concentration has been raised high enough[1] and the alcohol has subsequently disappeared from the blood, it continues to have measurable effects [139] on the brain, on the vestibular system, and in some cases on blood sugar. Motion sick individuals have observed that their susceptibility to for example, air sickness, is increased by even moderate amounts of alcohol in the previous 24 hours [122], and this effect of alcohol has also been observed on tolerance to cross-coupled stimulation during desensitization treatment [4]. Of course, excessive consumption of alcohol can result in vomiting without provocative motion.

The eight-hour rule is quite inadequate for heavy drinking[2], since there would still be significant concentrations of alcohol in the blood eight hours after a peak blood alcohol concentration of 150 mg%.

[1] Depending on the individual and the speed of ingestion, in most cases four to six drinks, would be sufficient to raise the blood alcohol concentration over 100 mg% which is certainly high enough.

[2] Heavy drinking is defined as having a blood alcohol concentration of 150 mg% among occasional drinkers in a social setting.

Under some circumstances, there are measurable decrements of pilot performance even at 14 hours after a peak blood alcohol concentration of only 100 mg%. The current recommendation for commercial airline pilots is 24 hours after a blood alcohol concentration of 150 mg% before flying. For those taking anti-motion sickness medications, it is important to note the increased sedation from alcohol.

Environmental Considerations

1) When possible, locate critical stations or passengers seating for those who are susceptible near the ship's effective centre of rotation and align the affected individual with a principal axis of the ship's hull.

2) Provide optimal environmental conditions, suitable temperature and ventilation when possible.

3) Provide an external visual frame of reference when possible.

4) As far as motion sickness treatment medications, etc., there is no "magic bullet". The affected individual should consult a physician to experiment with a number of standard anti-motion sickness drugs under supervision.

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