

Motion Sickness  
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**Introduction:**

Motion sickness is a response to unusual perception of motion, whether real or apparent.<sup>1</sup> It is a syndrome that can include gastrointestinal, central nervous system, and autonomic symptoms. It is known as a “physiologic” form of dizziness because there is no evidence of a pathological disease process and the syndrome of motion sickness can be induced in almost all normal human subjects with adequate provocation.<sup>1</sup> There is a large amount of variability in the susceptibility of motion sickness, as it can be produced with minimal irritation in some but be difficult to elicit in others. Motion sickness can be a nuisance for the avid traveler who enjoys jet setting or sailing the seas. This paper will review the pathogenesis, susceptibility factors, symptoms and treatment of motion sickness. It will also review the syndrome of mal de débarquement, which occurs on land after exposure to a motion stimulus.

**Pathogenesis:**

Motion sickness symptoms were first described by Hippocrates over two thousand years ago. Hippocrates observed that, “sailing on the sea proves that motion disorders the body” (a that time symptoms of motion sickness were most frequently seen with boat travel).<sup>2</sup> In fact, the principal symptom of motion sickness (nausea) is derived from the Greek word for ship (*naus*).<sup>2</sup> Initially, it was thought that symptoms of motion sickness were caused by the damp sea air, however, in the nineteenth century several theories were constructed inciting motion as the culprit.<sup>3</sup>

One theory was the “guts shift” theory. There are many variations of this theory, but all proposed mechanisms thought to cause the symptom of vomiting. The simplest theory proposed that vomiting was a reflex response to irritation of the gastric mucosa associated with motion. Other theories proposed that movements of the viscera irritated the liver, which as a result, released too much bile into the small intestine and eventually caused emesis.<sup>3</sup>

Other historical theories include manifestations from different vascular phenomenon. One theory suggested that there was a lack of blood flow to the brain because irritation of the eyes secondary to the perceived motion reflexively produced spasm of cerebral capillaries causing giddiness and vomiting.<sup>3</sup> Another theory proposed that motion caused “cerebral hyperemia,” meaning too much blood was going to the brain which in turn destabilized brain cells in the vomiting center of the medulla oblongata.<sup>3</sup>

Both the “blood and guts” theories of motion sickness were eventually discounted because it was noted that individuals who lack inner ear vestibular function were immune to motion sickness.<sup>4,5</sup> The vestibular system consists of the semicircular canals, which sense angular motion, and the otolith organs (sacculle and utricle), which sense linear acceleration and gravitational force. Most current theories propose motion sickness as a manifestation of some aspect of the vestibular system.

Currently, the most widely accepted theory for the cause of motion sickness is variations of the “sensory-conflict theory.” During self-generated movements, the motor command and sensory feedback are congruent. The brain receives sensory signals and vestibular cues from the labyrinth, visual information, and somatosensory signals, which in turn allows the brain to estimate motion and the spatial orientation of the head and body. During active movements, like

walking, the motor command of the brain can also estimate the motion of the head and body. So, in self-generated movements, the vestibular cues and motor command cues are congruent.

Motion sickness is thought to be a manifestation of the incongruence of the sensory feedback and motor commands, thus a “sensory conflict” occurs. In the absence of active movements, the brain can only rely on the vestibular, visual and somatosensory cues it obtains to make a determination of orientation in space. For example, motion sickness often occurs while on a boat. The visual system indicates the person is stationary (seeing the interior of the cabin), but the vestibular system senses ongoing head movements (motion of the ship crashing against waves). Therefore, the visual system and vestibular systems are in conflict, thus producing the symptoms of motion sickness.

One may ask, what is the evolutionary advantage of motion sickness? This can be explained by the “toxin detector” theory. It is known that the primary function of the vestibular system is spatial orientation, balance, and stabilizing vision through vestibular-ocular reflexes (VOR), however, it has been proposed that an additional vestibular function is to act as a toxin detector.<sup>2</sup> The toxin detector hypothesis proposes that the brain has evolved to recognize any changes in expected patterns of vestibular, visual, and kinesthetic information as evidence of central nervous malfunction and to initiate vomiting as a defense against a possible ingested neurotoxin.<sup>2</sup> This evolutionary based theory feels that the vestibular system provides a “back-up” to the main toxic detector system of chemoreceptors of the afferent vagal nerves and the chemoreceptor trigger zone of the brainstem.<sup>2</sup> This theory also supports the notion that people who are more susceptible to motion sickness are also more susceptible to toxins such as chemotherapy and postoperative nausea and vomiting.<sup>2</sup>

Although there are several hypotheses for the etiology of motion sickness, an important aspect of motion sickness is adaptation. Adaptation implies that motion sickness symptoms often resolve with continued exposure to stimuli.<sup>6,7</sup> This is evidence that the CNS gradually recognizes that conflicting sensory inputs are being received and adapts to this situation leading to a reduction in symptoms.<sup>7</sup> The process of adaptation occurs by two-three days of continuous stimulation.<sup>1</sup>

**Motion Sickness Susceptibility:**

Motion sickness can be induced in almost all normal human subjects with the correct amount of provocation. The variety of stimuli that can provoke motion sickness is extensive as seen in Table 1.<sup>2</sup>

**Provocative Stimuli**

Context	Examples of Provocative Stimuli
Land	Cars, coaches, tilting trains, ski, camels, elephants, funfair rides
Sea	Boats, ferries, survival rafts
Air	Transport planes, small aircraft, hovercraft, helicopters
Space	Shuttle, Spacelab
Optokinetic	Wide-screen cinemas, microfiche-readers, simulators, virtual reality
Correlated stimuli	Emetic toxins, chemotherapy, post operative nausea and vomiting (PONV)

Table 1: Adapted from Golding J. Motion sickness susceptibility. *Autonomic Neuroscience*. Oct 2006; 129(1-2).

Although, the list of stimuli that can provoke motion sickness is great, it is difficult to predict the severity of symptoms one will experience when exposed to these stimuli. It has been found in controlled laboratory and ship motion surveys that lower frequencies (< 1 HZ) of motion induce more nausea than higher frequencies, with a peak nausea reached at 0.2 HZ.<sup>1,2,8</sup> These low frequency motions are seen with ships, cars and planes. However, walking, running, and biking have higher frequencies and are thus felt to be less nauseogenic.<sup>2</sup> Also, the type of motion can induce motion sickness more than other types. The pattern of motion that most likely induces motion sickness is a repetitive linear or angular acceleration of the head.<sup>1</sup>

It is difficult to ascertain susceptibility to motion sickness, but it is believed that three processes are at work: “an initial sensitivity to motion, the rate of natural adaptation, and the ability to retain protective adaptation in the longer term.”<sup>2</sup> Also, the concept of motion sickness susceptibility is believed to overlap with susceptibility to other non-motion emetic stimuli, such as migraines, chemotherapy, and post-op nausea and vomiting because it is believed that there is involvement of the vestibular system in non-motion emetogenic stimuli.<sup>2</sup> However, some risk factors have been found to cause some individuals to be more susceptible to motion sickness than others. It was found that women are more susceptible to motion sickness than men.<sup>9</sup> Also, children (peaks around age 11) are more susceptible to motion sickness and then it decreases in adulthood.<sup>10</sup>

J. E. Bos et al did a study in which 2840 passengers aboard four ships (two cruise line ships and two smaller catamarans) completed questionnaires regarding seasickness. The questionnaire employed an illness rating (IR) from 0-3 (0= I felt alright; 1= I felt slightly unwell; 2= I felt quite ill; 3= I felt absolutely dreadful), which passengers felt during their voyage as related to their motion sickness. Table 2 shows the gender, age and sickness history distributions of these passengers.

Gender	Age	Sickness History
Female 1433 (50.5)	4-12 133 (4.7)	Not Sick Before 1784 (62.8)
Male 1407 (49.5)	13-24 668 (23.5)	Sick Before 1056 (37.2)
	25-42 911 (32.1)	
	43-60 824 (29)	
	61-88 304 (10.7)	

Table 2. Gender, age and sickness history distributions (percentages are given in parentheses relative to the 2840 complete sets of questionnaires).

Bos JE, Damala D, Lewis C, Ganguly A, Turan O. Susceptibility to seasickness. *Ergonomics* Jun 2007; 50(6): 890-901.

Using the IR as the dependent variable and the given categorization for age as the independent, an ANOVA revealed highly significant main effects for gender ( $p < 0.001$ ), age ( $p < 0.001$ ) and history ( $p < 0.001$ ). All two-way interactions were significant as well (gender\*age:  $p < 0.02$ ; gender\*history:  $p < 0.02$ ; age\*history:  $p < 0.05$ ).<sup>10</sup> As Figure 1 demonstrates, female illness ratings peaked at an age of 11 years, 1.5 times as high as male ratings, which peaked at an age of 21 years. At higher ages, illness ratings decrease to only 20% of their maximum, reducing gender differences to zero. Passengers with a previous history of seasickness rated their illness about two times higher than those who had not felt sick before.<sup>10</sup>

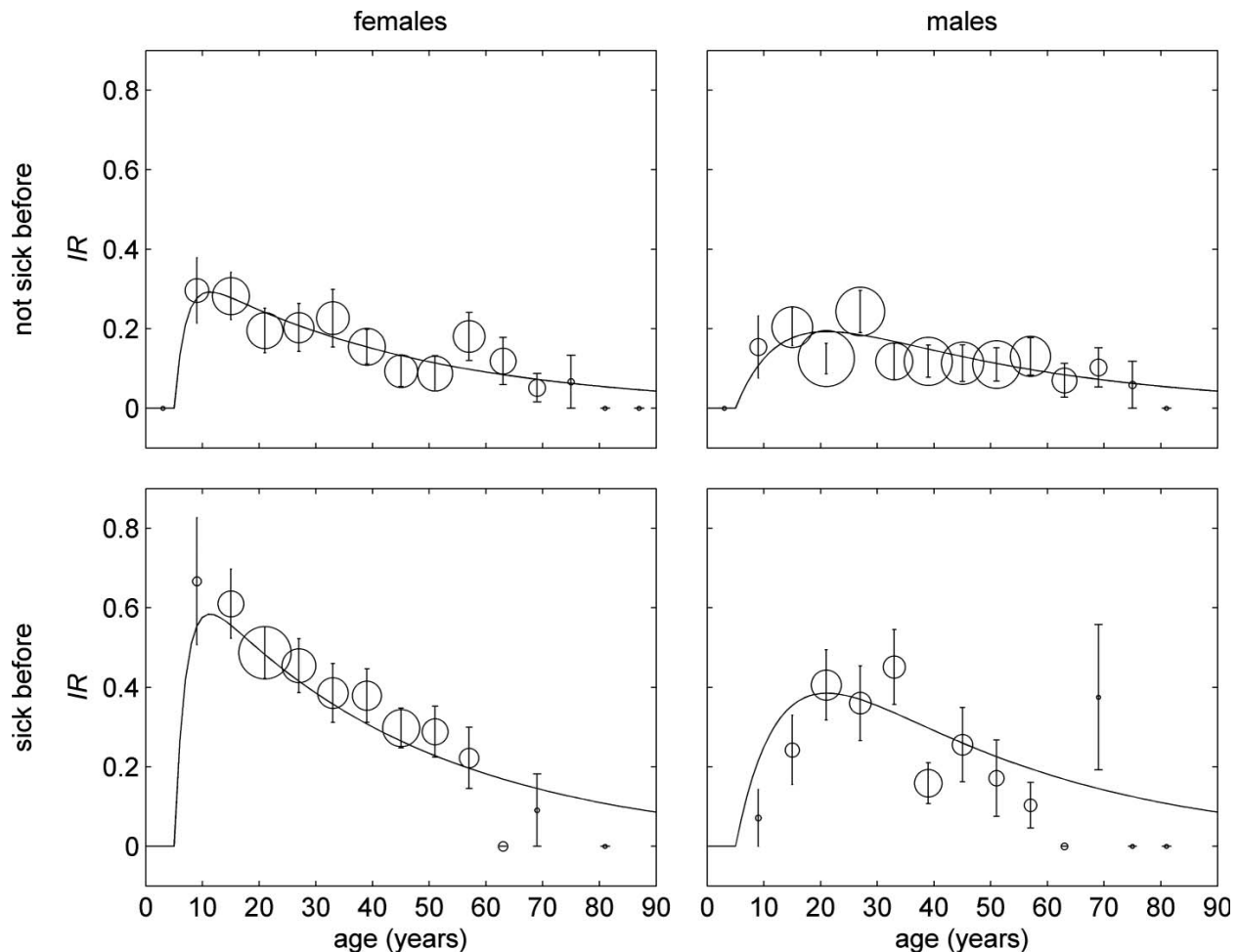


Figure 1. Mean illness ratings (IR) differentiated for gender, age and sickness history. Error bars indicate the SEM. The radius of each circle is proportional to the number of passengers included in each mean.  
 Bos JE, Damala D, Lewis C, Ganguly A, Turan O. Susceptibility to seasickness. *Ergonomics* Jun 2007; 50(6): 890-901.

It is believed that infants and very young children do not get motion sick because the perceptuo-motor map of the brain is still highly plastic and not fully formed until later in childhood.<sup>2</sup> The decline in motion sickness after the age of 20-30 is thought to reflect habituation.<sup>2</sup>

Other predictors in the susceptibility to motion sickness include the function of the vestibular function. Almost, the only individuals who are immune to motion sickness are those who have complete bilateral loss of labyrinthine function.<sup>1,2</sup> Genetics to motion sickness susceptibility has been thought to play a role; however, the evidence is limited. There has been an observation that a single nucleotide polymorphism of the alpha 2 adrenergic receptor increases autonomic responsiveness to provocative motion, but it is unclear if this is a marker for motion sickness susceptibility or a general marker for autonomic sensitivity.<sup>2,8</sup>

### Clinical Presentation

The development of motion sickness follows an orderly sequence that varies with the intensity of the motion stimulus and the susceptibility of the individual.<sup>1</sup> The initial symptom is usually discomfort around the upper abdomen (stomach awareness), which is followed by nausea,

increasing malaise, and peri-oral and facial pallor.<sup>1</sup> As symptoms worsen, there can be salivation changes, dizziness, diaphoresis, and recurrent vomiting.<sup>1,6</sup>

### **Treatment:**

Several interventions have been studied to prevent or alleviate the symptoms of motion sickness. These interventions include pharmacologic as well as behavioral countermeasures.

#### *Behavioral Methods:*

Habituation provides the surest counter measure to motion sickness. Habituation is a learned central process in which the decreased susceptibility to motion sickness is maintained even with significant intervals up to several weeks.<sup>1</sup> Habituation is taught via “motion sickness desensitization” programs.<sup>2</sup> Critical features of desensitization programs include the massing of stimuli (exposures to provocative motion stimuli must not be more than one week apart), use of graded stimuli to enable faster recoveries so more sessions can be scheduled, and a positive, psychological attitude to therapy.<sup>2</sup> It is also very important to use a variety of provocative stimuli as habituation itself is often stimulus specific.<sup>2</sup>

More immediate and short-term behavioral methods include reducing head movements by laying supine or obtaining a stable, fixed external horizon reference.<sup>1,2</sup> Other strategies include avoiding reading or engaging tasks because they require continual changes in the visual fixation and unnecessary head movements.<sup>1</sup>

Additional behavioral methods include controlled regular breathing or listening to music audiotapes. Yen Pik Sang et al conducted a study on twenty-four healthy subjects who were exposed to a rotating turntable. They were exposed to this stimulus under three conditions: 1) while focusing on controlling breathing; 2) listening to a music audiotape; 3) or with no intervention (control).<sup>11</sup> [The use of the breathing technique was used by these authors because of anecdotal reports of naval aviators saying controlled breathing suppressed motion sickness, an observation in accordance with the existence of an inhibitory reflex between respiration and vomiting].<sup>11</sup> Each subject participated in each condition at one week intervals. During each condition, subjects were rotated on a turntable and had to rate their feelings of sickness every 30 seconds as: 1= no symptoms; 2=initial symptoms; 3= mild nausea; 4=moderate nausea. Once the subject had reached a rating of 3 (mild nausea), the intervention of slow deep breathing or music audiotape was introduced. The motion was stopped once a subject reached a rating of 4 (moderate nausea). Tolerance to the motion challenge was assessed via two factors: first, the time from the start of the rotating turntable needed to reach moderate nausea and second, the time from mild nausea to progress to moderate nausea. The results of the study showed that the time for which motion could be tolerated from a rating of 3 (mild nausea) before the onset of a rating of 4 (moderate nausea) was longer for breathing with a mean of 10.7 minutes ( $p < 0.01$ ) and for music with a mean of 10.4 minutes ( $p < 0.01$ ) when compared with control at 9.2 minutes. However, there was no significant difference between breathing and music in prolonging tolerance to motion exposure. This study concluded that breathing and the music audiotape provided significant protection against motion sickness thus making these techniques an effective nonpharmacologic measure that is easy to use and free of side effects. Also, because the interventions were started when the subjects already had an element of mild nausea, these techniques can be used not only for prevention of motion sickness but also for treatment. It can be argued that these methods serve as a distraction. However, exploratory tests with other techniques (e.g., mental arithmetic task, subtraction of serial sevens) did not appear to be as

effective as controlling breathing or the music audiotape, indicating that mental distraction cannot be the complete explanation for the effectiveness of these methods.<sup>11</sup>

#### *Acupressure:*

Acupressure is another alternative method that has been studied to prevent motion sickness. Acupuncture is an ancient Chinese practice that stimulates points on various meridians in the body with needles. It is believed that nausea and vomiting are controlled by the Neiguan/P6 point on the pericardial meridian.<sup>12,13</sup> The P6 point is located between the flexor carpi radialis and the Palmaris longus tendon, one sixth the distance between the distal palm and antecubital fossa.<sup>13</sup> Acupuncture is thought to work because it is a method of balancing positive and negative energies via invisible channels (meridians) in the body.<sup>13</sup> For nausea, the right P6 point redirects negative energy away from the heart and positive energy finds its way to the left P6 point, and this balance allows for nausea to be controlled.<sup>13</sup> Obviously, at times when motion sickness presents itself, it is difficult to conduct an acupuncture session; therefore, noninvasive methods of stimulating P6 have been developed including acupressure and acustimulation. Several studies have been conducted regarding the efficacy of acupressure relieving the symptoms of motion sickness with varied results. Stern et al conducted a study to determine whether Acuband (an acupressure device with a ball that could be pushed at the P6 point), would relieve the symptoms of motion sickness. 25 subjects, who were otherwise healthy, were tested in an optokinetic drum wearing the band on: 1) wrist; 2) arm; and 3) no Acuband. The main outcome measures included subjective symptoms of motion sickness and measured gastric myoelectric activity or an electrogastrogram (EGG).<sup>12</sup> “EGG is a noninvasive measurement of stomach activity using surface electrodes positioned over the abdominal surface.”<sup>1</sup> Normal EGG activity is approximately 3 cpm (cycles per minute) and the presence of tachyarrhythmic activity (4.0-9.75 cpm) has been repeatedly associated with nausea.<sup>13</sup> The results of the study showed that the subjective symptoms of motion sickness were less for the group who wore the band on the wrist or arm versus wearing no band with a  $P > 0.01$  and that the percentage of tachyarrhythmias was less in the group who wore the band on the arm or wrist versus no band with a  $P < 0.05$ .<sup>12</sup> A subsequent study conducted by Miller et al looked at subjects using a ReliefBand (an acustimulation device with a maximum output of 0.35mA), an Acuband, and placebo (an Advance Healing Band-Aid placed on the dorsum of the subject’s hand). The acustimulation and acupressure groups were further divided into trained (subject was trained in using the product) versus untrained. The subjects were again exposed to an optokinetic drum rotation for a maximum of 20 minutes. The primary outcomes that were measured included a Motion Sickness Assessment Questionnaire (MSAQ), which asked questions regarding motion sickness symptoms of feeling queasy, faint, dizzy, etc. and also measuring EGG. The results of the study showed that in all conditions, motion sickness symptoms and gastric tachyarrhythmias increased. This study also demonstrated that despite being trained versus untrained in the use of the products, motion sickness symptoms still developed.<sup>12</sup>

The differences in the results of these studies may be secondary to the length of the exposure of the motion stimulus. In the first study, where Acuband seemed to have anti-motion sickness properties, the subjects were exposed to a maximum of 16 minutes of the motion stimuli versus 20 minutes in the Miller et al study. Therefore, the length of exposure to provocative motion stimuli may play a role in the utility of acupressure as a motion sickness countermeasure.

### *Pharmacologic Measures:*

The drugs used to target motion sickness can be divided into three main categories: antimuscarinics (e.g. scopolamine), H1 antihistamines (e.g. Dramamine), and sympathomimetics (e.g. amphetamine). These pharmacologic agents are thought to be effective because cholinergic and histaminergic fibers appear to be involved in the transmission of motion stimuli from the vestibular system to the vomiting centers, located in the medulla oblongata and other areas of the CNS.<sup>1</sup> Sympathomimetics are effective because this neuronal transmission is suppressed by adrenergic activation.<sup>1</sup> There are many modes of delivery for these medications, which is important because motion sickness induces gastric stasis thus preventing drug absorption.<sup>2</sup> Therefore, oral administration of a drug must anticipate motion, usually at least by thirty minutes. Transdermal routes offers advantages in that it provides protection for up to seventy-two hours with low constant concentrations in the bloodstream.<sup>2</sup> New formulations are being developed for medications including nasal sprays to allow for a faster route as well as chewing gum.

### Anticholinergics:

Scopolamine is one of the most commonly used medications for the treatment of motion sickness.<sup>7</sup> Its pharmacologic properties are thought to work by interfering with the transmission of vestibular input to the central nervous system, which in turn inhibits the vomiting impulse normally activated by motion sickness.<sup>7</sup> A Cochrane Review was conducted on Scopolamine in 2007 to assess the effectiveness of scopolamine versus no therapy, placebo, other drugs (anticholinergics, antihistamines, sympathomimetics, antiemetics, opioids), behavioral and complementary therapies or two or more of the above therapies in conjunction for motion sickness.<sup>7</sup> The criteria for considering studies for this Cochrane Review included all parallel-arm, randomized control trials focusing on scopolamine versus the above stated therapies, participants with motion sickness and no known vestibular, visual or central nervous pathology, and studies that used scopolamine as a single agent therapy or scopolamine in conjunction with another active agent, for treating and preventing motion sickness, regardless of route. The studies included must have also reported on the following outcome measures: primary outcome- prevention of onset and treatment of clinically defined motion sickness; and secondary outcomes- task ability and psychological tests, changes in physiologic parameters (heart rate, nystagmus, and EGG), and adverse effects (dry mouth, drowsiness, visual disturbances). Of the studies potentially relevant, 14 studies met the criteria for a total of 1025 subjects. In the studies, scopolamine could be administered in several ways, tablets, capsules, or oral solutions, but the most common was via a transdermal patch. It was also found that most studies recruited subjects with a history of motion sickness. The results of the Cochrane Review are broken into four sections based on the outcome measures:

### Primary Outcomes

1) Prevention of sickness symptoms (nausea): Five studies showed a superior effect of transdermal scopolamine over placebo for preventing sickness symptoms with a RR of 0.47 (95% CI 0.31-0.71). See Figure 2.

## GRAPHS AND OTHER TABLES

### Analysis 01.01. Comparison 01 Prevention of sickness symptoms (nausea), Outcome 01 Scopolamine vs placebo

Review: Scopolamine (hyoscine) for preventing and treating motion sickness

Comparison: 01 Prevention of sickness symptoms (nausea)

Outcome: 01 Scopolamine vs placebo

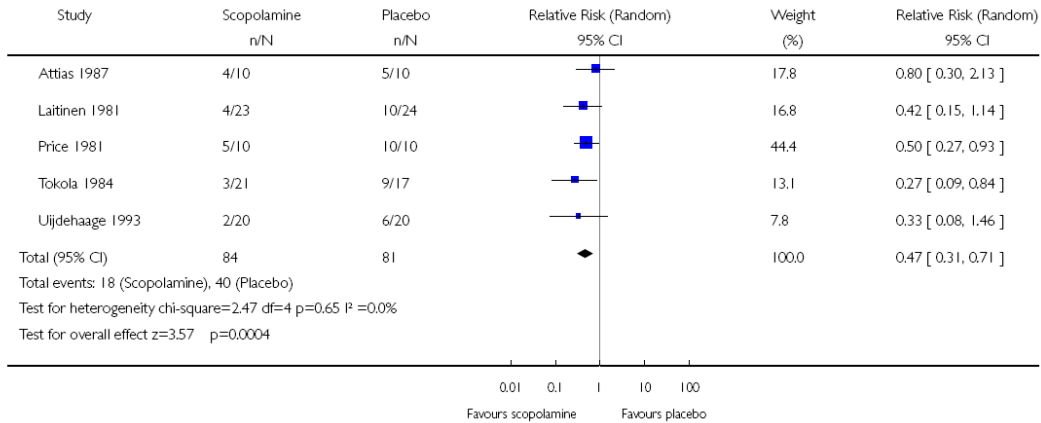


Figure 2

Spinks AB, Wasiak J, Villanueva EV, Bernath V. Scopolamine (hyoscine) for preventing and treating motion sickness. Cochrane Database of Systematic Reviews 2007, Issue 3.

2) Prevention of Vomiting: There was no significant difference in the prevention of vomiting between those taking scopolamine and placebo.<sup>7</sup>

#### Secondary Outcomes:

1) Task Ability and Psychological Tests: This was difficult to assess as the studies looking at these outcomes could not be pooled due to differences in outcomes.

2) Adverse Events: Two studies compared scopolamine with placebo for the side effect of drowsiness. There was no significant difference between the two treatments, but there was a trend toward greater drowsiness among participants using scopolamine with RR 1.42 (95% CI 0.79 to 2.56).<sup>7</sup> See Figure 3.

**Analysis 03.01. Comparison 03 Adverse event: drowsiness, Outcome 01 Scopolamine vs placebo**

Review: Scopolamine (hyoscine) for preventing and treating motion sickness  
 Comparison: 03 Adverse event: drowsiness  
 Outcome: 01 Scopolamine vs placebo

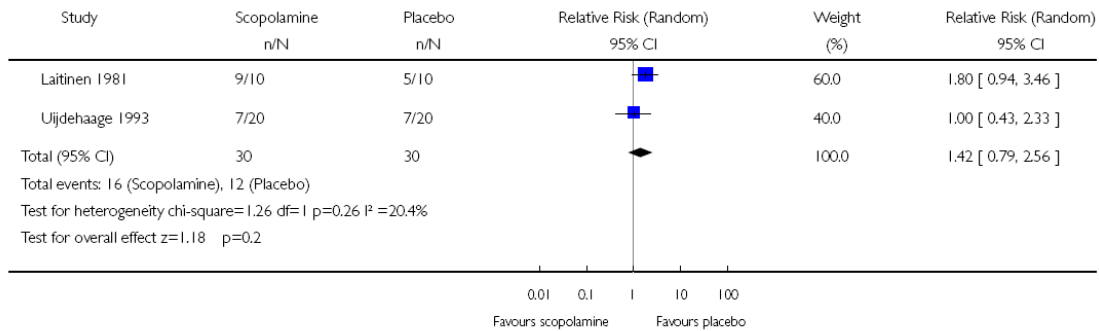


Figure 3

Spinks AB, Wasiak J, Villanueva EV, Bernath V. Scopolamine (hyoscine) for preventing and treating motion sickness. Cochrane Database of Systematic Reviews 2007, Issue 3.

Other adverse events including blurred vision, dry mouth, and dizziness had mixed results of significance when comparing scopolamine versus placebo.

**Histamine Blockers and Sympathomimetics:**

Histamine increases the firing rate in afferent nerves from the ampulla of the semicircular canals and thus H1 receptor antagonists are used as anti-motion sickness medications. Also, histamine H1 receptors are present in the vestibular nuclei at a high density thus allowing antihistaminergic drugs that cross the blood brain barrier effective against motion sickness.<sup>1</sup> Some examples of these medications include dimenhydrinate, meclizine, promethazine, and cinnarazine. Cinnarazine is very popular in Europe for the prevention of motion sickness with little side effects, but it is not available in the United States.<sup>1</sup>

It has been proposed that an increase in norepinephrine may counteract the increased activity of acetylcholine neurons stimulated by vestibular activation during motion sickness.<sup>1</sup> Amphetamine and ephedrine (alone and in combination) have been shown to have anti-motion sickness effects, but it is of no use for clinical practice given its high addiction potential.<sup>1</sup>

**Other Pharmacologic Therapies**

Ginger is one intervention that has been studied for the prevention of motion sickness because of its antiemetic properties. “The exact mechanism of action of ginger in relation to its antiemetic properties is unclear, although it appears to inhibit serotonin receptors and to exert antiemetic effects at the level of the gastrointestinal system and in the central nervous system.”<sup>14</sup> The literature on ginger (*Zingiber officinale*) has been varied. One study published in the Lancet evaluated ginger on the symptoms of motion sickness when compared to dimenhydrinate (Dramamine) and placebo. The study took 36 undergraduate students who reported a very high susceptibility to motion sickness. The subjects were blindfolded and placed in a rotating, titling chair to induce the symptoms of motion sickness. The subjects were asked to report their perceived gastrointestinal distress every fifteen seconds up to six minutes. It was found that the mean magnitude estimations of the subjects in their perceived GI distress increased most rapidly in the placebo group, then the Dramamine group, and then finally the ginger group. “A median

test showed that the mean estimations of the three groups were significant ( $p < 0.05$ ).” The study also looked at total time in the chair. Neither the placebo nor the Dramamine groups were able to stay in their chairs for the full 6 minutes. “Differences between the mean times in the chair varied significantly ( $p < 0.001$ )” with the placebo group lasting a mean of  $90 \pm 12$  sec, Dramamine  $216.2 \pm 10$  sec, and ginger  $335.8 \pm 8.2$  sec.”<sup>15</sup> However, subsequent studies have been conducted, which have shown no benefit of ginger for motion sickness.<sup>14</sup> The inconsistent results of ginger as an anti-motion agent is unclear, but is most likely secondary to the lack of randomized controlled trials evaluating ginger.

Benzodiazepines and barbiturates have been known to have anti-motion sickness actions, but their sedating side effects preclude routine use.<sup>2</sup> Opioids may also have a role. Although, opioids often elicit emesis, they have been shown in animals to have broad anti-emetic actions.<sup>2</sup> Loperamide, a mainly peripherally acting  $\mu$ -opiate receptor agonist, was reported to suppress ACTH release in human and vagal cholinergic mechanisms.<sup>16</sup> Otto et al conducted a study to evaluate the efficacy of loperamide as a medication to prevent motion sickness. Subjects were exposed to standardized rotation around a vertical axis combined with head movements to induce nausea three hours after receiving loperamide 16mg or placebo. The study evaluated subjects’ subjective ratings of the severity of their nausea as well as tested serum ACTH and ADH plasma levels (as it has been hypothesized that stress hormones may be involved in motion sickness). The results of the study showed that after subjects received loperamide when compared to placebo, they were found to have less nausea per rotation unit showing a reduction of kinetosis-induced nausea ( $p = 0.018$ ). The ACTH and ADH plasma levels of the subjects were found to increase less with loperamide versus placebo, but were not statistically significant. The limitation of this study was the small sample size ( $n = 7$ ), thus making it difficult to extrapolate the data to the average population. Also, the roles of ADH and ACTH in inducing symptoms of motion sickness need to be more clearly defined before loperamide can be considered a therapeutic agent against motion sickness.

### **Mal de Debarquement**

Mal de débarquement is the sensation of movement after the termination of motion.<sup>17</sup> It is usually experienced after a sea voyage, but can occur after car, train, or air travel. Mal de débarquement can be broken up into transient (symptoms lasting less than one month) or persistent (symptoms lasting one month or more).<sup>17</sup> Symptoms include sensations of rocking, swaying, swinging and unsteadiness. There is no associated audiologic or vestibular symptoms present.<sup>17</sup> Most cases begin immediately on returning to land and usually last less than 24 hours, however, a small portion may develop persistent symptoms.<sup>17</sup> The pathogenesis behind mal de débarquement is thought to be secondary to a period of “re-adaptation.” When a subject is exposed to a provocative motion stimulus, he/she undergoes a period of adaptation. Once this adaptation process occurs, post-motion movements on land are awkward until a similar compensatory re-adaptation to the conditions on land occurs.<sup>17</sup> Risk factors for the development of mal de débarquement include prolonged sea voyages and rough sea conditions; and women are more susceptible than men.<sup>17</sup> Most cases of mal de débarquement resolve spontaneously, however, for the persistent forms, no pharmacologic agents have been found to be helpful. Several agents including scopolamine, meclizine, benzodiazepines, and amitriptyline have shown to have little or no benefit in helping with the symptoms of mal de débarquement. Despite their symptoms, patient should be encouraged to participate in activities that may enhance re-adaptation such as walking.<sup>17</sup>

Although most cases of mal de débarquement lasts less than 24 hours, Hain et al investigated persistent mal de débarquement, in which subjects experience symptoms for more than one month. Twenty-seven participants were recruited via an advertisement of the Vestibular Disorders Association in Portland, Oregon. The subjects were included in the study after filling out a questionnaire, which included demographic information, symptoms, onset and provoking factors and treatment used for their mal de débarquement. The subjects also had to report a sensation of rocking or swaying that persisted for at least one month following exposure to motion on an airplane or boat and the subjects had to have a physician diagnosis of mal de débarquement. Of the twenty-seven participants who met criteria to enter the study, 26 were female and 1 was male. This study population may be a result of persistent mal de débarquement being a disorder occurring almost exclusively in women. Additional results of the data showed that 15 (56%) of the subjects were between the ages of 40-49 at the time of onset of persistent mal de débarquement (See Figure 4) thus making it a disorder of middle age.

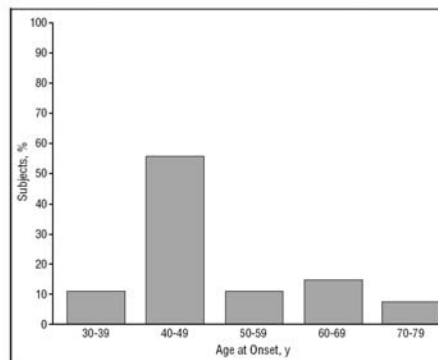


Figure 4. Age at onset of mal de débarquement syndrome, in decades  
(mean 49.3 years; SD 10.3 years)

Hain T et al Mal de Débarquement. Arch Otolaryngol Head Neck Surgery June 1999; 125

Other results obtained from this study included: the mean duration of symptoms was around 3.5 years; most subjects' symptoms were constant (85% of subjects); and that provoking factors of further motion exposure such as flights, car or boat rides was seen in 88% of the subjects.<sup>18</sup> Treatment options for persistent mal de débarquement remain limited. The questionnaire used in this study showed a positive effect of benzos and amitriptyline and no effect with meclizine and scopolamine.<sup>18</sup> More work is needed for a better understanding of persistent mal de débarquement and an "increased awareness among physicians may lead to the clinical knowledge necessary to develop effective treatment strategies."<sup>18</sup>

### Conclusion:

Motion sickness is the normal physiological response to real or perceived motion. There are several theories as to the cause of motion sickness, with the sensory-conflict theory currently being the most widely accepted. It is difficult to determine who is more likely to become motion sick and to what degree, however, several factors have been studied in the susceptibility to motion sickness including the type of motion and the personal characteristics of the subjects. It has been found that peak symptoms of motion sickness are found at 0.2 Hz and that women and children are more likely to develop motion sickness. Several therapeutic measures have been studied to prevent the symptoms of motion sickness including behavioral modalities, which include controlled, regular breathing, acupressure, and a variety of pharmacologic treatments.

The behavioral and alternative methods have had varied results. Pharmacologically, scopolamine has been the most widely studied and has shown significant benefit to target motion sickness symptoms (nausea) when compared to placebo.

Mal de débarquement refers to the sensations of movement after exposure to motion. Symptoms usually include sensations of rocking or swaying in the absence of audiologic or vestibular signs or symptoms. Most cases begin immediately on returning on land and last less than 24 hours, however persistent mal de débarquement can develop. Effective treatment strategies for mal de débarquement have yet to be discovered and developed.

The topic of motion sickness still has many areas to be studied. There is still no general consensus as to why motion sickness occurs. Also, the individual differences in susceptibility to motion sickness are still poorly understood. In addition, there needs to be further studies regarding the treatment of motion sickness, as current medications prevent motion sickness but do not treat it once it has occurred. Answering these questions will further enhance our knowledge of motion sickness, thus making traveling on boats, planes and even in outer space much more enjoyable.

## References

1. Shupak A, Gordon C. Motion Sickness: Advances in Pathogenesis, Prediction, Prevention, and Treatment. *Aviation, Space, and Environmental Medicine* December 2006; 77(12): 1213-1223.
2. Golding J. Motion sickness susceptibility. *Autonomic Neuroscience*. Oct 2006; 129(1-2): 67-76.
3. Yolton R, Citek K, Coffey B, Laukkanen H. Etiology and Management of Motion Sickness: A Review of Optometric Considerations 2006: <http://opt.pacificu.edu/ce/catalog/13459-GO/Motionsick.html>.
4. Bos J, MacKinnon S, Patterson A. Motion Sickness Symptoms in a Ship Motion Simulator: Effects of Inside, Outside, and No View. *Aviation, Space, and Environmental Medicine* December 2005; 76 (12): 1111-1118.
5. Oman CM. Motion sickness: a synthesis and evaluation of the sensory conflict theory. *Can J Physiol Pharmacol*. Feb 1990; 68(2): 294-303.
6. Gahlinger P. Cabin Location and the Likelihood of Motion Sickness in Cruise Ship Passengers. *J Travel Med* May-Jun 2000; 7(3): 120–124.
7. Spinks AB, Wasiak J, Villanueva EV, Bernath V. Scopolamine (hyoscine) for preventing and treating motion sickness. *Cochrane Database of Systematic Reviews* 2007, Issue 3.
8. Golding J, Gresty M. Motion Sickness. *Current Opinion in Neurology* 2005; 18:29–34.
9. Cooper C, Dunbar N, Mira M. Sex and seasickness on the Coral Sea. *Lancet*. Sept 1997; 350(9081): 892.
10. Bos JE, Damala D, Lewis C, Ganguly A, Turan O. Susceptibility to seasickness. *Ergonomics* Jun 2007; 50(6): 890-901.
11. Yen Pik Sang FD, Billar JP, Golding JF, Gresty MA. Behavioral methods of alleviating motion sickness: effectiveness of controlled breathing and a music audiotape. *J Travel Med*. Mar-Apr 2003; 10(2):108-11.
12. Stern R, Jokerst M, Muth E, Hollis C. Acupressure Relieves the Symptoms of Motion Sickness and Reduces Abnormal Gastric Activity. *Alternative Therapies* July/August 2001; 7(4): 91-94.
13. Miller K, Muth E. Efficacy of Acupressure and Acustimulation Bands for the Prevention of Motion Sickness. *Aviation, Space, and Environmental Medicine* March 2004; 75(3): 227-234.
14. White, B. Ginger: An Interview. *American Family Physician* June 2007; 75(11): 1689-1691.

15. Mowrey D, Clayson D. Motion Sickness, Ginger, and Psychophysics. *Lancet* Mar 1982; 1(8273): 655-657.

16. Otto B, Riepl RL, Otto C, Klose J, Enck P, Klosterhalfen S. mu-Opiate receptor agonists -- a new pharmacological approach to prevent motion sickness? *Br J Clin Pharmacol.* Jan 2006; 61(1): 27-30.

17. Lanka D. Mal de débarquement. November 2006:  
<http://www.medlink.com/medlinkcontent.asp>.

18. Hain T, Hanna P, Rheinberger M. Mal de Debarquement. *Arch Otolaryngol Head Neck Surgery* June 1999; 125: 615-620.