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Mal de Debarquement

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Abstract

Mal de debarquement (MdD), the "sickness of disembarkment," occurs when habituation to background rhythmic movement becomes resistant to readaption to stable conditions and results in a phantom perception of self motion typically described as rocking, bobbing, or swaying. Although several studies have shown that brief periods of MdD are common in healthy individuals, this otherwise natural phenomenon can become persistent in some individuals and lead to severe balance problems. Increased recognition of MdD in a persistent pathological form occurred after the publication of a case series of six patients by Brown and Baloh in 1987. Over 20 years later, although more is known about the clinical syndrome of persistent MdD, little is known about what leads to this persistence. This review addresses the clinical features of MdD, the associated symptoms in the persistent form, theories on pathogenesis, experience with treatment, and future directions for research.

Keywords

Mal de debarquement; persistent MdD; motion sickness; visual motion intolerance; vestibular adaptation

THE CLINICAL SYNDROME

An 1881 article in the *Lancet* by J.A. Irwin alluded to mal de debarquement (MdD) when describing the gait imbalance experienced by sailors when they tried to walk on the ground after being at sea[1]:

"This leads to the question of how all the phenomena of seasickness have usually a rapid tendency to pass away… In the same way upon which the ocean habit teaches the canals to adapt themselves to the new condition of this, and to pass over unheeded erroneous impressions of which were noticed at first. In fact, the new habit may become so strong that a disturbance of it, by a return to the land, will be marked by similar phenomena; hence the unsteady gait sometimes observable in a non-drunken sailor during his first few hours on shore after a long and stormy voyage."

Though many clinical syndromes may have similarities to MdD, the true definition of MdD requires a phantom self-motion perception that occurs after exposure to passive motion (Table 1). The body of case series literature to date indicates that the most common triggers for MdD involve water-based activities such as traveling on boats or ships (Table 2),[2-6] but MdD can also occur after other passive motion exposures such as air travel, extended land travel, or even less common triggers like sleeping on a moored boat and/or sleeping for one night on a

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waterbed.[2-6] Though MdD is a form of a phantom motion perception, it is never described as true rotational vertigo, and patients typically do not describe feelings of motion sickness. It is typically described as “rocking,” “bobbing,” or “swaying,” or with phrases such as, “[I feel like] I'm still on the boat,” or as if they are “walking on uneven ground.”

Short durations of phantom motion perceptions experienced after passive motion exposure are familiar to most people, but it appears to be unusual to experience the symptoms for 3 or more days. However, a rigorous prospective study of all passengers disembarking from a cruise or plane would have to be performed to truly estimate the normal duration. In two studies of crew of small seagoing vessels, Gordon et al showed that short durations of MdD are quite common. In their first study of 234 crew (average age of 20.5 years), 171 (73%) experienced MdD. Of these, 127 experienced MdD immediately upon returning to land with an additional 42 experiencing MdD within 6 hours.[7] In 159 subjects (93%), the symptoms lasted 6 hours or less and all were asymptomatic within 24 hours. There was no difference in the susceptibility to MdD based on experience at sea, but there was a direct correlation with susceptibility to seasickness.

The same group reported detailed features of the behavior of MdD in a follow-up survey of 116 crew members (average age 20.4 years).[8] Seventy-two percent reported a history of at least one episode of transient MdD in their lifetime. All subjects were men who engaged in sea travel in small vessels with average voyages lasting 5 to 8 hours. The latency to onset of MdD was immediately upon returning to land in 46% of subjects and within 1 hour in 80% of subjects; only 6% reported onset after 2 hours. MdD symptoms improved in 88% within 6 hours. Only two of the 116 subjects had symptoms lasting up to 48 hours.

This questionnaire study was followed up with a detailed interview of 30 subjects. Twenty-five reported exacerbations of the symptoms with eye closure, head tilt or lying down. Most subjects (66%) reported that the incidence of MdD was higher (more frequent) after earlier voyages. These studies showed that brief periods of MdD are common in otherwise healthy young men with short durations of motion exposure.[8]

A study by Cohen reported on MdD symptoms in 59 crew members (36 men, 23 women, average age 44.3 years) sailing for 4 days on a 117-year-old sailing vessel with each trip lasting 5 hours. The MdD experienced by crew members was very mild with symptoms only lasting up to 12 hours. Mal de Debarquement occurred in 37% of crew after the first day at sea, in 41% after the second day, and in 20% after the third and fourth days. It should be noted that in this study, the number and make-up of the crew varied each day. Though the sample size was small, there was a trend toward subjects who had worked aloft on the sails experiencing more MdD than those who had worked on deck. Crew who had worked on the sails would have experienced more motion because the top of the mast moves across a greater distance than the ship itself.[9]

PERSISTENT MAL DE DEBARQUEMENT

Although these studies were very important in describing the behavior of active MdD symptoms, they were not able to capture the rare patients who present for medical care for persistent MdD symptoms. It is this group of patients whose symptoms challenge physicians, as diagnostic tests fail to explain their symptoms, there is little information available on natural history, and therapeutic options are quite limited.

The age of onset of all patients with persistent MdD reported in the literature ranges from 15 to 77, but the most common age of a first episode of persistent MdD is in the late 30s to late 40s. Case series of persistent MdD show an excessive female preponderance not likely to be due to ascertainment bias (Table 2).

The average duration of persistent MdD symptoms is difficult to estimate, but studies report episodes lasting from weeks to decades. Though spontaneous resolution of the motion hallucination can occur even after several months, the probability of recovery becomes poorer the longer it lasts. In a UCLA study of 64 patients who had together experienced 206 distinct episodes, the probability of resolution of an individual episode dropped significantly with time, with the largest change occurring at one year.[3] The longer the symptoms lasted, the less likely they were to spontaneously remit. There were no cases of resolution of symptoms that lasted 5 or more years regardless of treatment. Though MdD has been referred to as a benign disorder with spontaneous resolution,[7,10] it is clearly a persistent and probably permanent disorder in many patients.

A fascinating feature shared by most patients with MdD is that reexposure to passive movement temporarily mitigates the internal rocking perception.[3,4,8,11] Reexposure to the initial motion trigger and typically driving or riding in a car give temporary relief, but it may also temporarily exacerbate the symptoms after the patient stops being passively moved.[4] In the UCLA study of 64 patients with MdD seen between 1980 and 2006, 34 were able to be contacted in the study year to complete a questionnaire and either a phone or office interview to detail features of their symptoms. Of the 34 subjects, 32 had clear responses regarding motion effects on the rocking perception. Of these 32 subjects, 22 only had pure motion triggered MdD episodes. The other 10 reported developing some spontaneous MdD-like episodes in addition to pure motion triggered MdD. Seventy-seven percent (17 of 22) of the pure MdD group reported decreased symptoms with reexposure to passive motion. None of these subjects reported worsening symptoms with passive motion. In the group with some spontaneous attacks, four subjects reported improved symptoms of their spontaneous onset of rocking when passively moved. One person reported worsened symptoms with movement and five reported no difference with movement. These results were similar in degree to those reported by Hain, in which 17 of 27 subjects (63%) with persistent MdD reported improvement in symptoms while driving.[4] In Gordon's study, 18 of the 30 (60%) interviewed subjects reported symptom relief when returning to the ship.[8]

ASSOCIATED SYMPTOMS

It is not known whether patients with persistent MdD are more susceptible to classical motion sickness, mainly because the true population rate of motion sickness cannot be adequately assessed. In Gordon's initial study of 234 crew of small seagoing vessels, there was a correlation between subjects who developed seasickness and those who developed transient MdD.[7] However, because the majority of subjects developed MdD, this relationship may not be that specific. Moreover, patients who develop persistent MdD usually develop it after exposure to large oceangoing vessels where there is a much less probability of seasickness. In Hain's questionnaire study of 27 people with persistent MdD, seven of the 21 individuals who had taken a cruise had used some kind of motion sickness prevention during the exposure that had led to MdD.[4] Estimates of the population prevalence of motion sickness has ranged from 21 to 59%[12-14] depending on the mode of transportation and experience of the subject. Females report a greater susceptibility to motion sickness than males.[15,16] Motion sickness-provoking laboratory maneuvers do not correlate well with a clinical history of motion sickness, so finding a good physiologic marker of motion sickness has been difficult.[17] It still remains to be determined how a reported history of motion sickness correlates with MdD because there are likely few individuals who, when sufficiently provoked, do not develop some degree of nausea, unless they lack peripheral vestibular function.[18]

One recently recognized phenomenon is that a significant number of patients with persistent MdD become less tolerant of visual motion during their MdD episode.[3] This has been described in various contexts such as turning pages of sheet music, playing video games, watching action

movies, or moving through visually complex environments. They did not report increased self-motion sickness, i.e., seasickness, carsickness, etc., after the onset of MdD. Studies on bilateral vestibular deficient subjects show that although they are less susceptible to self-motion sickness, they are still susceptible to nausea induced by optokinetic stimulation and other illusions of optical flow,[19] indicating that the pathophysiology of self-motion induced motion sickness and visually induced motion sickness are distinct and dissociable.[20] The mechanism of increased visual motion intolerance remains to be revealed, but at least two properties of visuovestibular interaction may be relevant. One is that the visual system can stabilize low-frequency postural sway.[21] If vision is being depended on to "anchor" head and body position in the setting of a vestibular system which is constantly indicating spontaneous motion, the patient may become less tolerant of rapid changes in the visual surround. Second, functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) studies in the last decade have shown that the visual and vestibular systems exert reciprocal inhibitory activity on each other.[22-24] This may be of functional significance to reduce visuovestibular conflict. If the motion perceptions of MdD represent continuous activity within the vestibular system, one consequence may be suppression of visual processing areas.

Some neurotologists consider MdD to be related to a history of migraine, but the relationship is not straightforward. The high female prevalence is shared by both MdD and migraine, and both disorders are reported to show symptomatic fluctuations with the female menstrual/ovulation cycles. However, in Hain's study, only 22% of 27 subjects met criteria for migraine.[4] Our own study also showed that 22% of the subjects with pure motion triggered MdD had a history of migraine, which is not significantly higher than the population baseline of ~17.1% in women and 5.6% in men.[25] However, the prevalence of migraine was significantly higher at 73% in patients who developed *spontaneous* perceptions of rocking, similar to MdD after an initial motion triggered MdD episode.[3] Paralleling this, when rocking perceptions occur spontaneously without any motion trigger, they usually occur in a background of migraine (unpublished data). These patients are typically younger, experience many more episodes, do not have as reliable a history of relief with reexposure to passive motion, and motion sickness symptoms may be more prominent.[3] There is no agreed upon nomenclature for this group, which has variably been called spontaneous MdDS, MdDS-like (where MdDS stands for Mal de Debarquement syndrome), migraine-related dizziness, migraine-related vertigo, and chronic rocking dizziness. Until a better understanding of the pathophysiology of the symptoms is obtained, there is no rationale for further descriptive terms.

A variety of additional symptoms develop with persistent MdD, but these are generally not emphasized in the published literature, most likely because these symptoms are often difficult to measure or to separate from the typical adjustment disorder that happens with chronic illness. Individual reports emphasize treatment of an associated mood disorder, particularly anxiety.[26] Many patients do develop depression and anxiety in relation to their disorder and because of the lifestyle changes required by their symptoms.[3] There is agreement among experienced neurotologists that MdD is not primarily a psychiatric disorder, as might be insinuated by those not familiar with this syndrome. However, it is important to address the development of depression and anxiety, particularly if it interferes with the treatment of the underlying disorder. Neurotologic disorders that get entangled with untreated mood disorders portend a poorer prognosis.[27]

A commonly reported, but overlooked symptom associated with MdD is the development of cognitive difficulty. Patients often refer to this as "brain fog." No study has systematically studied this, but numerous discussions with patients point to difficulties in working memory. Besides the difficulty in balance, the inability to multitask is reported as being one of the main reasons why patients limit or stop their professional and social activities. A fundamental question is whether the cognitive slowing is simply due to the distracting and distressing nature

of this persistent movement hallucination, or whether cognitive processing areas are actually affected.[28] Patients also report excessive fatigue, which requires them to slow down their activities or to take breaks during the day. The reason for this is not known, though possibly could be related to the extra effort exerted to maintain balance. Interestingly, this phenomenon was reported by Graybiel in a 1965 report of the clinical effects of living in a rotating room for 12 days.[29] In these experiments, four physically fit motivated military officers (2 Navy, 2 Marine) lived in a room rotating at 10 RPM for 12 days. They all experienced nausea and sometimes vomiting in the first few days of the experiment. However, even after this period had passed, they suffered from persistent fatigue and drowsiness leading to frequent naps during the day and unrestful sleep. Of note, these subjects never completely adapted to this environment, and they experienced less than 2 hours of gait imbalance when the experiment ended.

THEORIES ON PATHOGENESIS

The sensory rearrangement model proposed by J.T. Reason would explain this phenomena as one in which an internal model for how multiple sensory information is processed readapts to the new condition in which there is background persistent movement.[30] Visual, proprioceptive, cognitive, and somatosensory information would have to be weighed against a new pattern of vestibular input. Once the subject adapts to the novel environment, a return to the stable condition requires a new period of recalibration and readaptation. Prior to this readaptation, the subject would experience an abnormality in vestibular perception.

Moeller et al have suggested that what may be occurring is a release of “vestibular memory,” in that stored vestibular information from the background motion exposure becomes reexperienced as a hallucination.[26] They drew parallels with Charles Bonnet syndrome, in which visual deprivation leads to bizarre often very vivid visual hallucinations. They presented one patient who had developed MdD in the setting of Charles Bonnet syndrome, parkinsonism, and a sensory neuropathy with the suggestion that the patient was subject to multiple domains of sensory “release” phenomena.

Others have postulated that the disorder may be due to utricular dysfunction because of the usually linear properties of the motion perceptions,[6] but it is difficult to imagine how the motion exposures that subsequently lead to MdD could cause utricular dysfunction. A head tapping test has been proposed as a utricular specific test, with abnormalities in ocular contractions occurring contralateral to the problematic utricle,[31] but no specific tests of utricular function have been reported in MdD. The more commonly used vestibular evoked myogenic potential (VEMP) test, which involves a series of loud clicks to activate an inhibitory saccular-cervical pathway,[32] has been used clinically by many neurotologists, but the reports are that this test is not clinically useful in MdD. It is best applied to patients who have sound or pressure-induced vestibular symptoms, which are suggestive of canal dehiscence[33] or perilymphatic fistulas.[34]

All reports of vestibular function testing in MdD patients have either been normal or nonspecifically abnormal.[2,3,6] Two papers reported spontaneous positional nystagmus or high vertical/low horizontal gains on the vestibular autorotation test, but both findings are nonspecific, can be artifactual, or are seen in asymptomatic individuals.[2,6] One would not predict endorgan damage in MdD, as the exposures that trigger an MdD episode have no mechanism of damaging the peripheral vestibular system, and there are no symptoms at the time of exposure that indicate vestibular dysfunction. Similarly, structural imaging with brain MRIs are uniformly normal or show nonspecific changes.[3]

In essence, MdD is a disorder of neuroplasticity. The short-lived vestibular inputs experienced in normal landbased activities are not long enough to allow entrainment of central vestibular pathways. However, the persistent background oscillations of the typical environments that

lead to MdD send periodic and predictable inputs into the vestibular system. The development of an internal representation of this new external environment is crucial to allowing the unconscious maintenance of balance control. This is the process of developing one's “sea-legs.” This period of initial adaptation is what allows the spontaneous postural adjustments necessary to maintain balance on a moving boat. Our studies showed that when patients developed more than one episode of classical MdD, subsequent episodes tended to be longer. Some patients also developed the spontaneous reemergence of MdD symptoms after the initial motion triggered MdD episode had subsided. These phenomena indicate that memory of an externally oscillating world is maintained somewhere, and that this memory can be kindled or reactivated. A fundamental issue is where the memory of such an external world is stored.

CENTRAL MOTION PROCESSING AREAS

Vestibular information is processed in numerous brain areas, but there is no primary vestibular cortex. All vestibular processing areas have contributions from other sensory modalities that contribute head and body position information.[35] There are, however, some key areas that process vestibular information that may be particularly salient for vestibular memory. The human homologue of the parietoinsular vestibular cortex (PIVC) in macaque is the center of a network of cortical areas that process vestibular information. These other areas include Brodmann's area 2v, 3a, 6, and 7ab.[36,37] The PIVC is located deep in the Sylvian fissure in the posterior parietal operculum. fMRI with galvanic stimulation[38] and PET imaging with caloric stimulation show that a posterior insular area corresponding to the PIVC robustly activates in humans.[24]

The PIVC has also been shown to respond to acceleration of *visual* stimuli, but has differential activation based on whether the acceleration is congruent with gravity.[39] This indicates that the PIVC not only responds to true gravitoceptive input from otolith organs, it also can extract gravitoceptive information visually and maintains an internal representation of an earth-centered reference, rather than simply responding to all acceleration vectors equally.

Considering the development of visual motion intolerance in MdD, another candidate region is the dorsal aspect of the medial superior temporal area. The medial superior temporal area receives both visual and vestibular input, and is involved in detecting direction of heading. It has recently been determined to be an important center for determination of self-motion perception.[40,41] The medial superior temporal area lies just anterior to the motion sensitive area, which is typically located at the junction between the inferior temporal sulcus and the ascending limb of the inferior temporal sulcus.[42] Medial superior temporal area neurons are activated by optical flow and by translational and rotational vestibular signals.[41] Some are activated when both visual and vestibular information provide congruent heading information; others are activated when this information is incongruent.[40]

The hippocampus is widely known to be involved in the storage of episodic memory, but there may be an infrastructure in place to store vestibular memory. It has long been known that “place” cells within the hippocampus show location-specific firing,[43] whereas “space” cells fire when an animal is looking at a particular environment.[44] Experience can remap the place and space fields.[45-47] The importance of vestibular input to the hippocampus is evidenced by studies that show that peripheral vestibular loss leads to deficits in spatial learning, even leading to hippocampal atrophy.[48] Functional imaging studies show that the hippocampus can be activated by caloric stimulation,[49] and single cell recordings show that hippocampal neurons can be remarkably specific in their responses to visual and vestibular information.

Many other multimodal sensory processing areas may be involved in MdD owing to the numerous cortical areas receiving vestibular signals. Functional imaging studies may someday show which areas are key. In many ways, MdD behaves like tinnitus, inasmuch as both

conditions may represent a state of continuous reverberating cortical or subcortical activity. Functional imaging studies with both ^{18}[F]FDG and ^{15}O[H2O] in tinnitus have shown increased baseline hyperactivity in primary and secondary auditory cortices in tinnitus sufferers.[50-53] Interestingly, although the perception of tinnitus may be bilateral, there is usually unilateral hyperactivity in the left auditory cortex.[54] Theoretically, such elevated baseline hyperactivity in one of various regions that process self-motion perception could be associated with a persistent hallucination of motion. No study has evaluated this yet, but as the only medications that provide significant relief to MdD patients are benzodiazepines, it may suggest that a baseline continuously hyperactive focus may exist.

TREATMENT

Most cases of MdD resolve spontaneously without any specific treatment, but for patients whose symptoms last for months, the options are limited. There is general consensus that benzodiazepines provide the best symptomatic relief, with clonazepam being favored for its longer half life. Most practitioners use a dose between 0.25 mg twice daily to 0.5 mg twice daily. Higher doses are not more effective. The symptoms are never completely relieved with benzodiazepines, but in patients in whom there is an effect, the medication allows them to sleep and to regain some degree of balance function in the daytime. The issue, as with all indications for benzodiazepine use, is sedation and the development of tolerance and dependency. Selective serotonin reuptake inhibitors (SSRIs) can be very helpful in a limited number of patients, so are generally worth a trial. They are used by some neurotologists as baseline treatment, with benzodiazepines used as needed.

In our previous study of MdD patients,[3] 34 of 64 patients with MdD were able to be contacted to query their responses to treatment based on the following criteria: 1 = greatly relieved symptoms, allowing return to work or socializing; 2 = moderately improved symptoms; 3 = small but noticeable improvement; 4 = minimal improvement, if any; 5 = no improvement at all; 6 = made symptoms worse. Table 3 details therapeutic responses obtained from one study.

If MdD is a disorder of habituation to a set of vestibular, visual, and somatosensory signals, it would seem logical that focused rehabilitation would help in restoring the proper weighting of sensory inputs to maintain balance in stable conditions. However, the response to physical and vestibular therapy is often fairly unimpressive. It is most helpful for patients who are fearful of walking and who overrestrict their activities. Vestibular therapy aims to restore integration of different sensory inputs and reweighting the balance between those inputs if one is unreliable. It is also very helpful in enhancing central compensation for acute vestibular injury. A prior study using a posturography apparatus indicated that sailors most prone to developing MdD tended to place more weight on somatosensory input as opposed to visual or vestibular input.[55] However, all of these sailors had rapid resolution of MdD symptoms, so increased reliance on somatosensory cues does not explain persistent MdD.

It may be that some individuals are not able to dampen spontaneous central vestibular activity with the neutral cues of a stationary environment. The only purposeful activity that seems to null this internal signal is to add in other low-frequency oscillating vestibular input, i.e., return to the boat, drive, or rock oneself. The process may be akin to how noise cancelling headphones use active noise control. These devices generate an antinoise sound wave that is of the opposite polarity as incoming sound to destructively interfere with the bothersome noise before it arrives to the ear. Random spontaneous head movements do not make the symptoms better (and often make them worse) so it is not simply a matter of overriding the “internal noise.”

FUTURE DIRECTIONS

There has been growing awareness of MdD through the efforts of the MdDS Balance Foundation, neurotologists, and patients with the disorder. However, the disorder is still largely unrecognized by most physicians who may encounter such a clinical history. One important goal, therefore, is to increase awareness of the disorder so that patients receive a timely diagnosis and are spared the emotional toll that unnecessary diagnostic testing and unproven empiric therapy can take.

Second, little is known about the prevalence of the disorder and how long prolonged symptoms last. An unbiased survey of all passengers disembarking from a cruise or a long airplane flight would need to be performed to determine how often MdD really occurs, how long it lasts, and whether there were any experiences on the vessel that predispose certain groups of passengers to developing MdD. Cooperation from the cruise industry is necessary to accomplish this important goal, and efforts should be made to encourage collaboration. Moreover, if therapy is to be tried in these patients to prevent persistent MdD, early identification is key. In the current state, patients are diagnosed by a small group of specialists who are often seeing them after the symptoms have been persistent for many months or years. In the UCLA study, 39% of patients had already had at least one MdD episode lasting more than one year, indicating that there is a long delay in diagnosis.[3] Third, a better assessment of the associated symptoms of MdD may shed light on the pathophysiology of persistence. For example, a better quantification of auditory or mood symptoms may indicate a similar baseline “heightened” activity. Certainly, the reported cognitive symptoms and fatigue need to be more thoroughly investigated because these magnify the morbidity associated with the balance problems. Fourth, there may be hope in seeing functional imaging changes in patients with MdD much in the way that they are seen in tinnitus patients. There may be differences in the interactions between visual and vestibular cortices or there may be areas of hypermetabolism.

Finally, the ultimate question that all patients and their treating physicians have is when a cure for persistent MdD will be found. Creativity and collaboration will be needed in this realm. A multitude of pharmacologic treatments have been tried. Emerging therapeutic tools like repetitive transcranial magnetic stimulation or transcranial direct current stimulation, which have been used in the treatment of depression, tinnitus, chronic pain, and movement disorders, are areas to explore in MdD. Focused rehabilitation programs designed specifically for MdD patients keeping in mind the associated symptoms may help. Like these other disorders, MdD is likely a disorder of maladaptive cortical plasticity that will require some novel way to break the vicious cycle.

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Table 1

Major Features of Mal de Debarquement

Rocking, bobbing, swaying or any other self-motion perception that occurs after passive motion exposure such as on boats, airplanes, or cars
Transient improvement in symptoms with reexposure to passive motion (driving, sailing, etc.)
Symptomatic relief with benzodiazepines
Other symptoms can include visual motion intolerance, fatigue, cognitive slowing, and mood changes

Table 2

Mal de Debarquement (MdD) Publications

Year	Author	Subjects	Sex (F:M)	Age of Onset	Trigger	Duration
1987	Brown[2]	6	5:1	33–66	Boat (6), air (1)	Several months to 5 years
1992	Gordon[7]	234	0:234*	18–38	Boat (234)	<24 Hours
1993	Murphy[6]	4	4:0	35–48	Cruise (1), waterbed (1), scuba diving (1), cruise, motorboat, floating on raft (1)	4 Weeks to 1 year
1995	Gordon[8]	116	0:116	18–33	Boat (116)	<48 Hours
1996	Mair[5]	10	10:0	15–66	Boat (10), air (2)	3 Days to 2 years
1996	Cohen[9]	59†	23:36	Avg. 44.3±12.4	Boat	<12 Hours
1999	Hain[4]	27	26:1	35–72	Boat (25), air (2)	1–10 Years
2004	Lewis[11]	1	1:0	51	Car, air	Hours
2006	deFlorio[10]	1	0:1	22	Boat and air	18 Days
2007	Moeller[26]	2	1:1	34, 65	Boat (1), air (1)	2 Months, 12 years
2008	Cha[3]	64	48:16	Avg. 39±13	Boat (52), air (26), car (10)	3 Days to 22 years

*Presumed based on very similar demographics of follow-up study.

†Represents the total number of participants at risk for MdD, not the number who actually developed MdD. In any given day of five daily trips, 20 to 41% developed transient MdD.

Table 3

Therapeutic Responses to Mal de Debarquement

Benzodiazepine		**Anticonvulsant**	
Number scored*	12	Number scored	3
Average	2.6	Average	5.3
Median	2	Median	5
Antiemetic		**Acetazolamide or other diuretic**	
Number scored	14		
Average	4.6	Number scored	5
Median	5	Average	4.4
		Median	5
Selective serotonin reuptake inhibitor		**Decreased salt or diet modification**	
Number scored	13	Number scored	6
Average	2.9	Average	3.8
Median	3	Median	4.5
Tricyclic antidepressant		**Stress reduction**	
Number scored	9	Number scored	9
Average	4.3	Average	2.8
Median	5	Median	3
Beta blocker or calcium channel blocker		**Physical or vestibular therapy**	
Number scored	2	Number scored	15
Average	5	Average	3
Median	5	Median	4

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*Number scored=number of patients who had tried indicated therapy.