Survey of mal de debarquement

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Mal de debarquement, or "land sickness," is a transient sensation of tumbling, swinging, unsteadiness, and disequilibrium reported by passengers and crew after returning to land from long sea voyages. Although mal de debarquement is mentioned in classic texts on seasickness,[1,2] there are no specific studies concerning the nature and extent of this phenomenon. A computer search of the literature yielded only one publication dealing with persistent (not transient) mal de debarquement.[3] We report a survey of the incidence of mal de debarquement and its relation to experience at sea and susceptibility to seasickness.

Subjects, methods, and results

The information was collected by means of questionnaires distributed among 234 healthy crew members of seagoing vessels (300-500 tonnes). Subjects were aged 18-38 (mean 20·5) years and had 1-150 months' experience at sea (mean 17 (SD 18) months). None had a history of any disease which might cause vertigo or disequilibrium.

The occurrence and frequency of mal de debarquement were graded on a four point scale (very often, occasionally, only once, never). Latency from disembarkment to the onset of symptoms, duration of symptoms, and additional causative factors such as rough seas and length of voyage were also recorded. Present susceptibility to seasickness was graded on an eight point scale (not susceptible to very susceptible) according to Wiker *et al.*[4,5] Other parameters of susceptibility, such as nausea and vomiting during most recent voyages in rough seas and susceptibility to seasickness in the past,[2] were also graded.

One hundred and seventy one subjects (73%) reported having experienced mal de debarquement, 20 (9%) very often, 86 (37%) occasionally, and 65 (28%) only once. Mal de debarquement appeared immediately on returning to land in 127 (74%) of the 171 subjects and within six hours in 169 (99%). Its duration ranged from a few minutes to 24 hours (mean 156 (SD 308) minutes). In 159 (93%) subjects the phenomenon did not last more than six hours. The appearance of mal de debarquement was frequently related to a prolonged sea voyage (115 (67%) subjects) and to rough sea conditions (75 (44%)). The table lists the relevant correlations between parameters.

Kendall correlation coefficients

	Occurrence of mal de debarquement	p Value	No
Experience at sea	0·072	0·157	230
Present susceptibility to seasickness	0·290	0·0001	228
Nausea*	0·273	0·0001	228
Vomiting*	0·225	0·0001	231
Past susceptibility to seasickness	0·245	0·0001	227

*During most recent voyages in rough seas.

Comment

This study shows that mal de debarquement is a benign, transient picture of a tumbling or swinging sensation but not true vertigo. The phenomenon seems to be quite common among crew members of fairly small seagoing vessels (73%). None of our subjects suffered persistent mal de debarquement[3] or requested medical attention.

The occurrence of mal de debarquement was positively correlated with all the parameters of susceptibility to seasickness but was not correlated with experience at sea—that is, both inexperienced and experienced crew members had the sensation to a similar degree.

The nature of mal de debarquement, its short latency and limited duration, and its relation to prolonged sea voyages and rough sea conditions can be explained within the framework of sensory adaptation to ship motion. Passengers and crew on board ship are exposed to a series of unnatural and conflicting vestibular, visual, and proprioceptive stimuli which, according to the neural mismatch and sensory rearrangement theory, may cause seasickness and, at the same time, adaptation to specific ship motion.[2] This adaptation is often expressed in the familiar sensation of "getting one's sea legs," using the leg muscles to oppose the motion of the waves. After return to land these newly acquired sensorimotor patterns are no longer appropriate, giving rise to mal de debarquement, which lasts until readaptation is achieved.

Further studies measuring vestibulo-oculoproprioceptive interactions during mal de debarquement are warranted in order to clarify the neurophysiological basis of this phenomenon.

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Ranitidine, aspirin, food, and the stomach

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Optimal prophylaxis against development of peptic ulceration in patients taking aspirin or non-steroidal anti-inflammatory drugs remains controversial. Clinical studies of patients taking these drugs have shown ranitidine 150 mg twice daily to be highly effective in preventing the development of duodenal ulceration but to have less effect on gastric damage.[1] Higher doses could, however, afford greater protection since microbleeding induced by aspirin can be reduced to placebo levels by giving high doses of the proton pump inhibitor omeprazole.[2] Patients are advised to take these drugs with food, but there are few data on the validity of this advice. Indeed, parenteral indomethacin has been shown to induce antral ulcers in rats that have been fed but not fasted.[3] We therefore investigated the effects of standard and higher doses of ranitidine on gastric mucosal injury induced by aspirin and whether this was affected by timing of dose in relation to food.

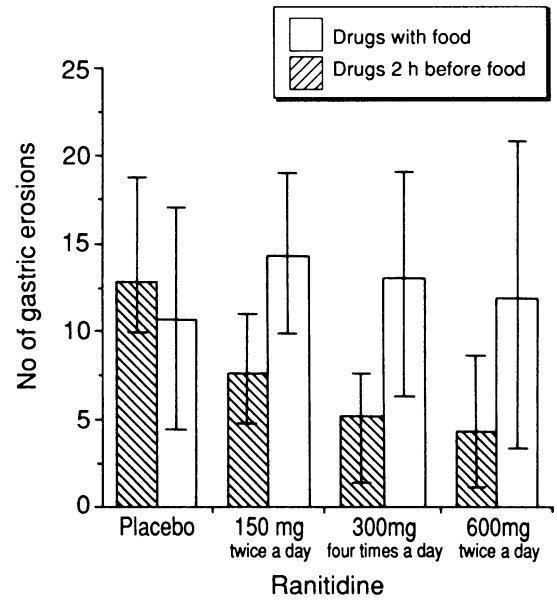
Subjects, methods, and results

Twenty healthy volunteers (13 men, seven women, aged 19-30 years) with normal results on screening endoscopy were given aspirin 600 mg four times a day

for five days on four different occasions. During each of these four dosing periods the subjects received concurrent treatment with placebo, ranitidine 150 mg twice a day, ranitidine 600 mg twice a day, or ranitidine 300 mg four times a day, each given simultaneously with aspirin. All subjects received each prophylactic regimen. During each treatment period the subjects were allowed to eat only at 9 am, 2 pm, 8 pm, and midnight. Half the subjects always took drugs at the same time as food. The other half always took their drugs two hours before food (at 7 am, midday, 6 pm, and 10 pm). The order in which subjects received the different treatment regimens was randomised by Latin square design. There was a washout period of nine days between each treatment regimen.

Subjects were studied in the morning, approximately eight hours after the last doses had been taken. Spontaneous microbleeding was measured as previously described,[4] followed by unsedated endoscopy with a paediatric endoscope, when erosions in the body, antrum, and duodenum were counted. Statistical analysis was by two way analysis of variance, with treatment and timing of dose in relation to food as the determining variables.

Aspirin increased the number of gastric erosions from none at baseline to a median of 10·6 (drugs given with food) or 12·8 (drugs given before food) (figure). Ranitidine reduced the total number of gastric erosions in a dose dependent fashion when the drugs were taken two hours before food ($p=0·006$), but had no effect when taken with food. Overall, taking drugs before food was associated with a significant reduction in mucosal injury ($p=0·003$) in comparison to taking them with food.



Effect of ranitidine and food on total number of gastric erosions in subjects given 2·4 g aspirin daily. Bars indicate 95% confidence intervals

Comment

Higher doses of ranitidine were more effective than standard doses, but only when the drugs were taken two hours before meals. The most plausible explanation for this finding is that this regimen achieves greater acid inhibition than when the drugs are given with food. However, an alternative explanation—that coadministration of food increases the toxicity of aspirin—remains possible. Although there was no difference in the number of erosions developing in the absence of ranitidine, injury may simply be maximal under these circumstances and differences may become apparent only under the protection of ranitidine. In support of this proposition, rats given indomethacin showed a dose dependent relation between the amount of food ingested and the extent of antral injury.[4] Faecal blood loss in humans taking aspirin with food has been reported to be higher than when aspirin was given without food, though the differences did not reach significance.[5] Thus, conventional advice to take aspirin and possibly other non-steroidal anti-inflammatory drugs with food may in fact be wrong. In any case, ranitidine together with aspirin offers greater mucosal protection if the drugs are taken two hours before meals rather than with food.

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Lipoprotein(a) in cirrhosis

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The serum concentration of lipoprotein(a) is a strong independent risk factor for the development of premature coronary heart disease.[1] Studies in patients undergoing liver transplantation suggest that lipoprotein(a) is synthesised in the liver.[2] To determine the influence of liver disease on lipoprotein(a) concentrations we compared concentrations in patients with varying degrees of severity of hepatic cirrhosis, controls, and patients with established coronary heart disease.

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Subjects, methods, and results

Thirty patients (aged 27-71 years) with histologically diagnosed cirrhosis were matched for age and sex with healthy controls (hospital/university staff and relatives, 22-69 years) and patients with established coronary heart disease (26-68 years), all with normal liver function. Cirrhosis was secondary to chronic alcohol intake (24 patients), chronic active hepatitis (five), and haemochromatosis (one), and patients were clinically stable. Concomitant treatment included diuretics (five) and prednisolone (two). The severity of liver disease was assessed independently by using the Child Turcotte classification, with 10 patients in each group—A (mild), B (moderate), and C (severe). Lipoprotein(a) concentrations were determined by an enzyme linked immunosorbent assay (ELISA) (Biopool, Tint Elise) (coefficient of variation 7·6%) on fasting serum samples stored at −20°C. Statistical assessment was by Wilcoxon rank sum and correlation by least square regression analysis.

Lipoprotein(a) concentrations were raised in patients with coronary heart disease and reduced in those with cirrhosis (figure). Concentrations tended to be lower in those with more severe disease but this trend was not significant. Lipoprotein(a) was not