# Motion sickness a guide to prevention and treatment

A GP can be a helpful source of advice for dealing with this common problem. Appropriate management takes into account the length of the trip, the severity of the stimulus and the individual's susceptibility, and includes not only pharmacological

treatment but also general measures to minimise the sickness.

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Dr Cooper is a Lecturer, Department of General Practice, University of Sydney, NSW. The term 'motion sickness' describes a syndrome of nausea, vomiting and palpitations that occurs on exposure to motion. In its mild form, the syndrome is a ubiquitous part of everyday life. Road and air travel are now commonplace, and sea travel, which is one of the most provocative motion environments, has undergone a renaissance of popularity in the last decade.<sup>1</sup>

In the fields of occupational and military medicine, motion sickness has important financial and logistical implications,<sup>2,3</sup> and motion sickness associated with complex 'virtual' environments is becoming a common feature of a variety of recreational settings.<sup>4</sup>

As a consequence, it is not uncommon for general practitioners to be consulted by patients for prophylaxis and treatment of the syndrome. The purpose of this article is to review current understanding of motion sickness and to provide clear practical guidelines for its management.

- IN SUMMARY
- Prolonged motion sickness is associated with malaise, anxiety, depression, apathy and a decrease in attention and concentration; however, most individuals will adapt to prolonged stimulus and experience complete recovery in three to four days.
- The prevalence of motion sickness is related primarily to the severity of the stimulus and the susceptibility of the individual.
- General measures to minimise the individual's reaction include: eating small, infrequent, low kilojoule meals before and during travel; focusing on a fixed point outside the vehicle or closing the eyes when this is not possible; reducing head movements; engaging in a mental activity or task.
- Hyoscine and antihistamines are the drugs of choice in the prevention and treatment of motion sickness.
- The efficacy and side effects of different drugs vary greatly from one individual to another, therefore if one drug is ineffective or not well tolerated it is useful to try another.

#### **Clinical syndrome**

The clinical syndrome of motion sickness is stereotyped and predictable in its evolution, although differences exist in the speed of progression and the severity of symptoms.

The earliest symptom is a sensation of epigastric heaviness which has been aptly described as 'stomach awareness'.<sup>5</sup> With further exposure to the stimulus, this progresses to the cardinal symptom of nausea, which is accompanied by an autonomic reaction clinically indistinguishable from acute anxiety (palpitations, pallor, diaphoresis).<sup>5</sup>

A feeling of warmth may produce a desire to seek cool air, which typically produces temporary, albeit transient, relief. Common additional features include sighing, yawning, eructation, flatulence, headache and dizziness. When the stimulus is insufficient to produce nausea, an excessive desire to sleep may be the only manifestation of the syndrome.

At some point, the symptoms undergo a sudden exacerbation ('the avalanche phenomenon') which culminates in vomiting.<sup>6</sup> Vomiting brings relief, but this is only temporary and is followed by cyclical nausea and vomiting, which may continue for days. Prolonged motion sickness is associated with malaise, anxiety, depression, apathy and a decrease in attention and concentration. Vomiting may be complicated by dehydration and electrolyte disturbances.<sup>7</sup>

The final stage – the acquisition of complete adaptation – is characterised by the complete resolution of symptoms. While adaptation exhibits significant inter-subject differences, a majority of individuals experience complete recovery in three to four days.<sup>8</sup>

#### Pathogenesis

Motion sickness is not, in fact, a sickness at all, but a physiological adaptation to a changed external environment.<sup>9</sup> Why a physiological response should produce such unpleasant symptoms has long been a matter of conjecture. Analogous physiological adaptations (for example, the adaptation of vision to darkness) do not produce unpleasant symptoms, and it may be that the function of motion sickness is to provide a warning signal, anaogous to pain, to withdraw from a potentially dangerous environment

Irrespective of the environment involved, prolonged stimulation gives rise to a process of sensory adaptation, which takes about three days to develop. This process of adaptation has a number of readily demonstrable characteristics. Firstly, it exhibits a high degree of specificity for a given environment. Darwin, for example,

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# Summary of general management of motion sickness

- Adapt to the offending stimulus by regular and repeated exposure.
- Eat small, infrequent, low kilojoule meals before and during travel.
- Choose vehicles with a soft, low frequency cycle of oscillation.
- Sit up on the deck of the ship, or in the front seat of the car.
- Establish external visual orientation with the horizon.
- Close the eyes when external visual orientation is not possible.
- Avoid gazing at moving objects outside the vehicle.
- Avoid scanning objects inside the vehicle.
- Avoid reading.
- Avoid visual contact with objects that stimulate the peripheral visual field.
- Use a head restraint (or voluntarily reduce head movements).
- Engage in a mental activity or task.
- When supine, lie along the longitudinal axis of the vehicle with the head in the direction of travel.

described how sailors who were adapted to life on board ship became seasick when they were transferred to a rowing boat.<sup>10</sup>

Secondly, adaptation produces a disturbance in perception on return to the 'normal' surroundings. This is illustrated by the well known disequilibrium experienced by sailors (*mal de debarquement*) on return to land, and by astronauts who report nausea and dizziness on return to Earth.<sup>11</sup>

Finally, adaptation is progressively lost if re-exposure to motion does not occur. Horatio Nelson, one of the greatest seafarers of all time, always became seasick after losing his 'sea legs' during a period on land. 'It never continued for very long', wrote Leavesley in a chapter about Nelson, 'but the embarrassment lasted all his life'.<sup>12</sup>

# **Prevalence and susceptibility**

It is a well established axiom of neurology that motion sickness affects all individuals providing they have an intact vestibular system and are exposed to an adequate stimulus.<sup>13</sup> Only abnormal individuals, with a nonfunctioning vestibular system, escape its effects. This is in accordance with epidemiological studies that have shown a prevalence rate varying from 1 to 100%.<sup>14</sup>

Prevalence has been shown to be related primarily to the severity of the stimulus and the susceptibility of the individual at risk.

# Stimulus severity

Sea travel carries the greatest risk of motion sickness and is related to vessel size and sea state.<sup>15</sup> Seasickness is encountered in 7% of passengers on board coastal passenger ferries,<sup>16</sup> 25% of sailors on a small naval vessel in storm conditions, and almost 100% of subjects unlucky enough to find themselves aboard a life raft in heavy seas.<sup>17</sup>

The prevalence of airsickness is related to the size of the aircraft and the degree of turbulence. The prevalence varies from 1% of passengers on early propeller-driven airliners to 8% of passengers on small, low-altitude civil aircraft.18 Airsickness aboard large, modern, civil passenger aircraft, which fly at high altitudes and encounter little turbulence, is anecdotally very uncommon. The highest recorded rates of airsickness (50%) occur among pilots of high performance aircraft used in pilot training19 and aboard aircraft engaged in hurricane penetration flights  $(90\%)^{20}$ 

Car sickness is less prevalent but more common overall because road use is ubiquitous. About half (47%) of undergraduates reported having suffered car sickness between the ages of 12 and 20 years.<sup>21</sup> Train sickness is rare (0.1%) aboard conventional trains,<sup>22</sup> but anecdotal reports suggest that it may be more common aboard modern high speed trains.<sup>5</sup>

# Individual susceptibility

#### Age, gender and ethnicity

Motion sickness is more common in women than men.<sup>22</sup> The reason for this is unknown, but observations that the incidence is high during menstruation and pregnancy may suggest a hormonal mediation.<sup>23</sup>

Motion sickness decreases in prevalence with age.<sup>22</sup> It is rare before the age of 2 years, reaches a maximum incidence between the ages of 3 and 12 years, decreases rapidly between the ages of 12 and 22, then continues to decrease, albeit more slowly, throughout adult life.<sup>22</sup>

Asian ethnicity carries greater risk of motion sickness than European or African ethnicity, suggesting susceptibility may be in part genetically determined.<sup>24</sup>

# Other factors

One of the most intriguing features of motion sickness is the very wide differences in individual susceptibility even after age, gender and ethnicity are taken into account.

Past experience may be a factor in some cases. Resistance to motion sickness has been shown to develop among individuals, such as dancers and sailors, who are repeatedly exposed to motion, and some groups are known to develop specific motor strategies following exposure to specific motion environments.<sup>25</sup> Bus drivers, for example, have been noted to lean in the opposite direction to passengers when navigating a bend,<sup>26</sup> and dancers learn to break up a rotational movement by fixing on a reference point, then whipping the head around for most of the turn.<sup>27</sup>

However, past experience does not

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explain the large differences in susceptibility encountered among the vast majority of individuals who rarely encounter significant motion environments.

The sensitivity of the emetic centre and chemoreceptor trigger zone appears to play an important role in motion sickness susceptibility.

Individuals who are susceptible to motion sickness also show an increased susceptibility to nausea and vomiting due to migraine,<sup>28</sup> narcotics,<sup>29</sup> radiotherapy, chemotherapy, anaesthesia<sup>30</sup> and pregnancy,<sup>31</sup> and following head injury.<sup>32</sup> Patients receiving narcotics for pain are more likely to develop nausea if they are ambulant, suggesting that the effect of motion and other emetic stimuli may be additive.

The fact that susceptibility occurs irrespective of the trigger-zone involved (vestibular, chemoreceptor, cerebral) suggests that the basis of individual sensitivity resides centrally, rather than in a peripheral trigger zone such as the vestibular apparatus.<sup>29</sup>

# Management General measures

The box on page 52 summarises the general management of motion sickness.

# Avoiding stimuli

Motion sickness may be prevented by avoiding the offending stimulus. While this may sound simple it may not be practical or possible to forgo travel by car, aeroplane or boat, or to remove oneself from the offending stimulus once motion sickness has developed.

# Adaptation

Adaptation is the most effective longterm means of prevention. However, it takes three or four days to develop and is quickly lost if re-exposure does not occur, and so is not useful on short or infrequent trips.<sup>5</sup> Once achieved, adaptation should be maintained by regular and repeated exposure.

#### Choice of vehicle

The incidence of motion sickness has been found to be related to the frequency of the cycle of oscillation of the vehicle concerned. This is based on observations aboard ships, and from anecdotal reports of car travel in which vehicles with a soft, low frequency cycle of oscillation cause less motion sickness.<sup>15</sup> Benson has hypothesised that this is because the most provocative frequencies lie outside the natural frequencies involved in the normal locomotion of walking and running (0.5 to 10 Hz).<sup>5</sup>

#### Dietary intake

The risk of motion sickness is positively correlated with meal frequency before and during travel. Food content that is high in kilojoules and protein is most likely to predispose to motion sickness.<sup>33</sup>

#### External visual orientation

The severity of motion sickness is reduced by visual orientation with a fixed external point that does not appear to move when viewed by the observer. On board a ship, this is best achieved by visual fixation on the horizon.<sup>34</sup> (This technique was so successful among the crew of naval vessels that it led to consideration of the introduction of an artificial horizon in submarines.)

In a car, it is best to sit in the front seat of the vehicle and to focus on the distance of the road ahead.<sup>5</sup> Passengers in the back seat of a car, or in the enclosed cabin of a ship, are more prone to motion sickness for this reason.<sup>35</sup>

When visual orientation with the horizon is not possible, it is better to close the eyes rather than fixate on moving objects within the vehicle.<sup>35</sup>

#### Avoiding gazing at moving objects

Visual input that correlates poorly with vehicle movement, such as scanning objects inside a vehicle (e.g. a map or chart) or gazing at the motion of waves from a ship, accelerates the development of motion sickness. Reading is a particularly provocative stimulus.

#### Minimising head movement

Measures that minimise movements of the head have been shown to reduce the severity of motion sickness.<sup>5</sup> Restriction of head movements may be voluntary or aided by the provision of a good head support.

#### Mental activity

Mental activity and involvement in tasks that focus attention have been shown to reduce the incidence and severity of motion sickness.<sup>36</sup> This is the basis for the commonly reported observation that the driver of a car, pilot of an aircraft or captain of a ship are much less likely to suffer from motion sickness than are passengers.<sup>36</sup>

# Posture

Motion sickness is less prevalent when passengers are orientated along the longitudinal axis of the vehicle. Transportation of patients by ship and motor vehicle should be in the lying position with the head in the direction of travel.<sup>20</sup> The best position in a helicopter is sitting with the head upright.<sup>20</sup>

# Pharmacological management

Efficacy and choice of medication Anticholinergics (hyoscine) and antihistamines are the drugs of choice in the prevention and treatment of motion sickness. They are equally effective, although efficacy varies greatly from one individual to another.<sup>37</sup> Phenothiazines and related drugs (for example, metoclopromide and prochlorperazine) are not recommended because they are less effective and can cause acute dystonic reactions. Evidence for the efficacy of powdered ginger root<sup>38</sup> and acupressure bands is inconclusive.<sup>39</sup>

The options for prevention and

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#### Table. Drugs recommended for prevention and treatment of motion sickness

#### PREVENTION

#### Long journeys

- Promethazine oral (Avomine, Phenergan) taken one to two hours before travel, then once or twice a day, or
- Dimenhydrinate oral (Dramamine) taken one to two hours before travel, then every four hours

#### Short journeys

Hyoscine oral (Kwells, Travacalm HO) taken half an hour before travel, then every four hours

#### TREATMENT\*

- Hyoscine oral every four hours as required, or
- · Promethazine oral every six to 12 hours as required, or
- Dimenhydrinate oral every four hours as required, or
- Promethazine intramuscular injection (Phenergan, Promethazine Hydrochloride Injection) every six to 12 hours as required.

\* The efficacy of different drugs can vary greatly from one individual to another, so if one drug is ineffective or not well tolerated it is useful to try another.

treatment are shown in the Table. Both hyoscine and antihistamines are more effective when used prophylactically, although they may be used in treatment with less success. In general, the choice of a medication should be determined by its efficacy, its duration of action (how long the trip is expected to take) and how well it is tolerated.

#### Duration of action

Oral hyoscine (Kwells, Travacalm HO) has a half-life of four hours and is useful for prophylaxis on short journeys and for treatment of established illness (Table). Among the commonly used antihistamines, dimenhydrinate (Dramamine) has a half-life of about six hours,<sup>5</sup> and promethazine (Avomine, Phenergan) has a half-life of 12 hours (and is useful for prophylaxis on longer journeys).

Hyoscine may also be given as a transdermal preparation, which is as effective as the oral route, and carries the same risk of side effects.<sup>40</sup> However, it is not readily available in Australia. It should not be used in children.

# Side effects

Both hyoscine and antihistamines have side effects which limit their use. Both groups of drugs are central nervous system depressants which impair the ability to drive and operate machinery, interact with alcohol and other sedatives, and produce a measurable decrease in functional performance.<sup>41</sup> Hyoscine may produce anticholinergic side effects of a dry mouth and blurred vision; it is contraindicated in glaucoma, and should be used with caution in the elderly and patients with urinary retention or pyloric stenosis.

#### **Combined** preparations

When a single agent is less than effective, combined treatment using hyoscine plus an antihistamine (e.g. hyoscine plus dimenhydrinate [Travacalm]) is often effective, although care should be taken because the side effects are additive.

#### Parenteral administration

Parenteral administration is necessary when vomiting supervenes.<sup>42</sup> Most drugs may be given by intramuscular or intravenous injection. Intramuscular promethazine (Phenergan, Promethazine Hydrochloride Injection) has greater efficacy and longer duration of action (12 hours v. four hours) than intramuscular hyoscine (Hyoscine Injection).<sup>42</sup>

# Conclusion

Motion sickness is a common problem. It can ruin a person's sea or car trip, and it has important implications in the fields of occupational and military medicine. The prevalence is related primarily to the severity of the stimulus and the susceptibility of the individual. While adaptation is the most effective long-term means of prevention, general measures of minimising the reaction to the stimulus include: eating small meals during travel, focusing on a fixed point outside the vehicle or closing the eyes, reducing head movements, and engaging in a mental activity or task.

Hyoscine and antihistamines are the recommended drugs for prevention and treatment. In prevention, the choice of drug depends on the length of the journey. Because the efficacy and side effects of different drugs vary greatly from one individual to another, if one drug is ineffective or not well tolerated it is useful to try another. MI

A list of references is available on request to the editorial office.

#### **MOTION SICKNESS - A GUIDE TO PREVENTION AND TREATMENT**

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# References

1. World Health Organization. International medical guide for ships. 2nd ed. Geneva: WHO, 1998: 223.

2. Wright MS. The incidence and effects of motion sickness among medical attendants during transport. J Emerg Med 1995; 13: 15-20.

3. Chinn Hi. Motion sickness in the military service. Mil Surg 1951; 108: 20-29.

4. Regan EC. The efficacy of hyoscine hydrobromide in reducing side-effects induced during immersion in virtual reality. Aviat Space Environ Med 1996; 67: 222-226.

5. Benson AJ. Motion sickness. In: Dix MR, Hood JD, eds. Vertigo. Chichester: Wiley, 1984.

6. Reason JT, Brand JJ. Motion sickness. London: Academic Press, 1975.

7. Reason JT. Motion sickness: a special case of sensory rearrangement. Adv Sci 1970; 26: 386-393.

8. Groen JJ. Problems of the semicircular canal from a mechanicophysiological point of view. Acta Otolaryngol Suppl 1960; 163: 59-66.

9. Triesman M. Motion sickness: an evolutionary hypothesis. Science 1997; 197: 493-495.

10. Darwin E. Zoonomia or the laws of organic life. Vol 1. 3rd ed. London: J Johnson, 1801.

11. Gordon CR. Clinical features of mal de debarquement: adaptation and habituation to sea conditions. J Vestib Res 1995; 5: 363-369.

12. Leavesley JH. The common touch. A doctor's diverting look at fourteen famous patients. Sydney: Australian Broadcasting Corporation and Fontana, 1995: 47-48.

13. Brizzee KR, Igarashi M. Effect of macular ablation on frequency and latency of motion induced emesis in the squirrel monkey. Aviat Space Environ Med 1986; 57: 1066-1070.

14. Money KE, Wood JD. Neural mechanisms underlying the symptomatology of motion sickness. In: Fourth Symposium on the Role of the Vestibular Organs in Space Exploration, Report SP-1970; 187. Washington, DC: NASA, 1970: 35-44.

15. Wiker SF, Kennedy RS, McCauley ME, Pepper RL. Susceptibility to seasickness: influence of

hull design and steaming direction. Aviat Space Environ Med 1979; 50: 1046-1051.

16. Lawther A, Griffin MJ. Motion sickness in sea-going passenger vessels: an interim report. Southampton: Human Factors Research Unit, ISVR, Southampton University, 1981.

17. Brand JJ, Colquhoun WP, Perry WLM. Side effect of l-hyoscine and cyclizine studied by objective tests. Aerospace Med 1968; 39: 999-1002.

18. Lederer LG, Kidera GG. Passenger comfort in commercial air travel with reference to motion sickness. Int Med 1954; 167: 661-668.

19. Tobie DG. Airsickness in aircrew. Report AG-1 77. Neuilly-sur-Seine: AGARD/NATO, 1974.

20. Kennedy RS, Moroney WF, Bale RM, Gregoire HG, Smith HG. Motion sickness symptomatology and performance decrements occasioned by hurricane penetrations in CA 21, CA 30 and P-3 navy aircraft. Aerospace Med 1972; 43: 1235-1239.

21. Reason JT. An investigation of some factors contributing to individual variation in motion sickness susceptibility. Flying Personnel Research Committee Report No. 1277. London: Ministry of Defence (Air), 1967.

22. Cooper C. Sex and seasickness on the Coral Sea. Lancet 1997; 350: 892.

23. Ramsay TM. The menstrual cycle and nausea or vomiting after wisdom teeth extraction. Can J Anaesth 1994; 41: 798-801.

24. Stern RM. Asian hypersusceptibility to motion sickness. Hum Hered 1996; 46: 7-14.

25. Cremieux J, Mesure S. The effects of judo training on postural control assessed by accelerometry. In: Brandt T, Paulus W, Bles W, Dietrich M, Krafczyk S, Straube A, eds. Disorders of posture and gait. New York: Georg Thieme Verlag, 1990.

26. Kahane J, Auerbach C. Effect of prior body experience on adaptation to visual displacment. Perception Psychophysics 1973; 35: 279-285.

27. McCabe BF. Vestibular suppression in figure skaters. Trans Am Acad Ophthalmol Otol 1960; 64: 264-268.

28. Carvalho D de S. Risk factors in headache in children from 7 to 15. Arq Neuropsiquiatr 1987;

45: 371-378.

29. Yardley D. Dizziness and vertigo. London: Routledge, 1994.

30. Palazzo M. Logistic regression analysis of fixed patients factors for postoperative sickness: a model for risk assessment. Br J Anaesth 1993; 70:135-140.

31. Golaszewski T. Treatment of hyperemesis gravidarum by electrostimulation of the vestibular apparatus. Z Geburtshilfe Neonatal 1995; 199(3): 107-110.

32. Jan MM. Vomiting after mild head injury is related to migraine. J Pediatr 1997; 130: 134-137.

33. Lindseth G. The relationship of diet to airsickness. Aviat Space Environ Med. 1995; 66: 537-541.

34. Cowings P, Toscano WB. The relationship of motion sickness susceptibility to learned autonomic control for symptom suppression. Aviat Space Environ Med 1982; 53: 570-575.

35. Money KE. Motion sickness. Physiol Rev 1970; 50: 1-38.

36. Rolnick A. Why is the driver rarely motion sick? The role of controllability in motion sickness. Ergonomics 1991; 34: 868-879.

37. Brand JJ, Perry WLM. Drugs used in motion sickness. Pharmacol Rev 1992; 18: 895-924.

38. Stewart JJ. Effects of ginger on motion sickness susceptibility and gastric function. Pharmacology 1991; 42: 111-120.

39. Bruce DG. Acupressure and motion sickness. Aviat Space Environ Med 1990; 61: 361-365.

40. Shojaku H. Effect of transdermally administered scopolamine on the vestibular system in humans. Acta Otolaryngol Suppl (Stockh) 1993; 504: 41-45.

41. Parrot AC. Transdermal scopolamine: a review of its effects upon motion sickness, psychological performance and physiological functioning. Aviat Space Environm Med 1989; 60: 1-9.

42. Wood CD. Effectiveness and duration of intramuscular antimotion sickness medications. J Clin Pharmacol 1992; 32: 1008-1012.