

Motion sickness

雙和醫院 鄭芸詠

- Acute motion sickness arises during passive transportation in motor vehicles; after the disappearance of the inducing stimulus, it resolves spontaneously within 1 day at the most
- The full picture of acute severe motion sickness evolves with
- initial symptoms of:
 - ✓ Light-headedness or foggy head
 - ✓ Tiredness
 - ✓ Periodic yawning
 - ✓ Pallor
 - ✓ Light dizziness with apparent surround-motion and self-motion
- An increase in facial pallor is followed by cold sweats, increased salivation, hypersensitivity to smells, occipital head pain, and feelings of pressure in the upper abdomen.
- Finally, the central symptoms of nausea, retching, and vomiting develop with motor incoordination, loss of drive and concentration, and fear of impending doom

- Motion sickness is not caused by vestibular “over-stimulation” during strong accelerations of the body, but by unfamiliar (i.e. non-adapted) motion stimuli and particularly by **intersensory perceptual incongruences among the visual, vestibular, and somatosensory systems.**
- The most important concept explaining the pathogenesis of motion sickness is the so-called mismatch theory
- According to this theory, the decisive trigger is the incongruence of the signals of motion from various sensory channels or the incongruence between expected and actual sensory stimulation.
- other hypothetical mechanisms are also discussed (Bertolini and Straumann 2016), e.g., based on experiments suggesting that the spatial orientation of the velocity storage of the vestibulo-ocular reflex plays a role in the generation of motion sickness due to head movements

- Well-known varieties of motion sickness are:
 - ✓ Car sickness (visual-vestibular conflict of stimuli)
 - ✓ Sea sickness (unfamiliar, complex linear, and angular accelerations of low frequency, below 1 Hz)
 - ✓ Vehicle simulator or virtual reality sickness (optokinetic motion sickness)
 - ✓ Space sickness (incongruent sensory stimuli of the otoliths, semicircular canals, and the visual system during active head movements in microgravity)



A review on the effects of frequency of oscillation on motion sickness

Bob Cheung

Ann Nakashima

Abstract

In support of the ADVANCE TDP (Advanced Vehicle Architecture for a Net-Enabled Combat Environment Technology Demonstration Project), and at the request of Director Armoured Vehicles Program Management (DAVPM), we undertook to provide a Phase 1 assessment on the effects of motion disturbance on the performance of operators based on a theoretical and comprehensive literature review. A comprehensive review on the effects of motion disturbance on human behaviour and well-being in all forms of transportation was completed. Based on information collected, a summary of the motion frequency and amplitude on human response was presented graphically. The main findings can be summarized as follows: The majority of information is obtained from ship-simulator or ship motion where vertical (heave) motion is the primary stimulus. Vertical motion does not correlate with the rate of carsickness. Fore-and-aft and lateral motion in the frequency range of 0.1-0.5 Hz is provocative in inducing carsickness. Postures and type of back/head rest could influence susceptibility to motion sickness. Laboratory studies indicated that the ability of the active suspension to protect against or contribute to motion sickness is influenced by whether or not the compensation is under the active control of the rider. **Vertical motion frequencies below 0.5 Hz are generally more nauseogenic. Whole body vibration at 2 Hz and above can cause discomfort or injury but will not provoke motion sickness. Based on limited data, frequencies below 0.1 Hz lessen the possibility of motion sickness.** The effect of vibration along the horizontal (x and y) axes on performance is unknown. Our recommendations include field studies on the incidence and severity of motion sickness in Canadian Forces enclosed armoured vehicles followed by simultaneous measurements of vehicle vibration, and physiological, psychophysical, and human performance in an enclosed vehicle during moving and stationary conditions in the second phase of the investigation.

- Epidemiological studies have found a statistically significant association of migraine and susceptibility to motion sickness (Neuhauser and Lempert 2004; Evans et al. 2007; Cuomo-Granston and Drummond 2010), above all in vestibular migraine (Boldingh et al. 2011).
- The prevalence of motion sickness is higher in patients with some vestibular disorders, but lower in bilateral vestibulopathy, probably because in the latter the visuo-vestibular mismatch is less effective (Takahashi et al. 1997; Paillard et al. 2013; Murdin et al. 2015; Golding 2016; Golding and Patel 2017).
- A relevant comorbidity of most vestibular disorders with an increased susceptibility to motion sickness was confirmed in a prospective epidemiological study (Strupp et al. 2018).
- In this study, similar to that of Murdin et al.(2015), patients with bilateral vestibulopathy were not immune to motion sickness but more resistant.
- The frequency of motion sickness susceptibility from infancy to adolescence is characterized by three phases, a high resistance in the first year of life, a prepubertal peak of oversensitivity, and a postpubertal decline
- Pregnant women are particularly susceptible (Takov and Tadi 2019).

Visual Vertigo, Motion Sickness, and Disorientation in Vehicles

A.M. Bronstein, MD, PhD, FRCP¹ J.F. Golding, DPhil² M.A. Gresty, PhD¹

¹Division of Brain Sciences, Imperial College London, Charing Cross Hospital, London, United Kingdom

²Department of Psychology, School for Social Sciences, University of Westminster, London, United Kingdom

Address for correspondence John F. Golding, DPhil, Department of Psychology, School for Social Sciences, University of Westminster, London W1W 6UW, United Kingdom (e-mail: goldinj@westminster.ac.uk).

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➤ Susceptibility

- The elevated susceptibility of females to motion sickness or indeed to PONV or chemotherapy-induced nausea and vomiting may serve an evolutionary function.
- From this perspective, more sensitive emetic thresholds in females may serve to prevent exposure of the fetus to harmful toxins during pregnancy.

Course and Therapeutic Principles

- Despite considerable interindividual variation in resistance, every healthy individual can experience motion sickness when exposed to extreme acceleration stimuli (e.g., crosscoupled accelerations like Coriolis effects)
- Figures on the incidence of motion sickness in different motor vehicles vary between 1% and 90%. During the first days of an Atlantic crossing with moderate turbulence, about 25–30% of ship passengers experience motion sickness, whereas 80% of subjects on small life rafts or adrift with flotation vests become severely seasick.
- Women are more susceptible than men, and children and young adults are more susceptible than the aged.
- Newborns and infants up to the age of 1 year are extremely resistant to motion sickness, apparently because they use the visual system for dynamic space orientation only after they have learned to stand alone and walk. They obviously do not experience a provoking visual-vestibular conflict of self-motion perception while being transported in a vehicle (Brandt et al.1976).
- Loss of labyrinthine function causes higher resistance to motion sickness (Strupp et al. 2018), whereas eye closure on a ship does not prevent sea sickness.

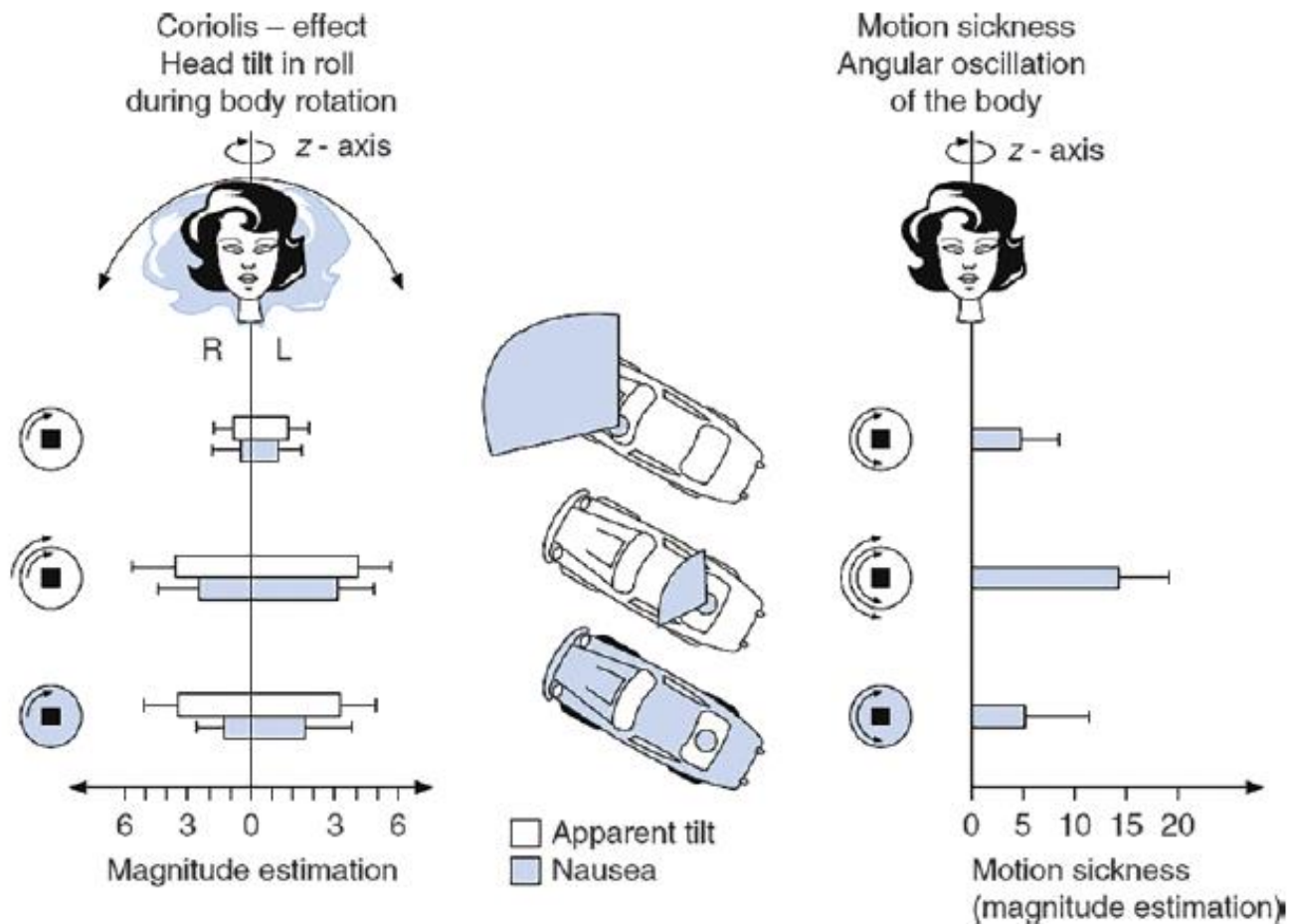


Fig. 19.1 Visual influences during body acceleration in a combined drum and chair system and their effects on vertigo and motion sickness. Left: magnitude estimations of apparent tilt and nausea induced by sideward tilts of the head during simultaneous body rotation while sitting. Coriolis effects are induced in the process by the cross-coupled accelerations (left). Magnitude estimation of motion sickness induced by 15 min of sinusoidal angular oscillation of the body on a rotatory chair with a frequency of 0.02 Hz and peak velocity of 100°/s (right). The three visual conditions were eyes open (top), rotary chair and drum movements mechanically coupled (middle),

and eyes open in complete darkness (bottom). The experimentally induced nausea was greatest when the vestibular and visual information on movement was contradictory (combined movement of chair and drum). This corresponds to the experience in moving vehicles. Motion sickness is least pronounced when vision can simultaneously check body acceleration (in the driver's seat); conversely, motion sickness is greatest when the vestibular acceleration apparently contradicts visual information (in the back seat with predominantly stationary contrasts in the visual field or when reading). (Reproduced with permission from Brandt et al. (1976))

- Motion sickness is an acute clinical syndrome.
- Nausea and vomiting develop within minutes to hours, and the symptoms show spontaneous remission within hours to 1 day after the stimulus ceases.
- If the stimulus continues (ship or space travel), relief occurs via centrally mediated adaptation (habituation) within 3 days (Koch et al. 2018).

- The most effective physical means of prevention of motion sickness is adaptation (habituation) by intermittent exposure to the stimulus (Koch et al. 2018).
- This adaptation, however, is only temporary and specific for each type of acceleration; i.e., resistance to sea-sickness does not protect against flight sickness.
- If “vestibular training” does not make the subject resistant, his/her head should be kept still during the stimulus, and additional accelerations that are complexly coupled with the vehicle motion should be avoided.
- Susceptibility is less when lying than when sitting (Golding et al., 1995) and less for the person in the front passenger seat than for someone sitting in the back seat (Takov and Tadi 2019).

- Motion sickness develops above all in closed vehicles or while reading on the back seat of a car, when the body is being accelerated but a stationary environment is being viewed, which contradicts the labyrinthine stimuli.
- By maintaining adequate visual control of the vehicle movement, one can significantly reduce motion sickness from that experienced under eyes-closed conditions.
- Conversely, susceptibility is significantly increased if primarily stationary contrasts fill the field of vision (Dichgans and Brandt 1973; Probst et al. 1982).

- Antivertiginous drugs such as dimenhydrinate (dramamine®) or scopolamine (transderm scop®) can inhibit the spontaneous activity of the vestibular nuclei neurons as well as the neuronal frequency modulation during body acceleration, thus reducing the susceptibility to motion sickness.



Pragmatic Therapy

- Doubling the usual single doses (100 mg dimenhydrinate; 0.6 mg scopolamine) clearly increases the central sedating side effects without causing any essential improvement in resistance to motion sickness (Wood et al. 1966).
- A combination of scopolamine with a sympathicomimetic drug, such as ephedrine or dextroamphetamine, increases the efficacy with simultaneous reduction of sedative adverse effects (Wood and Graybiel 1970; Golding 2016).
- Because of the dependence caused by dextroamphetamine, a combination with this substance is not generally recommended but reserved for exceptional cases, e.g., during military flight missions (Leung and Hon 2019).
- Phenytoin was also tested for efficacy against motion sickness (Knox et al. 1994); however, in view of its side effects, it is also not recommended (Murdin et al. 2011).

Table 19.1 Physical maneuvers to prevent motion sickness

Measure	Goal
<i>Before</i>	
“Vestibular training” by repeated exposure to stimulus and active head movements	Movement-specific central habituation
Vehicle simulator training	Utilization of the visual-vestibular transfer of habituation
<i>Acute</i>	
Fixation of the head	Avoidance of additional accelerations, which are complexly coupled with vehicle acceleration (e.g., Coriolis effect)
Head position (toward the vector of gravitation). Ship: supine; car: supine with head in the direction of movement; helicopter: sitting position	Utilization of the head-axis-specific difference in resistance to the acceleration; acceleration along the z-axis most favorable
Possible counter-regulation of the body motion induced by vehicle acceleration (e.g., inclining toward the curve)	
Visual control of the vehicle movement; if not possible, then close the eyes	Avoidance of a visual-vestibular conflict (mismatch) of perception

Table 19.2 Drug prophylaxis for motion sickness

Drugs	Side effects
Antihistamine 50–100 mg dimenhydrinate (Dramamine)	Sedation, reduced reactions and concentration, dryness of the mouth, blurred vision, light-headedness
Belladonna alkaloids 0.5 mg scopolamine as transdermal therapeutic system (Transderm Scop) 4–6 h before trip onset, effective up to 72 h	

- Thank you!!!