Mechanisms and Treatment of Motion Illness

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Introduction

The question of why man develops motion sickness may be approached from several points of view. The human species is not alone in being susceptible to motion sickness: an equivalent pattern of symptoms can be induced by appropriate types of motion in a wide range of mammals (e.g. monkeys, horses, dogs, cats, seals) in birds, and even in fish. To the evolutionist, the problem is to understand what the survival value is of a pattern of symptoms evoked by certain types of motion stimulus that culminates in vomiting. From the practical point of view, it is necessary to know what the characteristics of those forms of motion are which lead to motion sickness as compared with those that do not. Such information also gives some indication of the likely neurophysiological mechanisms involved in motion sickness. From the point of view of the physiologist, interest is focussed on the mechanisms by which orientation and motion are sensed and on the sequence of events both within the central nervous system (CNS) and in other body systems that constitute the syndrome of motion sickness. Individual susceptibility in man varies widely and attempts have been made to find psychological and physiological measures that correlate with susceptibility. A further insight into the causal mechanisms of motion sickness comes from observation of the drugs which offer some protection. Such prophylactic drugs are drawn from several pharmacological groups, an indication that several neurotransmitters may be involved in the sequence of events leading to motion sickness.

Symptomatology of Motion Sickness

The sequence of symptoms and signs that constitutes motion sickness is fairly characteristic. Premonitory symptoms often include yawning or sighing, lethargy, somnolence and a loss of enthusiasm and concern for the task in hand. Increasing malaise is directed towards the epigastrium, a sensation best described as “stomach awareness”, which progresses to nausea. Diversion of blood flow from the skin towards the muscles results in pallor. A feeling of warmth and a desire for cool air is often accompanied by sweating. Frontal headache and a sensation of disorientation or lightheadedness may also occur. As symptoms progress, vomiting becomes increasingly likely. In some subjects vomiting occurs early in the se-

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quence of symptoms, in others malaise is severe and prolonged and vomiting is delayed.

After vomiting, there is often a temporary improvement in well being, though, with continued provocative motion, symptoms again build up and vomiting recurs.

This stage of the condition is humorously summed up by the adage, “for the first hour you fear that you will die, by the second hour you fear that you won’t”. However, the threat to life resulting from the repeated vomiting of sea sickness may be very real, for example aboard life rafts if rescue is delayed.

A number of objective physiological measures show changes as motion sickness develops. Plethysmography confirms the redistribution of blood flow from skin to muscles, but there are no consistent changes in pulse rate or blood pressure. There is some degree of hyperventilation and end-tidal CO2 is reduced and arterial blood pH increased (Sinha 1968). The galvanic skin response (GSR) is a sensitive indicator of sweating, and changes can be detected on those areas of skin associated with emotive or thermal sweating (McClure et al. 1972). Oliguria during motion sickness results from an increased secretion of anti-diuretic hormone, and there is a concomitant though less dramatic increase in other pituitary hormones in particular growth hormone, prolactin and adrenocorticotrophin (Eversmann et al. 1977). These hormone responses are similar to those that occur in response to other forms of physical and surgical stress.

**Characteristics of Stimuli that Provoke Motion Sickness**

There are numerous circumstances in which motion sickness can be produced, many of them having been given specific names: sea sickness, car sickness, swing sickness, camel sickness, cinerama sickness, simulator sickness and most recently, space sickness. However, it is not necessary for the subject himself to be in motion in order to develop symptoms. Pilots flying fixed base aircraft simulators and members of an audience watching large screen films taken from a moving vehicle (for example, an aircraft performing aerobatics) may experience symptoms of motion sickness while they themselves remain stationary. What are the attributes of these various types of motion, either real or perceived, that lead to motion sickness? The most coherent explanation for the development of motion sickness is provided by the sensory conflict theory.

**Sensory Conflict Theory**

The body makes use of several sensory modalities to contribute to a perception of orientation in space and of changes in orientation that imply that the body is in motion. The suggestion that motion sickness may result from conflict between these various sensory modalities was made as long ago as 1881 by Irwin. The concept was further elaborated by Claremont (1931), Brooks (1939) and more recently by Reason (1970). Motion with respect to the outside world is sensed