The Evidence Based Etiology of Space Motion Sickness

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Space Motion Sickness (SMS) was reported on the second orbit of the second manned flight. Almost five decades later there is no satisfactory etiology [1]. Virtually all approaches to the problem to date are based on the concept of motions which produce conflicting responses from different sensory modalities. [1,2] Characteristic symptoms are triggered by such sensory conflicts through vestibular inputs to the chemoreceptor trigger zone. Both symptoms and courses of motion sickness (MS) and SMS differ. SMS symptoms are consistent with demonstrated otolith organ function [3] and observed and recorded inflight effects [4]. The crux of vestibular effects of weightlessness is inherent in the graviceptor sensors of the otolith organs. Functionally they are weighted single axis pendulum accelerometers, spring loaded to a neutral position in the absence of displacing force. Thousands of these transducers are arranged geometrically to sense any orientation of the head to gravity. In weightlessness the variously oriented sensors all indicate a gravity vector normal to their mounting axis. This produces an intrinsic single modality sensory conflict which causes a paresis of the gastro-intestinal tract and produces the episodic vomiting and other symptoms characteristic of primary SMS. [5] Also, in weightlessness the otolithic sensors cannot produce their usual signals in response to angular head motion. This produces a bimodal sensory conflict with semi-circular canal signals, even with normal head motions and probably accounts for the characteristic of MS sometimes seen in SMS [2]. Thus weightlessness causes anomalous otolithic signals with both an intrinsic conflict and a bimodal motion dependent conflict. These produce differing symptoms apparently through different connections to the gastrointestinal system [4].

References