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Dennis K. McBride

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BIO MOTION PROJECT

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Dennis K. McBride
David A. Graeber
Elizabeth M. Sheldon

Institute for Simulation and Training
University of Central Florida
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INSTITUTE for SIMULATION & TRAINING

PREPARED BY

UNIVERSITY OF CENTRAL FLORIDA
INSTITUTE FOR SIMULATION AND TRAINING
3280 PROGRESS DR.
ORLANDO, FL 32826

POC: Dr. Thomas L. Clarke, (407) 882-1327, tclarke@ist.ucf.edu

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Evolutionary function of motion sickness

The existence of motion sickness, its prevalence through a variety of species, and its persistence throughout evolution has perplexed motion sickness researchers pursuing plausible explanations. Scientists are developing predictive theories that encompass a plethora of known motion sickness inducing environments. Despite the quantity and quality of effort, there is frustration. Part of this is because the neurological underpinnings of motion sickness are not fully understood, as will be discussed in subsequent sections on the evolution of sensory integration. Meanwhile, the philosophical focus in provocative motion study has taken a characteristic, engineering approach—partly because the goal of many of such efforts has been to solve extant design problems. This makes sense.

We suggest that by taking a biological—moreover an evolutionary—approach, we may gain insight into the true nature of the malady. In fact, that we consider motion sickness a "disorder" is telling. Perhaps its ultimate origins and function were of adaptive value during say, Pleistocene, and that the phenomenon has become maladaptive in its proximate, contemporaneous manifestation. In any case, through systematic tracing of the emergence, elaboration, and mechanisms of motion sickness in the phylogenetic record, we believe that there are clues to its adaptive nature. From such findings, we derive and offer a tentative, integrative model of motion sickness, one that may be substantiated subsequently.

For a comprehensive overview of motion sickness, particularly that of Homo sapiens, the reader should consult Thomas Dobie’s excellent book, Motion Sickness: A Motion Adaptation Syndrome (in press). In fact, there is much human-related motion sickness phenomenology that the reader might consult, such as its prevalence, and age and sex differences in susceptibility (e.g., Dobie, McBride, Dobie, & May, 2001). These issues are beyond the scope of this report.

Evolutionary Development and Persistence of Motion Sickness; Current Theories

Money (1990) addressed the issue of motion sickness and evolution based on evolutionary assumptions. He talks about various “givens” in regards to motion sickness and evolution, one of these being that motion sickness stimuli were not integral in the evolutionary development of motion sickness mechanisms. Another given is based, of course, on the work of Darwin and Wallace (1858): physiological mechanisms that offer reproductive advantage are selected for; those that are disadvantageous are selected against. Money (1990) suggests that based on the aforementioned assumptions, the physiological components of motion sickness did not ascend by chance and then endure due to a lack of evolutionary pressure to select against it. However, it is perplexing that motion sickness appears to have no function (e.g., survival value), particularly because it reflects a physiological mechanism, and physiological mechanisms are normally associated with functions.

In an effort to explain the function of motion sickness, various theories have arisen, three of which are reviewed very briefly. The first is Treisman’s (1977) poison hypothesis which claims that motion sickness, particularly vomiting, is a protective response against poisons that create sensory conflict by affecting the processing of visual and vestibular information. He suggests that vomiting would protect against the poison’s effects, and the associated negative experience would reinforce memory (i.e., one trial learning) for foods or substances to avoid. Empirical evidence partially supports Treisman’s poison hypothesis. Most notably among them are Money and Cheung’s (1983) work on bilateral labyrinthectomy in dogs in conjunction with the administration of modest levels of toxins. In addition, the work of Ossenkopp and Tu (1984), Wilpizeski et al. (1987a), and Lambert et al. (1989) support the learning aspects of Treisman’s
theory. However, the poison hypothesis of motion sickness has been criticized because by the
time a toxin enters the body and affects the vestibular system, vomiting to expel the toxins still in
the stomach may be of little use for protection (Yates, Miller, & Lucot, 1998; Guedry, Rupert, &
Reschke, 1998).

Yates, Miller, and Lucot (1998) suggest an alternate explanation to the poison hypothesis. They note that individuals afflicted with vestibular system diseases often experience motion
sickness symptoms, particularly nausea and vomiting, that may prompt the individuals to remain
sedentary. Taking an evolutionary viewpoint, it could be argued that the sickness and inactivity
are forms of positive adaptation to vestibular disease to keep the individual from harming
themselves. Interestingly, it has been suggested that the function of emesis is not only the
expulsion of stomach contents, but also to keep a disoriented or dizzy individual from moving
about the environment in search of food when he or she would be at risk doing so (Longbridge,
1983). Therefore, as suggested by Yates, Miller, and Lucot (1998), it is possible that motion
sickness is the result of a wayward activation of the vestibular nerve and related central nervous
system components that maintain postural stability, not a poison response.

Guedry, Rupert, and Reschke (1998) put forth the third evolutionary explanation of
motion sickness, proposing a motor learning component. In essence, they suggest that motion
sickness results from inefficient movements and prevents further development of these
inefficient perceptual-motor programs. They hypothesize that:

Innate displeasure from conflict involving the vestibular system is a mechanism that
operates in addition to and irrespective of the presence or absence of reward and
punishment afforded by parental care and goal attainment. This reaction continuum,
pleasure-displeasure, is a mechanism by which the spatial orientation system is trained
and conditioned to develop perceptual motor programs that are efficient in the operating
environment of the individual (Guedry, Rupert, & Reschke, 1998; pg. 479).

Guedry, Rupert, and Reschke (1998) also provide an evolutionary explanation for the emetic
response that occasionally accompanies motion sickness. They suggest that while the motion
sickness symptoms provide a powerful means for learning, the quick relief from symptoms
following emesis allows learning to proceed when adaptation is required.

In reviewing the various theories on motion sickness and evolution, it is apparent that no
one theory dominates. Furthermore, theories that attempt to explain the mechanisms of motion
sickness (e.g., sensory conflict theory, subjective vertical model of sensory conflict, postural
instability theory, and eye movement hypotheses) are post hoc explanations that are not proven
predictive theories and are often limited to particular sensory environments. As a result, the true
function of motion sickness has not been agreed upon among motion sickness researchers and
may remain an intriguing mystery until its neurological basis has been fully unraveled. We will
provide a fourth explanation below; one which also serves to integrate the three current
explanations. However, in an effort to better understand the phenomenon of motion sickness, a
review of the evolution of sensory systems and sensory integration is necessary.

Evolution of sensory systems and sensory integration
The various sensory modalities are thought to have evolved from a “supramodal,”
primordial system that is not very selective about what it responds to. It is believed that in a
supramodal system, all effective sensory stimuli (e.g., chemical, thermal, mechanical, radiant,
etc.) entail comparable consequences based on stimulus intensity. High-intensity stimuli are thought to elicit avoidance or withdrawal behaviors, whereas low-intensity stimuli elicit approach behaviors (Stein & Meredith, 1993). The development of specialized receptors sensitive to a particular form of stimuli is thought to be the result of the evolutionary process of specialization, thereby creating sensory differentiation (Marks, 1978; Butterworth, 1981).

Support for a single, nonspecific ancestral sensory receptor is lacking, but it is plausible that the earliest primordial organism consisting of a single cell would have been supramodal. The most primitive eukaryotic organisms are unicellular. Living examples evolved from species that appeared about 1.4 billion years ago (Stein & Meredith, 1993). Eukaryotes derived from prokaryotes (the most ancient unicellular organism—living bacteria are extant examples) in a marine environment and unlike the prokaryotes, they developed internal organelles (e.g., mitochondria, true cilia, miotic spindles, nuclei). Early unicellular organisms also probably led diverse sensory lives, more than might be expected of a unicellular organism (Stein & Meredith, 1993). A salient example of unicellular organisms that can be studied today is the *Paramecium* (a ciliated protozoan). This organism provides a very suitable beginning for a discussion on the development of sensory systems and their integration.

Touch- and stretch-induced receptor potentials are common among, and even predate, protozoa (Morris, 1990). Interestingly, the mechanisms by which these primordial organisms mediate the sensory functions of transduction and excitation are fundamentally similar to the mechanisms in higher organisms (Hille, 1984). In the *Paramecium*, ion channel specificity exists, with distinct channels for receptor and for action potentials (Ogura & Machemer, 1980). In addition, it is believed that receptor specificity exists in the case of protozoa with "eyespots" (van Houten & Preston, 1988). This suggests that protozoa, at the cellular level, are supramodal because they are unicellular organisms responding to two or more sensory stimuli (e.g., at least mechanical and chemical sources). Therefore, *Paramecium*, in a cellular context, is a clear example of elemental multisensory integration because its behavior is a direct result of ongoing synthesis of stimuli that its membrane is responsive to.

As more complex organisms evolved, an increase in sensory specialization and segregation also occurred. The initial step in sensory segregation occurred in early multicellular invertebrates (Stein & Meredith, 1993), for example, sponges. Mackie and Singla (1983) note that sponges are the phylogenetically the lowest of multicellular species and consist solely of a network of cells (i.e., they do not possess nerves or muscles). However, Mackie and Singla (1983) note that sponges are able to transmit information from an area of receptor activation to the entire cellular mass via protoplasmic continuity. They also suggest that sponges are able to identify various stimuli because they are observed ingesting food and retracting from harmful stimuli. However, this ability to discriminate between various stimuli does not appear to require modality segregation nor diversion of information to specialized groups of cells. Similarly, in some primitive coelenterates, such as some species of medusae, sensory signals are transformed via conductive epithelia (Mackie & Passano, 1986). Mackie and Passano (1986) note that the sensory signals are simultaneously transmitted in all direction, thereby negating the potential for sensory segregation. However, Stein and Meredith (1993) note that it was within this phylum that nerve cells, synapses, and nerve nets first developed. In addition, the authors indicate that this phylum was the first in which cell groupings formed specialized sensory organs, for example, sense organs for detecting bodily rotation, vibration, and photic stimuli. While the development of specialized sensory organs provides the means for sensory segregation, there is no evidence that it was actually achieved in coelenterates. Instead, it has been shown that in
coelenterates, impulses can pass through multiple cells, both neural and nonneural, via electronic coupling (Anderson & Schwab, 1982). This may allow diverse sensory stimuli to access a large percentage of the organism, thereby, making all cells multisensory.

Stein and Meredith (1993) note that a jump in sensory system complexity and response flexibility evolved in the nervous system of the phylum Platyhelminthes (flatworms). The development, embellishment, and fusion of ganglia produced an encephalized, bilaterally symmetrical organism (i.e., not radially symmetrical, like the coelenterates) containing the first brain and peripheral nervous system (i.e., plexus). While elongated axons had begun development in coelenterates via nerve rings and giant motor axons, they are utilized more effectively in Platyhelminthes by allowing transmission of information over long distances, bypassing unnecessary cells. This provided the ability for the first segregation of stimulus inputs and distinct response outputs to segregated stimuli.

It should also be mentioned that sensor integration is clear in water-borne organisms more sophisticated than the coelenterates. Clearly exemplary is observation in Lollinguncula brevis (squid) by Press and Budelmann (1995):

When illuminated from the side in visually homogeneous surroundings, a free-swimming squid rolls the dorsal side of its head and trunk 10-20 degrees towards the light. With the trunk restricted in a holder, the squid rolls its head 4-5 degrees towards the light; this reaction increases by about 50% when the statocysts are bilaterally removed and increases further when the neck receptor organ is also destroyed. The results indicate a multi-modal interaction of visual, statocyst and proprioceptive inputs during postural control (p. 1157).

Stein and Meredith (1993) suggest that capacity for concurrent segregation and integration among sensory modalities occurred relatively early in the development of nervous systems. The finding that the essentials of nervous systems in advanced vertebrates were evident in complex invertebrates emphasizes this point. In essence, these complex invertebrates transduce sensory inputs via modality-specific organs, which are then relayed to centralized structures by way of afferent pathways for continued signal processing and evaluation. Finally, signals are sent to the effector organs using defined output pathways. Furthermore, complex invertebrates possess an advancement in sensory system evolution that provides a mixture of unisensory and multisensory afferents (Stein & Meredith, 1993).

Complex invertebrates also demonstrate the beginnings of the distinction between afferent selectivity and central and efferent sensory integration (Stein & Meredith, 1993). Interestingly, the afferent dissociation that afforded parts of the brain to become modality-specific processors permitted other portions to become sensory integrators. By allowing integration of multisensory information in central neurons, responses can be enhanced, particularly when the various stimuli are weak. It is thought that multisensory stimuli enhance reactions by summing the energies of the several stimuli (Gielen et al., 1983; Andreassi & Greco, 1975). However, two or more stimuli of the same modality may inhibit one another or fail to summate as expected (Shipley, 1980). Multisensory integration and expression on output pathways is also an effective means to produce like behavior despite the sensory channel stimulated. Complex invertebrates have also demonstrated the ability to form cross-modal associations via pooling of various sensory cues in common neurons. Through cross-modal
association, a complex invertebrate can learn to organize anticipatory responses to sequential stimuli of different modalities (Stein & Meredith, 1993).

Insects display an ability to perform sensory integration with respect to their flight-controlling and body-controlling motoneurons. In Gypsy Moths, motoneurons controlling flight contain dendrites in the thoracic ganglion, which allows them to be contacted by efferent interneurons descending from the brain by way of the ventral nerve cord (Willis & Carde, 1990). Studies by Olberg and Willis (1990) on intracellular recordings have shown that a large portion of these descending interneurons are multisensory, and that they are capable of sensory integration to alter their output, thereby exerting control on flight motoneurons.

Vertebrates represent the pinnacle of afferent segmentation by sensory modality, while also possessing the capability for sensory integration in first and second order afferent neurons (Stein & Meredith, 1993). This is particularly true in the vestibular system, where integration of somatosensory and visual inputs in vestibular processing is a ubiquitous characteristic of vertebrates (Dichgans, et al., 1973; Caston & Bricout-Berthout, 1985; Horn et al., 1983; Precht & Strata, 1980). Unlike the vestibular system, it is thought that visual, auditory, and somatosensory stimuli initiate a stream of modality-specific information along their primary projection pathways. However, it is accepted that in particular situations these modality-specific projection pathways may also be altered by auxiliary sensory inputs (Stein & Meredith, 1993).

Stein and Meredith (1993) note that vertebrates possess a variety of sensory integration centers, for example, the superior colliculus in mammals and its non-mammalian counterpart, the optic tectum. The superior colliculus is located in the midbrain and is associated with attentive and orientation behaviors. However, it is only one of many sites in the central nervous system in which various sensory inputs converge on a group of neurons. The reticular activating system is another site of convergence for multisensory neurons; and it is responsible for general arousal (Yen & Blum, 1984). In the brain stem, multisensory neurons are found in the locus coeruleus (Grant et al., 1988), the external nucleus of the inferior colliculus (Tokunaga, et al., 1984), and the superior colliculus. Rasmussen et al. (1984) note that at the thalamic level, modality-specific primary projection pathways (e.g., lateral geniculate for visual stimuli, medial geniculate for auditory stimuli, and ventrobasal complex for somatosensory stimuli) coexist with multisensory convergence structures in the posterior and lateral thalamus.

The cortical regions of vertebrates, particularly mammals, are capable of receiving multisensory afferents, in addition to converging inputs from various unisensory thalamic nuclei (Jones & Powell, 1970). In primates, the superior temporal and intraparietal cortices contain multisensory neurons (Duhamel et al., 1991), as do the frontal and prefrontal cortex (Vaadia, 1986). These multisensory cortical areas are thought to be association areas that play a role in higher cognitive, perceptual, and attentive behaviors.

In the output portion of the central nervous system (e.g., premotor and motor limb of the central nervous system), multisensory neurons can also be found. For example, the basal ganglia are a group of interrelated structures that are essential for coordinating movement, which can be influenced by somatosensory, visual, auditory, and noxious stimuli (Hikosaka, et al., 1989). Outputs from the superior colliculus and parts of the cerebellum can also be affected in a manner similar to the basal ganglia (Aizi & Woodward, 1990).

In essence, the review of sensory system evolution (e.g., sensory system specialization, differentiation, and integration) shows that components have been preserved or elaborated in an effort to provide sensory information to the organism that may otherwise be undetectable if the various sensory components were completely segregated. This ability to detect stimuli and
integrate information in a meaningful and effective manner appears to have reached its peak in vertebrates. Interestingly, it is in vertebrates that motion sickness has been documented in the literature and not in invertebrates. The question is, is it due to the multitude of sensory integration and output integration centers in the central nervous system of vertebrates that makes vertebrates susceptible to motion sickness? Or, could it be that our ability to detect motion sickness in invertebrate species, or desire to do so, is lacking. Sensory integration conflicts, particularly with respect to the vestibular system, have been posited as the primary driver of motion sickness. Reviewed below is what is known about motion sickness in various vertebrate species and the neural structures that are thought to play a role in motion sickness.

Species in which motion sickness has been found to occur and the potential requisite underlying neural structures

Motion sickness has been found in various forms in a variety of species. It has been reported to occur in dogs (Morton, 1942; McNally, Stuart, & Morton, 1942; Babkin, & Bornstein, 1943a,b; Noble, 1945, 1948; Money & Friedberg, 1964), cats (Babkin, Dworkin, & Schachter, 1946; Johnson, et al., 1951; Crampton, & Lucot, 1985), horses (McEachern, Morton, & Lehman, 1942; Tyler & Bard, 1949; Anonymous, 1955; Chinn & Smith, 1955), cows (McEachern, Morton, & Lehman, 1942; Tyler & Bard, 1949; Chinn and Smith, 1955), sheep (McEachern, Morton, & Lehman, 1942), squirrel monkeys (Wilpizeski, et al., 1987b; Johnson, Meek, & Graybiel, 1962; Brizgee, & Igarashi, 1986; Igarashi, et al., 1983, 1986), chimpanzees (Graybiel, et al., 1960), seals (DeWit, 1953, Chinn & Smith, 1955; Anonymous, 1955), birds (Desnoes, 1926, Tyler & Bard, 1949; Ossenkopp & Tu, 1984), fish (McKenzie, 1935; Chinn & Smith, 1955), guinea pigs (Ossenkopp, & Ossenkopp, 1990), shrews (Matsuki, Ueno, Kaji, & Saito, 1988), mice (Ossenkopp, et al., 1988; Fox, et al., 1984), ferrets (Florezyk, Schurig, & Bradner, 1981), and rats (Sutton, Fox, & Daunton, 1988; Ossenkopp, & Frisken, 1982; Lambert et al., 1989). Here again, we see vertebrates only. From motion sickness studies on these various species, a portion of the critical neural structures involved in motion sickness have been identified. These are discussed next.

Vestibular System

The vestibular system is the most studied, and probably the most important neural component in the motion sickness pathway. It has been shown that individuals without functioning vestibular systems are immune to motion sickness, including visually induced motion sickness (Money, 1990). Furthermore, direct stimulation of the vestibular system can produce motion sickness (e.g., heavy water stimulation of the semicircular canals, caloric vestibular stimulation, or a Meniere’s attack). It therefore appears likely that the motion sickness inducing stimuli acts on the vestibular system either through direct stimulation of the vestibular system or through visual and proprioceptive inputs.

Research on motion sickness using animal models has clearly demonstrated the requirement of a functioning vestibular system for the genesis of motion sickness. In experiments on dogs it was shown that immunity to motion sickness can be conferred by bilateral labyrinthectomy (Babkin & Bornstein, 1943a; Babkin & Bornstein, 1943b; McNally, Stuart, & Morton, 1942; Money & Friedberg, 1964; Wang & Chinn, 1956). Furthermore, deactivating just the semicircular canals has shown to be effective in abating motion sickness in dogs (Money & Friedberg, 1964). Primate models (e.g. squirrel monkeys and chimpanzees), which are thought to be more representative of humans (Ordy & Brizgee, 1980), have also
demonstrated the effectiveness of bilateral and unilateral labyrinthectomy (Johnson, Meek, & Graybiel, 1962; Graybiel, Meek, Beischer, & Riopelle, 1960).

The question about whether the semicircular canals or otolith organs play the dominant role in spurring motion sickness has not been firmly answered, however, it appears that the semicircular canals may be the more critical component of the vestibular system. The discounting of the otolith overstimulation theory of motion sickness combined with findings that nystagmus (normally the result of stimulation of the semicircular canals) can be driven by linear accelerations, and that responses to both rotation and changing linear accelerations can be observed in a single central vestibular neuron suggests that the semicircular canals may be the more dominant component of the vestibular system with regard to motion sickness (Benson & Bodin, 1965, 1966; Correia & Guedry, 1966; Guedry, 1965a; Benson, Guedry, & Melville-Jones, 1967; Melville-Jones & Milsum, 1966). However, Brizzee and Igarashi (1986) suggest that the semicircular canals alone are not sufficient for the elicitation of motion sickness. In summary, it appears that the vestibular system is crucial for the genesis of the motion sickness and that the semicircular canals may play a more critical role than the otolith organs.

In addition to rotation and linear accelerations stimulating the vestibular system and potentially causing motion sickness, visual inputs may also influence the vestibular system. In multisensory integration, the visual modality tends to dominate, unless dramatic differences in stimulus intensity exist (Stein & Meredith, 1993). Studies by Graybiel et al. (1965) and Guedry (1965b) suggest that vestibular responses can be conditioned to respond to alternate stimuli (e.g., visual input). It is foreseeable that visual stimuli modify vestibular output and that a visual stimulus typically associated with a vestibular stimulus would yield vestibular activity in the absence of the vestibular stimulus. In visually induced motion sickness studies in which the head is restrained (Witkin, 1949), the occurrence of motion sickness suggests that the malaise resulted from conditioned activation of the vestibular centers.

**Area Postrema**

The area postrema (AP) is synonymous with the chemoceptive trigger zone (Brizzee, Ordy, & Mehler, 1980) and thought to be a critical structure in the motion sickness reflex arc (Wang & Chinn, 1954; Brizzee, Ordy, & Mehler, 1980), in addition to its role as the “integrative vomiting center” (Cummins, 1958). The AP is a circumventricular organ located on the floor of the fourth ventricle; the “integrative vomiting center” is located near the fasciculus solitarius in the dorsal part of the underlying lateral reticular formation (Brizzee, Ordy, & Mehler, 1980; Cummins, 1958). Chinn and Smith (1955) suggest that the AP’s role in the vomiting that occasionally accompanies motion sickness is a release of chemicals that spurn gastric distress when the stimulus is intense enough. This is consistent with the work of Isaacs (1957) that suggests that abnormal vestibular stimulation alone can sway the AP to induce emesis. Further support for the criticality of the AP in the motion sickness pathway comes from AP lesions in rats resulting in mitigation of drug-induced (e.g., LiCl and methylscopolamine) conditioned taste aversion (CTA; note that rats are incapable of vomiting so a CTA paradigm is used to index degree of motion sickness; Ossenkopp, 1983; Ritter, McGlone, & Kelley, 1980; Sutton, Fox, & Daunton, 1988). These findings about the role of the AP in motion sickness suggest that a functional relationship between the vestibular system and AP exists. Furthermore, Brizzee, Ordy, and Mehler (1980) suggest that while the emetic response associated with motion sickness may be mediated by the AP, the other symptoms associated with motion sickness (e.g., malaise, nausea, pallor, etc.) may be controlled by brain structures above the level of the caudal medulla.
Cerebellum: Uvula-Nodulus Complex

In the cerebellum, the nodulus and uvula are thought to play a role in motion sickness. It is suggested that vestibular afferents mediating motion sickness terminate in the nodulus, and to a lesser degree, in the uvula (Wang & Chinn, 1956). Wang and Chinn (1956) also note that the uvula receives afferent connections from the spinal cord and the corticopontine system, unlike the nodulus, which receives only vestibular afferents. Studies on dogs have shown that ablating the uvula and nodulus confer an immunity to motion sickness, while ablating the vermis between the primary fissure and the pyramis does not decrease susceptibility (Wang & Chinn, 1953; Wang & Chinn, 1956; Bard et al., 1947, 1949). Furthermore, research has shown that removal or partial ablation of solely the nodulus is effective in mitigating motion sickness, while it is necessary to remove practically the entire uvula to produce similar results (Wang & Chinn, 1956). These findings suggest that the nodulus and uvula of the cerebellum, which receive vestibular afferents, are involved in motion sickness and are part of the motion sickness pathway that traverses the labyrinths, uvula and nodulus, and area postrema before reaching the medullary vomiting center (Wang, Chinn, & Renzi, 1957). Furthermore, the work of Wang and Chinn (1956) demonstrates that it is not critical to remove all cerebellar receptors of vestibular afferents, rather, removal of 1/3 to 1/2 of the nodulus-uvula complex is sufficient for mitigating motion sickness, simultaneously not causing detectable neurological defects.

Viscera

It has been suggested that visceral afferents from the gastrointestinal tracts do not play an essential role in the vomiting associated with motion sickness (Wang, Chinn, & Renzi, 1957; Wang & Tyson, 1954). At best, Wang, Chinn, and Renzi (1957) found that denervation of the viscera in dogs resulted in a decrease in susceptibility, but did not abate emesis. They also suggest that the autonomic nervous system plays no vital role in emesis from motion sickness. Furthermore, it has been noted that vomiting in humans is not modified after vagotomy, and that to prevent emesis in experimental peritonitis, both the vagus and splanchnic nerves must be sectioned (Walton et al., 1931). Although the autonomic nervous system might not play a vital role in emesis, it clearly plays a role in balance control and motion adaptation in general. We will address this issue later.

Cerebrum

The cerebrum is not considered a necessary component in the genesis of motion sickness because motion sickness, as well as gastrointestinal and vasomotor responses to vestibular stimulation, can occur in the absence of a cerebrum (Spiegel, 1946; Spiegel, Henny, Oppenheimer, & Wycis, 1944; Spiegel, Henny, & Wycis, 1944; Spiegel, Oppenheimer, & Wycis, 1944; Speigel & Sokalchuk, 1950; Speigel & Sommer, 1950). In addition, unilateral decortication and bilateral removal of the cortex from temporal, occipital, or parietal areas in dogs has revealed that cortical structures are not critical for motion sickness to occur (Bard, 1954). As a result, it may be that mental activity (including anxiety; a point we address later) is not requisite for motion sickness. This is emphasized by an anecdote described by Doig, Wolf, and Wolff (1953) in which a relatively stable decorticate man exhibited pallor and vomiting during turbulent flight. However, despite the presence of motion sickness in the absence of the cerebrum, when the cerebrum is present its control over the brain-stem mechanisms and
cerebellar mechanisms may be highly influential in the suppression or facilitation of motion sickness (Money, 1970). For example, Kirkner (1949) has shown that voluntary mental activity during stimulus exposure can influence the intensity of motion sickness, and that suggestions that motion sickness may occur tends to increase the incidence of sickness, especially if motion is mild.

From the above sections on the vestibular system, area postrema, uvula-nodulus complex, viscera, and cerebrum it is apparent that there are a few distinct areas that are crucial for the genesis of motion sickness. If the vestibular system, area postrema, or uvula-nodulus complex are not intact, or are inhibited, then motion sickness will likely be abated or at the very least drastically mitigated. These requisite constellations are areas that display multisensory integration, and it may be that the evolutionary advancements in these areas are what make vertebrates susceptible to motion sickness. Fortunately, sensory integration (e.g., the potential for sensory conflict, which Reason [1975] posits as a major contributor to motion sickness) may be governed by a set of rules. If these rules of integration can be exploited properly, the suggestion is that motion sickness may be mitigated. Presented below is a proposed taxonomy of sensory integration accompanied by prospective rules for sensory integration.

**Emotion in Humans and Its Relationship with Motion Sickness**

As indicated above, the role of the autonomic system is not thought to be key to the emetic response, per se. However, emotion— affect— does in fact play a significant role in motion sickness. An examination below reveals those parameters necessary for the construction of a biologically based model of motion sickness. Arousal (i.e., effort) and distress are characteristics of human affect (emotion or mood) that may be important presently and have been described by a two-dimensional, physiologically-based model of affect (i.e., Franceschini, McBride, and Sheldon, 2001; Frankenhauser, 1986; Henry & Meehan, 1981; Lundberg, 1980; Sheldon, 2001). These two dimensions reflect neurochemical activity (Frankenhauser, 1986; Henry & Meehan, 1981; Lundberg, 1980): (1) catecholamines (i.e. epinephrine and norepinephrine), which are associated with the arousal dimension, and (2) corticosteroids (i.e. cortisol), which are associated with the distress dimension. Specifically, increases in the arousal dimension are associated with effort, engagement, and attentiveness, whereas increases in the distress dimension are associated with uncertainty and uncontrollable situations. Researchers investigating the phenomena of motion sickness have also studied neurochemical activity. For instance, increased corticosteroid secretion has been associated with increases in the occurrence of motion sickness symptoms (Janowsky, 1984, Reichard et al., 1998; Stalla, 1985), while increased catecholamine secretion, specifically epinephrine, has been associated with a greater tolerance to motion sickness symptoms (Zubek, 1968).

A recent investigation (Sheldon, 2001) demonstrated simultaneous measures of the sympathetic nervous system (SNS; i.e. skin conductance, heart rate [HR]); parasympathetic nervous system (PNS; i.e. HR), and somatic nervous system (i.e. muscle tension) reflected emotional responses according to this two-dimensional model. Additionally, it has been proposed that physiological evaluation of affect accounts for the SNS, PNS, and somatic nervous system (Gellhorn, 1964; Kiely, 1974; Porges, 1995). Porges (1995) proposed that the limbic system is not merely associated with emotion control (MacLean, 1993), but is responsible for motion control. Leiman-Patt, Biastrocchi, and Moia (1988) reported a relationship between increased somatic activity (cervical muscle contractures) and motion sickness symptoms. Muth
et al. (1998) have reported a relationship between increased gastric-myoelectrical activity, which is associated with nausea (a symptom of motion sickness), and decreased SNS activity.

Personality traits influence an individual's interactions with the environment (Matthews, 1999), as exhibited by subjective (Kardum, 1999; Rusting & Larsen, 1998) and physiological (Eysenck & Eysenck, 1985, Markov et al., 1995) differences in emotional experiences. Personality traits are also considered a contributing factor to motion sickness susceptibility (Reason, 1978). With respect to the distress dimension, increases are associated with feelings of anxiety. The personality trait of anxiety (Bandura, 1997; Costa & McCrae, 1995; Eysenck & Eysenck, 1985; Spielberger, 1966) is related to an increased susceptibility of perceiving the environment as threatening or uncontrollable and experiencing feelings of anxiety. Furthermore, there is evidence that higher levels of trait anxiety, as well as higher levels of state anxiety, reported prior to the experience of provocative motion are associated with motion sickness susceptibility (Mirabile & Glueck, 1993), motion sickness symptoms (Bick, 1983; Cornum, Caldwell, & Ludwick, 1993, Fox & Arnon, 1988; Gordon et al., 1994; Lindseth & Lindseth, 1992), decreased spatial orientation abilities (Betihoz & Viaud-Delmon, 1999; Smith, 1958), vestibular dysfunctions (Furman, Jacob, & Redfern, 1998; Jacob et al., 1992), and over-reactivity to visual disorientation (Milne, 1972). Additionally, the personality trait of psychoticism (Gordon et al., 1994) has been associated with tolerance to motion aftereffects.

Other factors, such as self-efficacy (Bandura, 1997; Schunk, 1989), perceived autonomy or locus of control (Noels, Clement, & Pelletier, 1999), and attitude (Bandura, 1997; Kanfer & Heggestad, 1999), influence an individual's perception of the environment, thereby contributing to the individual's affect. These factors have also been investigated with respect to motion sickness. For instance, increased locus of control has been associated with reduced motion sickness symptoms (Collins & Lutz, 1977; Keinan et al., 1981). Eden and Zuk (1995) reduced seasickness effects in Israeli Defense Forces Navy cadets through efforts aimed at increasing the cadets' self-efficacy regarding their tolerance to experiencing seasickness. Also, Grunfield et al. demonstrated a relationship between an individual's positive attitude regarding his susceptibility to experiencing motion sickness effects was associated with reduced motion sickness symptoms. Consequently, it is not surprising that cognitive-behavioral therapies (Dobie & May, 1994; Dobie et al., 1989; Koselka, 2000) and relaxation techniques (Dobie et al., 1994; Jackson, 1994) have been successful at reducing the occurrence of motion sickness aftereffects.

An individual's posture has been associated with emotional responding (Eckman, 1993; Gellhorn, 1968). There has also been research regarding the effects that rest posture orientation (Harm et al., 1998) and postural instability (Owen, Leadbetter & Yardley, 1998; Warwick-Evans et al., 1998) have on motion aftereffects.

Researchers have investigated the influence of visual motion on emotional responding. However, the reports of these efforts are incongruous. For instance, Simons et al. (1999) suggested visual motion increased arousal, while Detenber and Reeves (1996) reported contradictory results. A plausible explanation for this conflict is that the Simons et al. (1999) study used physiological measures (skin conductance) in conjunction with subjective responses to obtain data on arousal and valance, whereas the Detenber and Reeves (1996) investigation only used subjective measures. As subjective measures are considered poor indicators of emotional experiences (Cacioppo & Petty, 1986), and there is evidence of physiological responding that contradicts subjective reports (Schwartz, 1986; Sheldon, 2001), it is reasonable that the Detenber and Reeves (1996) study would have obtained results similar to the Simons et al. (1999) study if physiological measures had been used.
Prospective Taxonomy and Rules of Sensory Integration

The field of intelligent sensor fusion in robotics has provided fertile ground for the commingling of biological and cognitive aspects of sensory integration. There are increasing challenges for machine makers as the underlying technology becomes more capable of simulating human behavior. For example, it is not obvious that a robot should be “down-loaded” with code sufficient to solve any reasonable problem, or whether there is more efficiency or effectiveness in designing robust learning capability instead. A look at sensor fusion technology is helpful in our attempt to understand motion sickness (though, certainly, robots will not show symptoms), because the engineering world is relying increasingly on biomimetic approaches to design. Following is a synopsis of some representative work.

Murphy (1996) discusses the biological and cognitive components of sensory integration in regards to sensor fusion in robotics and in doing so presents various viewpoints regarding the process of sensory integration. For instance, Marks’ (1978) theory of sensory correspondence is presented, as well as Bower’s (1974) taxonomy of sensory integration. Marks’ (1978) theory of sensory correspondence is comprised of five Doctrines. The first doctrine is the Doctrine of Equivalent Information, which suggests that the same percept can be deduced from individual receptors, despite their receiving distinct and nonintersecting stimuli. The second is the Doctrine of Analogous Attributes and Qualities that states that all stimuli have a common stimulus property (e.g., intensity, duration, size, form, and number). The third, Doctrine of Common Psychophysical Properties, notes that stimuli are processed by like, or multisensory, receptors. The Doctrine of Neural Correspondences is the fourth doctrine and posits the existence of a multisensory integrator, which requires stimuli to be represented in a common format or knowledge representation. The final doctrine is comprised of four previously mentioned doctrines and is referred to as the Unity of the Senses. It suggests that because the senses are so similar, they should be thought of as modalities of a general sense.

Bower’s taxonomy of sensory integration is comprised of four levels. The first level is complete sensory unity, in which all receptors receiving the stimulus are combined without a mechanism for detecting discordances. In level one, the receptors for a particular stimulus are in synchrony such that discrepancies do not arise. At this level, sensory integration is done blindly without a means for detecting discordances between the modalities. The second level of the taxonomy is unity with awareness of discordance and the possibility of adaptation. At this level, discordances between afferent signals can be detected and dealt with via adaptation of the most deviant afferent signal. This level of the taxonomy is most akin to an individual’s ability to capitalize on inherent neural plasticity and utilize adaptation or habituation to mitigate motion sickness. The third level of the taxonomy is awareness of discordance with a tendency to suppress it. At this level, discrepancies between receptors are detected, but instead of adapting, the most discordant receptor is suppressed. This may be the case when an individual becomes dual adapted to a provocative environment and is capable of suppressing a motion sickness inducing conflict long enough to complete a rapid acclimation process associated with dual adaptation. The fourth and final level of the taxonomy is no unity at all. At this level, the different receptors process aspects of the stimulus, but the aspects have no correspondence among each other. Bower notes that this level rarely occurs in humans.

Another system that describes hierarchies of control systems is one provided by Powers (1973). This system suggests that there are characteristically eight levels of sensor integration in
cybernetic control systems. In ascending order they include control of: sensitivity, vector, configuration, transition, sequence, relationship, strategy, and principles.

Stein and Meredith (1993) present various rules of sensory integration. These rules are briefly presented and discussed below.

- Spatially coincident multisensory stimuli tend to produce response enhancement, whereas spatially disparate stimuli produce either depression or no interaction.

This rule suggests that enhancement occurs only when sensory stimuli are presented within their respective receptive fields. For example, a visual-auditory stimulus pair must have the visual stimulus within the visual receptive field and the auditory stimulus with the auditory receptive field. Due to the sensory overlap in a multisensory neuron, co-located stimuli enhance each other’s effects. If one of the sensory stimuli is presented outside the range of its receptive field, it will fail to enhance the effect of a second stimulus within its receptive field and may depress any response the primary excitatory stimulus.

- Maximal multisensory interactions are not dependent on matching the onset of two different sensory stimuli, or their latencies, but on how the activity patterns resulting from the two inputs overlap.

Some of the temporal limitations on multisensory integration are lucid, such as stimuli presented in close temporal proximity interact, while stimuli with large temporal separations are processed as separate events. Stein and Meredith (1993) note that the existence of a relatively large temporal window is logical once the travel and processing times for various stimuli are considered. They suggest a temporal window up to 1500 msec must exist to be able to process the longest delays found in visual-auditory stimulus pairs. They also note that from an evolutionary standpoint it makes perfect sense because a long temporal window provides leeway in detecting and responding to minimal and important stimuli at various distances from an organism. This also makes evolutionary sense because the depression of discordant stimuli when they are in temporal proximity provides a means for focusing attention on the strongest, and presumably the most important, stimulus when distracters are present.

- Receptive field properties are neither created nor eliminated by combining inputs from different sensory systems.

The essence of this rule is that receptive field properties act as neuronal filters, deciding which stimuli activate a neuron and how intense that activation will be.

- The multiplicative interactions that characterize superior colliculus responses to two stimuli from different modalities are not apparent when the stimuli are from the same modality.

- Maximal enhancement occurs with minimally effective stimuli.

A Preliminary Integrative Model of Motion Sickness

Depicted below is a tentative model of motion sickness that is based on the three existing models (outlined above), and the biological information considered above.
The essence of the model is obvious from its flow. First, consider the fundamental nature of the diagram; that is, that toxic filtration is its foundation, much as the poison hypothesis posits. The organism acquires food, in a context (location, scents, etc.) that is associated with the acquisition, including the predatory fixed action pattern, itself. The digestion process begins when saliva is secreted, and here again cues from the context (textures, tastes, etc.) are present and become associated with the acquisition.

Two signal detection analyses are continuously working in the animal. The first is associated with detecting the presence of toxins based on assessment of contextual cues (taste, scent, etc.). The other is concerned with detection given that there is in fact toxicity in the digestive system, or more to the point, it is concerned with detection given that there is no toxin in the system. Importantly, the presence of toxins or any other material is not a binary proposition. Rather, detection is an issue of fuzzy logic, as all biological detection is. Thus, the animal must assess (unconsciously, of course) the likelihood that there is poison in the gastrointestinal tract. Unnecessary emesis—false alarm—is in fact expensive, not only because of lost sunk costs (of hunting, etc.), but also because of the opportunity costs, and the raw caloric cost of the emesis sequence, itself. Dehydration amplifies the expense to the organism.

Thus, key to the model are the parameters associated with the signal detection components. Clearly organisms inherit some initial and boundary conditions on these parameters. Just as clearly, there is plasticity or learning (conditioning) in toxin detection and avoidance. We describe next, briefly, what is essentially this filtration process. (For a complete treatment of signal detection theory, see Swets, 1996).

The signal detection parameters as depicted in figure 1 are d' and Beta. The former represents signal separation strength from background noise (or from an alternative signal); the latter is an indication of the organism’s bias, known also as decision criterion. If toxin is clearly recognizable, d’ is said to be large, and the probability of error on the part of the animal is minimal. On the other hand, if toxicity is disguised (as is usually the case in nature), d’ is low, ipso facto. Momentary d’ is governed by the strength of the poison cues against their surround
(external or internal environment). Arguably, a compromised sensor system contributes to effective signal separation, as well.

![Graph](image)

Figure 2. Receiver Operating Characteristic depicting the improvement in detection as Signal Separation (d') increases.

Bias, on the other hand, is more situationally dependent. For example, as an animal continues to forage or hunt, and as the prospects for acquiring a next meal fail to improve, the probability of accepting a piece of food, say slightly tinged, improves. That is, the animal must manage its bias so as carefully to maintain calorie investments while avoiding toxis: that is, minimize the probability of committing type I or type II errors. The former (detecting toxin falsely) means loss or opportunity loss (and perhaps all of the other costs indicated, above); the latter (failure to detect the poison) might mean death. Thus, each organism behaves in a way that describes a receiver operating characteristic (ROC), as is shown in figure 2. The variables of each unique ROC are governed by the quality of the sensor system they inherit (and which matures), and by the quality of feedback they have produced (long term store) to date.

The model described suggests that the mechanism of motion sickness is sensory mismatch, and that those organisms which are sensitive to mismatch, for whatever reason it is imposed situationally, react naturally by engaging some level of the emetic complex. There are comparative differences in this regard, as outlined, above, and there are individual differences within species, and as the model suggests, within individuals. Fundamentally, provocation causes some probability that the emetic complex be engaged, which (as feedback loop A indicates) initiates an evaluation against an experience data base (long term store), which modulates the beta in evaluation B. Necessary to any confirmation of this simple model, of course, is acquisition and application of known or theoretical values for the parameters identified. Valid models of d' and beta (and thus an enriched theory) could be derived from empirically existing or experimentally produced ROCs.

A straightforward application of the model is with regard to flight simulator sickness (a surprisingly prevalent phenomenon: see Cornum, Caldwell, & Ludwick, 1993; Fox & Arnon, 1988; Jackson, 1994; Jones, 1984; Lindseth & Lindseth, 1992). Perhaps as a pilot acquires very precise mastery of “his aircraft” through thousands of hours of practice, the number of perceptual
just noticeable differences (jnd's) he has acquired is very significantly greater than those of a novice. That is, even at an unconscious level, his perceptuomotor control system detects very small differences between the dynamics of "his aircraft" (let's call it the veridical) and those of a high-fidelity simulator. Such that during a simulated practice flight, he "mistakenly loads" the "veridical data base" associated with his F-14D, because the simulator cockpit signals to do so. However, the simulator is just that: its dynamics are not quite the same as those of the veridical. Provocation is thus inspired at the level of jnd mastery that our hypothetical, highly skilled pilot has achieved. Expectation does not meet reality. This would explain also why flying say a small single engine recreational aircraft on the weekend is less likely to cause motion sickness, because the "wrong data base" does not get invoked in the pilot's perceptuomotor control system.

The model outlined presently would predict that simulator sickness susceptibility increases with experience rather than the reverse. Moreover, the model might help describe or understand delayed simulator sickness phenomena. It could well be that two dynamics data bases (the veridical and the simulated) are actually "compared" during the trace consolidation (which may include unconscious re-enactment) phase of skill acquisition (that is, after or even well after the simulation flight). Because they are dissimilar, provocation results.
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