Brain Research Bulletin 81 (2010) 7–11

Contents lists available at ScienceDirect

Brain Research Bulletin

journal homepage: www.elsevier.com/locate/brainresbull

Review

Motion sickness: A negative reinforcement model

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ARTICLE INFO

Article history:
Received 22 June 2009
Received in revised form
17 September 2009
Accepted 29 September 2009
Available online 4 October 2009

Keywords:
Motion sickness
Negative reinforcement
Evolution
Toxin
Defense

ABSTRACT

Theories pertaining to the "why" of motion sickness are in short supply relative to those detailing the "how." Considering the profoundly disturbing and dysfunctional symptoms of motion sickness, it is difficult to conceive of why this condition is so strongly biologically based in humans and most other mammalian and primate species. It is posited that motion sickness evolved as a potent negative reinforcement system designed to terminate motion involving sensory conflict or postural instability. During our evolution and that of many other species, motion of this type would have impaired evolutionary fitness via injury and/or signaling weakness and vulnerability to predators. The symptoms of motion sickness strongly motivate the individual to terminate the offending motion by early avoidance, cessation of movement, or removal of oneself from the source. The motion sickness negative reinforcement mechanism functions much like pain to strongly motivate evolutionary fitness preserving behavior. Alternative why theories focusing on the elimination of neurotoxins and the discouragement of motion programs yielding vestibular conflict suffer from several problems, foremost that neither can account for the rarity of motion sickness in infants and toddlers. The negative reinforcement model proposed here readily accounts for the absence of motion sickness in infants and toddlers, in that providing strong motivation to terminate aberrant motion does not make sense until a child is old enough to act on this motivation.

Contents

1. Introduction

The "why" of motion sickness has received very little attention compared to the "how" of this very disturbing condition. There are numerous articles pertaining to variations of sensory conflict theory and postural instability hypothesis, currently the two most popular perspectives regarding the "how" of motion sickness. In contrast there are only two theories, with little attention paid to them, focusing on why such a seemingly non-adaptive physiological event occurs. Based on its presence in so many species and virtually every human possessing an intact vestibular system, motion sickness has a very strong biological basis [11,8]. This reality suggests that there must be an evolutionary fitness enhancing function to the condition.

The two "why" theories examining the evolutionary fitness advantage of motion sickness will be referred to as the toxin and movement program theories. Both theories have significant shortcomings and neither is able to account for why motion sickness is rare or absent in infants and toddlers. An alternative highly parsimonious explanation is posited here that accounts for the lack of motion sickness in the very early years of life. Essentially, motion sickness evolved as a form of negative reinforcement providing potent motivation for the cessation of any motion producing sensory conflict or postural instability. Aberrant motion of this form

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doi:10.1016/j.brainresbull.2009.09.017

would have greatly increased the risk of injury or signaled weakness and vulnerability to predators, thereby reducing evolutionary fitness.

2. Alternative "why" theories

Motion sickness has been described as an evolutionary anomaly, given that such a powerful mechanism seems to have evolved in so many species when there does not appear to be any survival value in the occurrence [4,2]. Yates et al. [12] suggest that there might not be an evolutionary aspect to motion sickness. According to these researchers motion sickness results from aberrant activation of neural pathways that serve to maintain a stable internal environment, with conflicting signals regarding body position in space producing atypical activation of brainstem neurons normally serving to maintain homeostasis, resulting in emesis. Instead of offering a non-evolutionary why theory of motion sickness, their explanation provides a possible how mechanism based on sensory conflict. There is no elaboration of why aberrant activation occurs in the first place—the domain of why theories. We are left guessing why this would occur, but assuming it is non-evolutionary the only reasonable explanation is a disease process either metabolic or infectious to explain the aberrant activation.

Beyond the reductionist nature of such an explanation when applied to phenomena of universal prevalence, motion sickness does not fit a disease model. Short of rare time limited pandemic infections, disease occurs in a subset of the larger population and arises from an interaction of genetic (diathesis) and environmental influences (stress). For example, with Type II diabetes there is a genetic vulnerability to disordered glucose metabolism and the stress of insulin resistance related to aging and excess body weight. Not everyone is able to develop diabetes and other diseases. With motion sickness present over recorded history in everyone with an intact vestibular system, and multiple and diverse animal types, a disease model does not fit at all. The inter-species and intra-species commonality supports an evolutionary basis.

Proposing an evolutionary advantage for inherently positive behavior is much easier than for what clearly appears to be maladaptive behavior, likely accounting for so few theories regarding the "why" of motion sickness. After all, how could it possibly be adaptive to feel violently ill and become dysfunctional during challenging circumstances? Such an occurrence would seem to represent an instance where biologically based behavior is maladaptive. Hence, the toxin and movement program theories have an uphill battle from the start. Treisman [11] proposed that movement control mechanisms provide an early warning system for the detection of neurotoxins. Working from a sensory conflict perspective regarding the "how" of motion sickness, Treisman [11] indicates that neurotoxins will produce mismatch between sensory (vestibular) and eye coordination systems given the continual action and high degree of susceptibility to disruption of these processes. The toxin mechanism serves as a backup to taste and emesis evoked by effects on the gastrointestinal lining or stimulation of chemoreceptors after absorption.

While intriguing there are several major problems with the toxin theory beyond it representing a non-parsimonious and highly complex mechanism. As Treisman [11] indicates, evolution has already provided mechanisms for dealing with toxins in the form of taste and the response of the gastrointestinal systems before and after absorption. In addition, the liver has evolved as an organ largely responsible for ridding the body of toxins. To evoke an additional mechanism, and then only for toxins capable of crossing the blood–brain barrier, might be considered somewhat redundant. Furthermore, not all motion sickness leads to vomiting, and significantly less so than physical disgust reactions. If toxin removal constitutes the key purpose for motion sickness vomiting would always occur. Of course, for neurotoxins to induce the motion sickness response they must already be present in the brain where they cannot be removed by vomiting.

To produce the desired effect the toxin mechanism relies on direct sensory conflict between the vestibular and eye coordination systems. Some versions of sensory conflict theory emphasize direct conflict between the senses as implied by the name, but it is doubtful that different types of sensory input can actually be compared directly [10]. A more valid version of sensory conflict theory takes the form of a "neural mismatch" hypothesis whereby perceived motion is at variance with expected motion [7,6]. Whereas the brain might not be able to directly compare different types of sensory input, it does seem to be capable of forming expectations of motion based on experience. For example, no one perceives walking as unusual, whereas most people perceive flipping upside-down to be odd. Another major challenge to the toxin theory is that infants and toddlers with rapidly developing brains most sensitive to toxins do not experience motion sickness [8,9]. It simply does not follow that fully developed brains less vulnerable to most neurotoxins would have a pronounced toxin ejection mechanism, and the brains of those highly sensitive to most neurotoxins would lack the mechanism.

Regarding support for the toxin theory there has only been one instance of evidence since the theory was proposed [12,5]. Money and Cheung [5] observed that labrynthectomy in 7 dogs increased the latency and threshold for vomiting in response to some emetic drugs. Their results fail to support the theory for several reasons. First, the substances tested are not toxins per se but emetic agents. Second, while the emetic response was reduced for some of these drugs (lobeline, levodopa, nicotine) it was not for others (apomorphine, pilocarbine). If the toxin theory is valid there should not be a selective effect for only some "toxins" that cross the blood–brain barrier. The mechanism is designed as a final backup, and thus has to act on all toxins that enter the brain. Third, as Yates et al. [12] indicate the removal of vestibular input due to labrynthectomy results in disfacilitation of central emetic circuitry, providing a more plausible mechanism for the results of Money and Cheung [5]. Fourth, dogs are distinct from many other species in so far as drug effects on motion sickness are concerned, and consequently the dog model of motion sickness has largely been abandoned [12]. Hence, generalization of the Money and Cheung results for labrynthectomized dogs to any other species is dubious at the best. Fifth, there is the possibility that the results cannot be generalized at all given some inexplicable findings of the study. The emetic response to apomorphine, lobeline, levodopa, and nicotine act on the area postrema, while that of pilocarbine depends on forebrain structures [1]. Money and Cheung found that the emetic response of apomorphine and pilocarbine were unaffected but that of lobeline, levodopa, and nicotine was reduced, a finding that seemingly lacks a neurobiological basis given that the results for apomorphine and pilocarbine should logically diverge. Therefore, for a variety of solid reasons the very limited "support" for the toxin theory provided by the Money and Cheung study cannot be viewed as valid.

The second "why" theory of motion sickness suggests that the innate displeasure generated by movement programs yielding vestibular conflict discourage the development of these programs [3]. The displeasure resulting from vestibular conflict trains and conditions the spatial orientation system to develop perceptual-motor programs that are efficient in the operating environment of the individual. Once again we see the emphasis on conflict between sensory inputs that might not be directly comparable. However, three other considerations are more damaging to this theory. First, positive reinforcement provides a powerful training mechanism for spatial orientation programs making another system largely redundant. As an example, a young child obtains