Motion sickness adaptation: a neural mismatch model[1]

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Introduction

In almost all susceptible individuals, continued exposure to a provocative motion stimulus leads to the diminution and eventual disappearance of established motion sickness reactions (*see* Reason & Brand 1975). This reduction in symptomatology takes place without any change in the nauseogenic stimulus; indeed, it is the absence of such variation that promotes its occurrence. These observations are clearly of considerable importance for elucidating the factors involved in the aetiology of motion sickness, as they demonstrate the existence of processes within the individual that are capable of counteracting the disturbing properties of the imposed motion without recourse to any external agency. It follows, therefore, that a better appreciation of how this *vis medicatrix naturae* operates must bring us closer to understanding how these curious and inappropriate reactions become established in the first place, since adaptation and provocation appear to be two sides of the same coin.

The theoretical arguments presented here are directed toward answering two basic questions concerning the aetiology of motion sickness. First, what is the essential nature of the provocative stimulus? How do we explain the occurrence of motion sickness over such a wide range of apparently diverse circumstances? What, for example, do the conditions that elicit space sickness have in common with those that induce seasickness, airsickness, car sickness, camel sickness, swing sickness, simulator sickness, and so on? This question is dealt with briefly as a necessary prelude to the main concern of this paper, namely: what are the mechanisms underlying the acquisition of protective adaptation, and of its sequel *mal de débarquement?* The principal research findings relating to the effects and after-effects of protective adaptation are summarized, and a neural mismatch model is described which attempts to account for these data.

The provocative stimulus

Sensory rearrangement theory

The sensory rearrangement theory seeks to specify those sensory characteristics common to all the many different circumstances that induce motion sickness, and thus define the essential nature of the provocative stimulus. Although the notions embodied in this theory have only gained widespread acceptance within the last decade, they have been expressed in one form or another for at least a century. One of the earliest exponents was Irwin (1881) who wrote: 'In the visual vertigo of seaksickness there appears to be a discord between the immediate or true visual impressions and a certain visual habit or visual sense of the fitness and order of things,

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which passes into consciousness as a distressing feeling of uncertainty, dizziness and nausea'. In this passage, Irwin makes it clear that he appreciated the true significance of sensory conflict, namely that a discord between normally congruous sources of spatial information (i.e. in active, self-propelled locomotion) inevitably results in a conflict between what is currently being signalled by the position and motion senses and what is expected on the basis of prior experience. Claremont (1931) advanced a similar explanation, but in a different context. Having felt queasy in the cinema as a result of watching a film shot from the back of a swaying train, he argued that motion sickness '. . . is due to the unaccustomed conflict between sensations normally combined in other ways'. Later exponents of the conflict notion include Hill (1936), Morales (1946), Kirkner (1949), Lansberg (1960), Steele (1961), Gillingham (1966), Guedry (1964, 1968) and Reason (1969, 1970).

The sensory rearrangement theory (*see* Reason & Brand 1975) can be stated in the form of two premises. The first is that all situations which provoke motion sickness are characterized by a condition of sensory rearrangement in which the motion signals transmitted by the eyes, the vestibular system and the nonvestibular proprioceptors are at variance one with another, and hence with what is expected on the basis of previous transactions with the spatial environment. The nauseogenic conflict is between the present sensory information and that retained from the immediate past, or what Held (1961) has called 'exposure-history'. That the conflict existing within the present pattern of sensory inputs is by itself insufficient to cause motion sickness is evident from the earlier observation that continued interaction with the nauseogenic stimulus results in the eventual disappearance of symptoms even though the incongruity between the various sources of spatial information remains. It is this crucial temporal comparison between present and past patterns of spatial stimulation that provides the necessary explanatory link between the sensory rearrangement notion and protective adaptation.

The second premise of this theory is that irrespective of what other spatial senses are part to these conflicts, the vestibular system must be implicated, either directly or indirectly (as in visually-induced sickness), for motion sickness reactions to ensue. Not only does this accommodate the well-established fact that susceptibility depends upon an intact vestibular system (*see* Money 1970), but it also tells us something about the nature of the effective motion stimulus; namely, that it must involve a changing rather than a constant velocity component since the vestibular receptors are only responsive to angular and linear accelerations. This is important because it allows us to predict with some confidence which of the many different kinds of sensory rearrangement are likely to produce sickness, and which will not.

Types of sensory rearrangement

Sensory rearrangements can take many forms depending on the prevailing circumstances and which senses are involved; but for convenience they can be subsumed under two headings: (1) predominantly visual-vestibular rearrangements, where the conflict is between the sense modalities; and (2) canal-otolith rearrangements in which there is an intramodality conflict between the vestibular receptor systems. Under biologically natural conditions of self-propelled locomotion in an Earth gravity environment, there is a synergistic relationship between the visual and vestibular systems and between the semicircular canals and the otoliths. In other words, they all tell the brain the same story regarding the body's orientation in space. But this harmonious relationship can be disturbed in at least three ways. If A and B represent portions of these normally correlated receptor systems, then the three conflicts can be described as follows: *type 1*, when A and B simultaneously signal contradictory or uncorrelated information; *type 2*, when A signals in the absence of an expected corroborating signal from B; and *type 3*, when B signals in the absence of an expected signal from A. On the basis of this classification we can specify six kinds of sensory rearrangement in which motion sickness might reasonably be expected to occur. Laboratory and everyday examples of each situation are summarized in Table 1.

Three points need to be made about this classification. First, visual-vestibular and canal-

Table 1. Examples of the six categories of sensory rearrangement

Visual(A)–vestibular(B) rearrangements		
Type 1: (A and B together)	Looking from the side or rear window of a moving vehicle	
	Watching waves over the side of a ship	
	Making head movements while wearing an optical device that distorts the visual field	Stratton (1897)
Type 2: (A in the absence of B)	Watching a cinerama-type motion picture shot from a moving vehicle subjected to linear and/or angular accelerations	Benfari (1964), Parker (1971)
	Riding in a 'haunted swing' fairground device	Wood (1895)
	Operating a fixed-base vehicle simulator with a dynamic visual display	Miller & Goodson (1960), Reason & Diaz (1971)
Type 3: (B in the absence of A)	Attempting to read a book or map in a moving vehicle	
	Riding in an enclosed vehicle without external visual reference	
Canal(A)–otolith(B) rearrangements		
Type 1: (A and B together)	Exposure to cross-coupled (or Coriolis) accelerations	Guedry & Montague (1961), Reason & Brand (1975)
	Making rapid turning movements of the head in weightless flight	Benson (1977)
Type 2: (A in the absence of B)	Caloric stimulation	McNally & Stuart (1967)
	Positional alcoholic nystagmus and ingestion of deuterium oxide	Money & Miles (1974)
	Pressure vertigo due to ambient pressure changes	A J Benson (1975, personal communication)
Type 3: (B in the absence of A)	Low frequency (less than 0.5 Hz) vertical oscillation	Benson (1973), O'Hanlon & McCauley (1974)
	Rotation about an earth-horizontal or off-vertical axis at constant angular velocity	Correia & Guedry (1967), Miller & Graybiel (1970)
	Counter-rotation	Graybiel & Johnson (1963)

otolith conflicts can and frequently do coexist in many atypical force environments. Thus, canal-otolith conflicts are often exacerbated by the presence of discordant visual information. Second, it must be stressed that no fundamental distinction is drawn between actual bodily motion and the apparent bodily motion induced by viewing a scene that, under more familiar circumstances, would be accompanied by vestibular stimulation. Third, all the situations cited in Table 1 are known to produce motion sickness, or at least something so closely akin to it as to make the distinction meaningless.

Protective adaptation: principal findings

Most of the studies considered below have used cross-coupled stimulation where subjects are required to make controlled head movements aboard a rotating platform about some axis other than the axis of rotation. For this reason it is convenient to preface this section with a brief description of the typical experimental situation.

Studies designed to investigate adaptation to this potent nauseogenic stimulus have required subjects to make a series of controlled head and body movements. At the completion of each movement, they are asked to indicate whether or not they experienced any unusual sensations due to the cross-coupled stimulus (i.e. somatogyral or oculogyral effects). For any given platform velocity, the rate of adaptation is indicated by the number of head movements eliciting an illusory perception. The usual practice is to increase the angular velocity in a series of 1 r/min steps up to some terminal velocity. At each step, the subjects are required to attain a predetermined adaptation criterion (a given number of head movements eliciting a 'no-illusion' response) before proceeding to a higher velocity. This stepwise exposure allows the investigator to measure the amount of stimulation needed to neutralize the illusory sensation,

while at the same time minimizing the risk of motion sickness occurring *en route*. The chief advantages of the rotating environment are that it permits a high degree of stimulus control, and also yields readily quantifiable data.

The temporal pattern of adaptation

Observations derived from a number of different provocative circumstances (Groen 1960, Guedry 1965, Reason 1969) indicate that there are three distinct phases in the adaptation process: (1) an *initial exposure phase* in which illusions of motion, various psychomotor disturbances and motion sickness reactions make their appearance with latencies ranging from milliseconds to hours, depending upon individual susceptibility, the nature of the rearrangement and its severity; (2) a *continued exposure phase* in which the illusory perceptions and the various adverse reactions (with the exception of drowsiness) diminish and eventually disappear through continued interaction with the rearranged environment; and (3) an *after-effect phase* following return to the previously typical force environment, in which reactions characteristic of the initial exposure phase are reinstated. Where these reactions have a directional sign, as in the illusions of apparent motion, this is reversed with respect to the initial exposure phase. With further exposure to the typical environment, these after-effects rapidly subside in most (but not all) individuals until, by all outward signs at least, they are restored to their pre-exposure state of well-being. Postrotatory sickness (*mal de débarquement*) may be as severe in the early part of the after-effect phase as the symptoms experienced during the initial rotatory exposure. Another point to stress with regard to cross-coupled stimulation is that motion sickness is only contingent upon head movements in both the initial exposure and after-effect phases.

Long-term retention of protective adaptation

Two studies have indicated that adaptation to cross-coupled stimulation is retained in some degree for long periods of time (Reason & Diaz 1975). They also showed that whatever loss of adaptation there was occurred fairly soon after the end of the adapting session, and that thereafter the level of protective adaptation remained relatively constant. There was an exponential pattern of adaptation decay, and the findings of these studies suggested that the time-constant of this decay curve increased as a function of the number of adapting sessions that precede it. In other words, the greater the number of previous adapting sessions, the slower was the rate of adaptation decay following the last of them, and the higher was the stable level of retained adaptation.

The extent of the sensory rearrangement

In any nauseogenic environment, the extent of the rearrangement depends upon two factors: the number of discrepant sensory channels and the magnitude of the discrepancy within any one sensory channel. There is now evidence from both the rotating and weightless situations to suggest that the rate of adaptation is inversely related to the extent of the rearrangement (Reason & Diaz 1970, Graybiel *et al.* 1975).

The specificity and transfer of adaptation

The available experimental evidence indicates that when the adapting exposure is of short to moderate duration, the adaptation so acquired is highly specific to the prevailing stimulus (Guedry *et al.* 1962, 1964). But when the adapting session is extended well beyond the point of adaptation (i.e. when illusory sensations are no longer detectable), some degree of generalization to other forms of the same type of sensory rearrangement does occur, although its extent has not yet been fully determined (Reason & Brand 1975).

Active versus passive movements

Several studies have shown that active movements are superior to passive movement in acquiring adaptation to visual distortion involving the displacement, curvature or tilt of the seen environment (Held 1965). Comparatively little is known about whether or not the same is

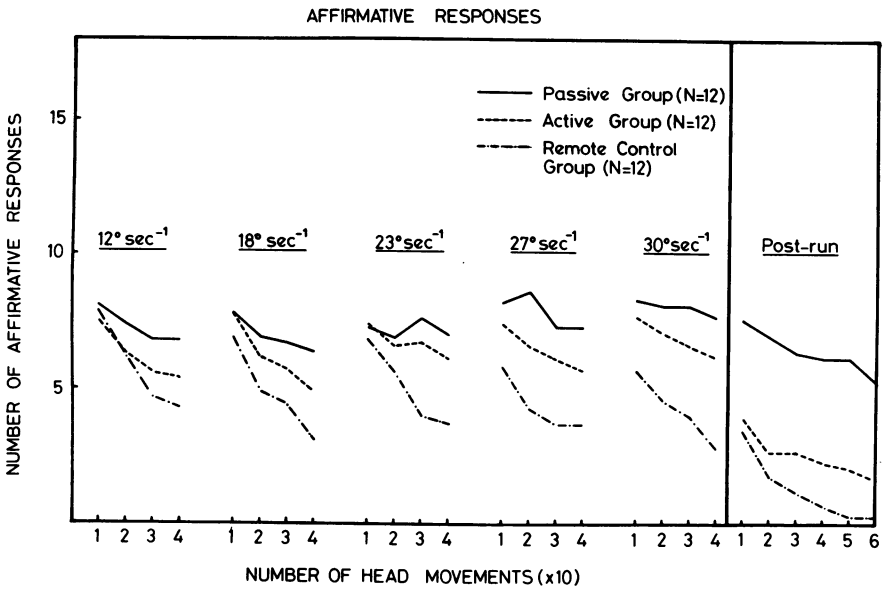

Figure 1. Comparing rates of adaptation under three conditions of voluntary movement control. The graph shows the mean number of affirmative responses (occasions when the subjects detected the presence of the oculogyral illusion) over each sequential ten head movements at each of 5 rotational velocities, and during the post-run phase. The slopes of these plots indicate the rates at which the oculogyral illusions were neutralized, i.e., the rate at which adaptation was aquired

true for inertial rearrangements such as cross-coupled accelerations, since most of the laboratory work in this area has involved only active, self-produced movements on the part of the subjects. The expectation, however, based on the optical distortion work, is that adaptation can occur with passively-induced head movements, but it is likely to be acquired far more effectively as the result of voluntary activity. The theoretical basis for this prediction will be considered at a later point.

An experiment designed to test this hypothesis was carried out very recently (Reason & Benson 1978). The study compared rates of adaptation to the same graded levels of cross-coupled stimulation under three conditions of voluntary movement control: (1) a *passive condition* in which the 45° lateral tilts of the subject's chair upon a rotating platform were initiated and controlled entirely by the experimenter; (2) an *active condition* in which the subjects executed the same tilting motions of the chair directly through their own effector activity; and (3) a *remote-control condition* in which the subjects governed the motion of the chair indirectly through microswitches located on its arms. At the end of each movement, the subjects were asked to report upon the apparent movement (or stability) of a dimly-illuminated visual target, i.e. the Coriolis oculogyral illusion. The results are summarized in Figure 1. These results clearly confirmed the expectation that the passive condition would be the least effective mode for developing protective adaptation. The rate at which the Coriolis oculogyral illusion was neutralized at each prerotational step, and in the postrotational phase, was significantly slower for the passive group than for the other two experimental groups ($P<0.01$). It is not clear, however, why the remote-control group was consistently faster in acquiring adaptation than the active group. This rather surprising finding has interesting theoretical and practical implications, and needs to be investigated further.

Neural mismatch model

The suggested mechanism of the adaptation process set out below makes little claim to originality, except perhaps in its specific application to atypical force environments. The

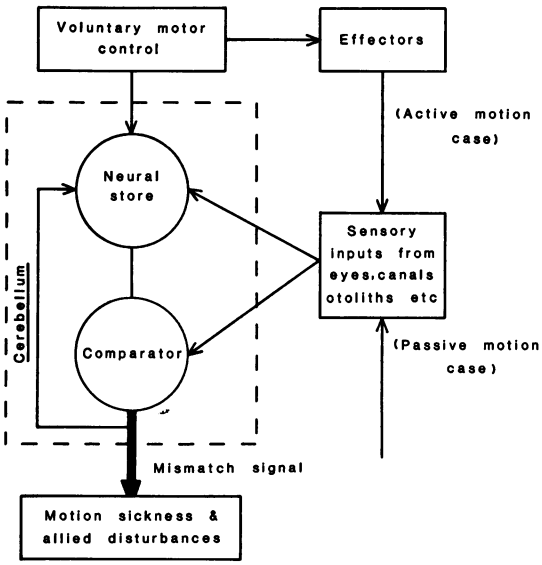

Figure 2. The basic structural components of the neural mismatch model

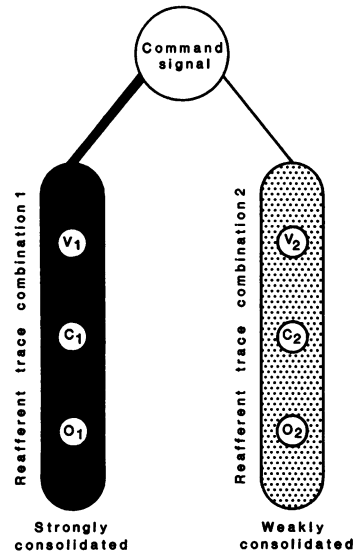

Figure 3. A schematic representation of a hypothetical spatial engram. V, visual input; C, canal input; 0, otolith etc. inputs

origins of the neural mismatch notion can be traced directly to von Holst's (1954) 'reafference principle', and to the later modifications of this concept proposed by Groen (1960) and Held (1961).

Structural components of the theory

The basic structural components of the model and their interrelationships are summarized in Figure 2. The key component for adaptation is the neural store. Following Held (1961), it is proposed that this retains traces of previous combinations of command signals (efference) and the integrated patterns of inputs from the orientation senses generated by them (reafference). A schematic representation of one such hypothetical trace combination, or spatial engram, is shown in Figure 3. Let us suppose that this particular command signal was 'Tilt the head 45° to the left shoulder'. The presence of two associated reafferent traces indicates that this efference has been executed in two different force environments. The left-hand reafferent trace is strongly consolidated and attached to the efferent trace by a well-established associative bond. This represents a record of the correlated inputs from the eyes, semicircular canals and otoliths when the head movement was carried out in the normal terrestrial environment. The right-hand trace, containing different inputs from the same orientation senses, is much less defined and more weakly associated with the command signal. For our present purposes, it is presumed that this trace was laid down as the result of exposure to cross-coupled angular stimulation (that is, to executing lateral head tilts in a rotating environment); but it could equally well have resulted from an encounter with some other form of sensory rearrangement, such as weightlessness. The purpose of Figure 3, therefore, is to illustrate how the same command signal can become linked within the store to two quite disparate reafferent traces. The significance of this will become evident when the functioning of the neural mismatch model is explained.

When an active movement is initiated, a copy of the command signal (efference-copy) is transmitted to the neural store where it retrieves and reactivates the reafferent trace combinations previously associated with it. The function of the comparator is to match the current sensory inputs with the reafferent trace combinations selected from the neural store by

the efference-copy. If there is a discrepancy between the present inputs and these stored patterns, a mismatch signal is generated which triggers the various neural and neurohumoral mechanisms mediating the nausea syndrome and the allied perceptual disturbances. A component of the mismatch signal is also fed back to the neural store where it causes a different retrieval strategy to be adopted. The precise nature of these alternative strategies will be discussed later.

Assumptions governing the strength of the mismatch signal

Three assumptions, derived from the empirical observations stated earlier, govern the strength of the mismatch signal. The severity of the motion sickness reactions and the allied perceptual phenomena are presumed to be directly proportional to the strength of this signal.

(1) The mismatch signal increases as a function of the extent of the discrepancy present in any one sensory channel. In the case of a rotating environment, for example, it would increase in proportion to the angular velocity and the angle through which the head is tilted – factors that determine the strength of the cross-coupled stimulus.

(2) The mismatch signal increases with the number of discrepant sensory channels contributing to the sensory rearrangement. For example, the mismatch signal will be greater when discrepant visual information is superimposed upon a canal-otolith conflict than when vision is absent, other factors being equal.

(3) The mismatch signal is inversely proportional to the degree of consolidation of the reafferent trace combination, where consolidation depends upon the number of previous exposures. This assumption means that even when all other conditions are met for a match between the current sensory inputs and the reafferent trace combination, a mismatch signal will still be generated if the latter has not reached a sufficient degree of consolidation.

How the model works

Inputs from the orientation senses can either be obtained actively through self-produced movements involving voluntary motor control, or they can be imposed passively by some external force. As we have seen, protective adaptation is acquired more rapidly in the former case than in the latter, and the purpose of this section is to indicate why this should be so within the terms of the neural mismatch model.

The self-produced movement case: Figure 4 shows a computer type flow-diagram summarizing the sequence of events involved in adapting to actively-induced rearrangements. This sequence begins, as indicated earlier, with the command signal (say, 'Tilt the head 45° to the left shoulder'). The efference is transmitted simultaneously to the effectors and to the neural store. It is hypothesized that the first instruction given to the neural store is to select the reafferent trace combination having the highest degree of consolidation and the strongest associative bond with the current command signal. To find it, the neural store first seeks a match between the current command signal and the efference components of the spatial engrams within the store. Having found the appropriate engram, it checks the attached reafferent trace combinations for strength, and then transmits the strongest to the comparator for matching with the current reafference. It is precisely this rapidity of the information-retrieval process that is presumed to confer such advantage to the self-produced movement case. Under these conditions, the appropriate reafferent trace combinations are 'addressed' by their associated efferent components, and are thus readily accessed from store.

If the match at this stage is satisfactory (as it would be in the natural force environment), the adaptive sequence stops. But if there is a discrepancy between the stored reafference and current inputs, the mismatch signal is generated which, among other things, is fed back to the neural store.

On receipt of the mismatch signal, the neural store is programmed to select the next strongest reafferent trace combination for matching within the comparator. If this also yields a mismatch signal, the cycle repeats itself until all the reafferent trace combinations associated

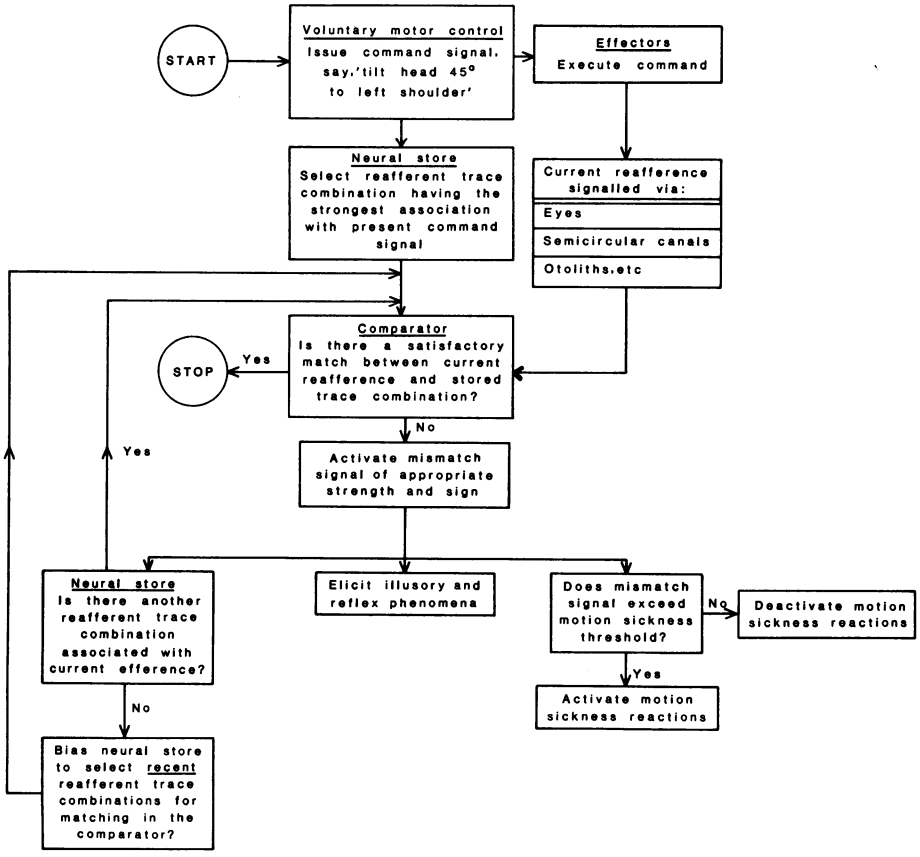

Figure 4. Flow-diagram showing the functioning of the neural mismatch model in the case of self-produced movements

with the command signal have been exhausted, or until an appropriate reafferent trace has been found and consolidated to a sufficient degree for the comparator to accept a match. But if there are no other traces associated with the efference, the neural store is presumed to operate on the third instruction in this iterative process: it becomes biased to select recent reafferent trace combinations for matching. In other words, it selects those trace combinations guaranteed to achieve a match once sufficiently consolidated; namely, those generated by the prevailing atypical force environment. It is at this point that the third assumption governing the strength of the mismatch signal becomes crucial. Since recently-acquired reafferent traces will be too fragile or ephemeral to satisfy the matching criteria adopted by the comparator, they need to be further consolidated by many repetitions of the same combination of sensory inputs. In the case of cross-coupled stimulation, for example, they need to be strengthened by repeated head movements producing the same reafferent combination of visual, canal and otolith inputs. When this point is reached, the comparator accepts the match and the adaptation process is terminated.

When the adapted individual is returned to his typical force environment, a process of re-adaptation occurs which is similar in most respects to the process described above. But there are two important points of difference. First, the appropriate reafferent trace combination will be highly consolidated, so that it will be accessed very rapidly and will be of sufficient strength for satisfactory matching within the comparator. Hence, readaptation will be much quicker than adapting to a novel force environment. Second, the sign of the mismatch signal during the re-

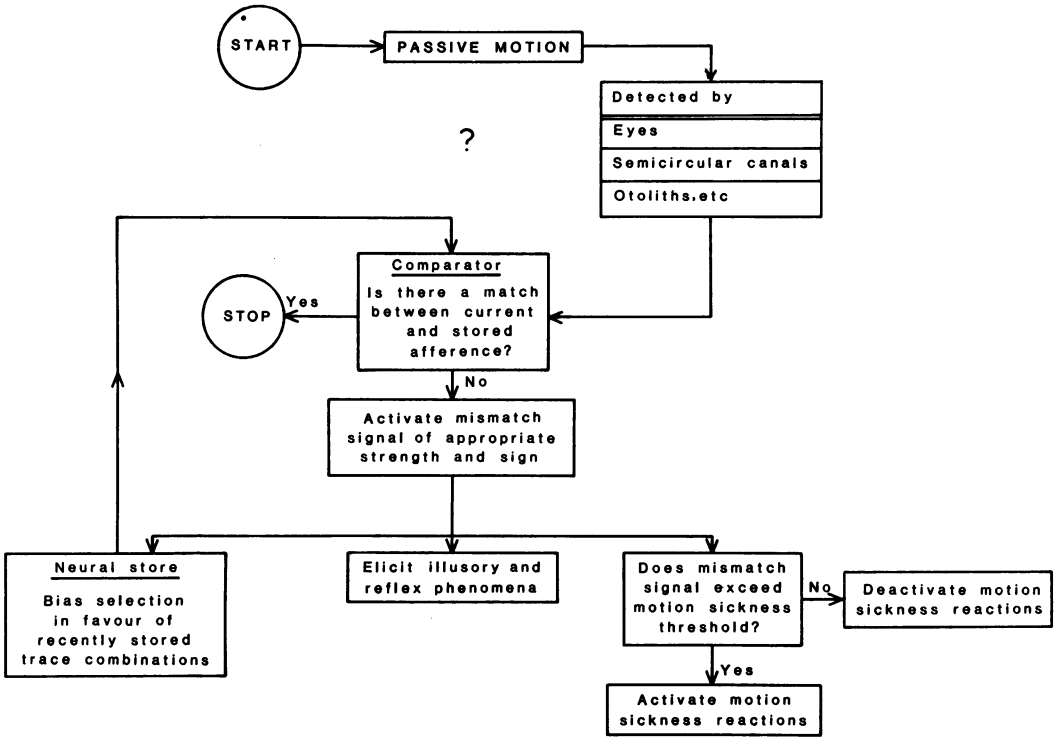

Figure 5. Flow-diagram showing the functioning of the neural mismatch model in the case of passively-induced movements

adaptation period will be reversed with respect to that generated during the initial exposure to the sensory rearrangement. As a consequence, the illusions of apparent motion elicited during this after-effect phase will be opposite in direction to those experienced in the preceding phase.

The passive movement case: When the subject is moved passively by some means outside his control, no appropriate command signal is generated, and so he is denied the rapid accessing of stimulus traces described above. Under these circumstances, the only strategy available to the neural store is to bias selection in favour of recently-stored trace combinations. To what extent these recently-stored inputs from the orientation senses will be organized in the absence of an appropriate efference 'label' is not clear; but the fact that adaptation does occur, albeit slowly, in passive movement conditions suggests that retrieval and consolidation of these traces can eventually occur. It seems reasonable to assume that the speed with which adaptation develops will depend in large measure on the regularity and predictability of the imposed motion. A speculative account of how the model functions under passive movement conditions is shown in Figure 5.

Concluding remarks

Taken together the sensory rearrangement theory and its corollary, the neural mismatch model, constitute partial answers to the basic questions of 'Where does motion sickness occur?', and 'When does it occur?'. On the basis of these theoretical arguments, we may define motion sickness as 'a self-inflicted maladaptation phenomenon that occurs at the onset and cessation of conditions of sensory rearrangement when the prevailing inputs from the visual and vestibular systems are at variance with stored patterns derived from previous transactions with the spatial environment'.

However, this does not explain why the nausea syndrome rather than some other reflex activity should occur in response to conditions of sensory rearrangement. It is hard to imagine that cold sweating, pallor, nausea and vomiting could confer any survival advantage in the conditions that produce motion sickness. Treisman (1977) proposed that motion sickness evolved as an accidental byproduct of an early-warning system for detecting the effects of ingested neurotoxins. Whether or not this is the case, the fact remains that motion sickness is not an inevitable consequence of the human condition: if we had remained as self-propelled animals content to stay within our normal Earth gravity environment, the problem would not have arisen. To this extent, therefore, it is a self-inflicted malady.

Although the theoretical arguments presented here were developed to explain the onset and remission of motion sickness in atypical force environments, they could be applied equally well to understanding the consequences of pathological or surgical disturbances of vestibular function. Unilateral labyrinthectomy, for example, creates a nauseogenic sensory conflict which gradually diminishes as the patient adapts postoperatively to the new pattern of inputs from the orientation senses. Techniques for accelerating the development of protective adaptation in atypical force environments, that were predicated on the neural mismatch model, could also be useful both in minimizing postoperative distress, and in hastening the restoration of normal activity. It has been shown that the acquisition of adaptation to a rotating environment can be achieved very rapidly and with minimal loss of well-being by employing adaptation schedules tailored to accommodate differing levels of susceptibility (Reason & Brand 1975). The basic features of these schedules are twofold: (1) a carefully graded exposure to increasing degrees of sensory rearrangement; and (2) the elimination of concurrent visual conflicts during the initial stages of the schedule (Reason & Diaz 1970), either by occluding vision altogether, or by restricting it to the central portion of the visual field. Considerable evidence has now accumulated to show that the central field of view is minimally involved in determining the position and motion of the body; so that discordant information received through this region of the field is unlikely to contribute in any significant way to the development of motion sickness (Dichgans *et al* 1973, Held *et al.* 1974).

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