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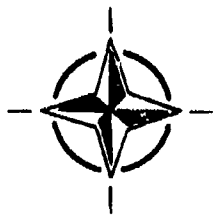
AGARD LECTURE SERIES 175

Motion Sickness: Significance in Aerospace Operations and Prophylaxis

(Le Mal des Transports: son Importance pour les Opérations Aérospatiales et Prophylaxies)

This material in this publication was assembled to support a Lecture Series under the sponsorship of the Aerospace Medical Panel of AGARD and the Consultant and Exchange Programme of AGARD presented on 7th—8th October 1991 in Toronto, Canada, 24th—25th October 1991 in Athens, Greece and 28th—29th October 1991 in De Bilt, The Netherlands.



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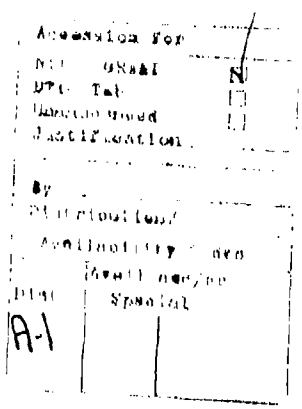

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North Atlantic Treaty Organization
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Contents

Abstract

In aerospace activities, motion sickness, specifically airsickness, continues to be a problem during flying training and in regular operations for airerew and passengers (e.g. paratroops). Simulator sickness can degrade the effectiveness of simulator training and space sickness reduces the efficiency of astronauts. Seasickness is also of aeromedical concern in so far as it affects airerew operating from ships and the survivability of ditched aviators.

The Lecture Series has been designed, primarily, to aid practising Flight Surgeons in the performance of their primary care duties. It should also be of interest to others who wish to obtain an overview of recent advances in the understanding of the aetiology and treatment of motion sickness:

The topics to be covered in the 12 lectures are:

1. Clinical features of motion sickness
2. Operational significance of motion sickness
3. Aetiology and neurophysiological mechanisms
4. Physical characteristics of provocative motion.
5. Special features of air, space, sea and simulator sickness
6. Selection and assessment of susceptibility
7. Prophylaxis and management.

The Lecture Series will conclude with a Round Table Discussion.

This Lecture Series, sponsored by the Aerospace Medical Panel of AGARD, has been implemented by the Consultant and Exchange Programme.

Abrégé

Dans les operations aerospatiales, le mal des transports, et, en particulier, le mal de l'air, continue à poser des problèmes pour les équipages et les passagers (c'est à dire les parachutistes), qu'il s'agisse de missions operationnelles ou de vols d'entraînement. Le mal de simulateur peut nuire à l'efficacité de l'entraînement en simulateur et le mal de l'espace reduit les capacites des spationautes. Le mal de mer est également un domaine d'etude aéromédical dans la mesure ou, il concerne les équipages bases à bord de navires et la suvivabilité des aviateurs consécutif aux amerrissages.

L'objet principal de ce cycle de conférences est d'apporter une aide aux médecins de l'air dans l'administration des soins primaires. Il intéressera d'autres personnes voulant obtenir une synthèse des derniers progrès enregistrés dans le domaine de l'aetiologie et du traitement du mal des transports.

Les sujets qui seront traites lors des 12 conférences sont les suivants:

1. Les aspects cliniques du mal des transports
2. L'impact opérationnel du mal des transports
3. L'aétiologie et les mécanismes neurophysiologiques
4. Les caractéristiques physiques du mouvement provocant
5. Les particularités des maux de l'air, de l'espace de mer et de simulateur
6. La seléction et l'évaluation de la susceptibilité
7. La prophylaxie et la gestion

Un table ronde sera organisée en fin de séance.

Ce cycle de conferences est présente dans le cadre du programme des Consultants et des Echanges, sous l'égide du Panel AGARD de Medecine Aerospatiale.

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SIGNS AND SYMPTOMS OF MOTION SICKNESS AND ITS BASIC NATURE

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ABSTRACT

The cardinal signs and symptoms of motion sickness are malaise, pallor, (and or/flushing), cold sweating, abdominal discomfort, changes in gastric motility, and changes in levels of circulating hormones. Cardiovascular, respiratory, and other signs have also been reported, as have a variety of other sensations, feelings and performance changes.

It is reasonable to think that motion sickness is basically the activation, by motion, of a poison-response mechanism.

BASIC NATURE

In essence, motion sickness is the activation, by motion, of a poison-response mechanism. This is obvious: motion gives rises to vomiting.

In certain motion environments, something that must be called "peculiar" happens. Motion sickness occurs. Motion sickness will be described here, and the description of motion sickness will be based on the assumption that only one peculiar thing happens: a poison response is provoked by motion. Common sense suggests that two or three peculiar things do not independently occur at the same time.

There are two currently-popular theories of why this peculiar thing happens, why motion gives rise to a poison response. The older of these two theories, the conflict theory, holds that in motion sickness environments the poison response occurs because of a conflict. In a motion sickness environment, the pattern of sensory inputs concerning orientation and motion (vestibular, visual, and proprioceptor inputs) is in conflict with the pattern of inputs anticipated on the basis of previous experience (6,17). The conflict somehow gives rise to vomiting. The theory (unmodified) does not explain why such a conflict is utterly unable (8,9,14,15) to provoke vomiting in individuals who are lacking the vestibular apparatus of the inner ear. The theory also does not explain why such a conflict should produce vomiting instead of deep breathing, discharge from the nose, or orgasm. The theory would apply equally well if the conflict p oduced one of these things instead of vomiting.

The more modern theory, that of Treisman (25), incorporates the conflict theory and does explain why vomiting should occur. Treisman's theory holds that the conflicting sensory inputs are interpreted centrally as neurophysiological dysfunction caused by poisoning. Treisman postulated that the brain stem mechanisms of orientation and motion normally perform an additional function as well as maintenance of bodily equilibrium, stabilility of gaze, etc: the additional function is to detect and respond to certain poisons. In motion sickness situations the conflict in sensory inputs simulates poisoning in those neural mechanisms.

There is convincing evidence of the basic validity of Treisman's theory. It seems clear that the brain stem mechanisms of orientation and motion do in fact function also to detect and respond to certain poisons. The experiment showing this (13,14) found that, in experimental animals, surgical removal of the vestibular apparatus of the inner ear rendered the animals defective in the emetic response to certain poisons injected intramuscularly. This is positive evidence that the vestibular system is involved in poison response. The surgery encroached only on the ear and, of course, it also rendered the animals completely nonsusceptible to motion sickness. The vestibular system is involved in poison response in circumstances of motion sickness and also in circumstances of poisoning with certain toxic chemicals.

Motion sickness is a poison response provoked by motion. It is a poison response provoked by motion acting (directly or indirectly) on the ventibular system.

SIGNS AND SYMPTOMS

The signs and symptoms of motion sickness can be considered the manifestations of the poison response. Although Treisman confined his theory of motion sickness to nausea and vomiting (25), it is clear that, whenever vomiting is provoked by motion, other bodily responses are also provoked, and sometimes these other responses occur and vomiting does not occur. Sometimes pallor, cold sweating, and malaise occur in a motion environment and vomiting does not. Motion sickness is a poison response provoked by motion, not just vomiting.

Motion sickness is possibly the purest and simplest poison response available for study, because it can be produced for study without the complicating presence of a poison. It is produced by a motion stimulus that masquerades as a poison. It is interesting to think that a person known to be poisoned (for example, by apomorphine)

would think that the poison is a terrible one because of the horrible nausea, pallor, cold sweating, vomiting, etc; but the reality might be that the dose of apomorphine had no adverse effect on the body, except that it triggered one of the body's poison-response mechanisms; then the body inflicted all those miseries on itself. A poison that causes facial pallor might not do so by a direct influence on the blood vessels of the skin, or even by a direct influence on the autonomic nervous system; it could do so by activating one of the body's poison-response mechanisms, and that mechanism could then act on the hypothalamic centers controlling the autonomic nervous sytem to produce the facial pallor. In motion sickness it seems that when the poison-response mechanism is activated the body then inflicts on itself a variety of signs and symptoms, to be described below.

Motion sickness signs and symptoms are often thought of as, first, nausea and vomiting, and second, all the "other" ones. However, as indicated above, it will be assumed here that only one peculiar thing happens in motion sickness environments: a poison response is provoked. There is no reason to think that anything else is happening, and all the signs and symptoms will be considered to be part of the poison response. The signs and symptoms of motion sickness will therefore be classified as, first, those associated with emptying the stomach, and second, those associated with counteracting or surviving the part of the poison detected (incorrectly) in the bloodstream.

There is a considerable number and variety of signs and symptoms that differ in different individuals (1,2,3,4,6,12,17). However, some signs and symptoms appear to occur in all, or almost all, cases of motion sickness in humans, and only these signs and symptoms will be listed here.

SIGNS AND SYMPTOMS ASSOCIATED WITH EMPTYING THE STOMACH

Abdominal awareness
Abdominal discomfort
Nausea
Motility of the stomach
Vomiting

SIGNS AND SYMPTOMS ASSOCIATED WITH COUNTERACTING OR SURVIVING THE POISON IN THE BLOOD

Malaise
Sleepiness
Headache
Pallor (and/or flushing)
Cold sweating
Cardiovascular changes
Endocrinological changes

Abdominal awareness, abdominal discomfort, and nausea are possibly parts of a gradient of a single symptom that would have the effect of preventing the further consumption of a toxic food. They would also tend to have the effect of preventing the consumption of that food again on future occasions (25). They are possibly the conscious reflection of unusual activity in the central emetic mechanisms (6,12).

Motility and tonus of the stomach decrease with motion sickness in most human subjects (7,12). Gastric (and intestinal) motility has been observed in motion sickness in humans by listening to the gut sounds with stethescopes and microphones (24); the sounds diminish, usually to silence, with motion sickness in spaceflight. X-ray studies of the gut before and after exposure to nauseogenic motion (16) also reveal decreased motility with nausea in most subjects. Balloons have been placed in the stomachs of humans subjected to motion sickness (3,7,26); overall, decreases of tonus and motility predominate, but some prominent exceptions are reported (3). In one subject with a gastric fistula, direct visualization of the stomach during nausea was possible, and this subject together with three normal subjects with gastric balloons was exposed to vestibular and other nauseogenic stimuli; "nausea occurred only during gastric relaxation and hypomotility" (26).

The electrical activity of the gut has also been studied during motion sickness (6,19,20) using electrodes attached to the skin over the stomach. The electrical activity recorded in this way is called the electrogastrogram or EGG. The EGG changes with motion sickness; the change is typically a decreased magnitude of the EGG voltage (6,20) and an increase in the basic electrical rhythm from the normal 3 cycles per minute to 5-7 cycles per minute (19,20). This increase of the basic electrical rhythm of the EGG, although it is called tachygastria, is in fact associated with gastric stasis, decreased gastric motility (20,23), so that the increased EGG frequency is consistent with the decreased gastric motility observed with microphones, balloons, and x-rays. The decreased amplitude of the EGG is also associated with decreased motility.

The EGG is an index of interest in studies of motion sickness; it is an index of something underlying motility. There is no simple relationship between recorded EGG values and, for example, recordings (in dogs) from force tranducers fixed to the gastric serosa (18). The basic electrical rhythm of the EGG reflects "pacesetter potentials of the stomach, but not gastric contractions" (20). However, when gastric contractions do occur they are time-locked to the EGG. At the higher basic EGG rhythms, contractions

tend not to occur at all. Also, "the amplitude of the EGG increases when a contraction occurs" (20). In motion sickness, both the increased frequency of the basic electrical rhythm of the EGG and the decreased EGG amplitude indicate decreased gastric motility.

It is possible that the decrease in gastric motility would have survival value in someone who had ingested a poison: it would tend to keep the poison in the stomach, where absorption might be slower and where vomiting could remove it.

Vomiting removes the poison (that is not there) from the stomach. The physiology of vomiting has been recently reviewed (6).

It is not clear how (or whether) malaise, sleepiness, and headache would improve the chances of surviving poisoning, but they would tend to encourage the poisoned individual to lie down.

The pallor, the cold sweating, the cardiovascular changes, and the endocrinological changes of motion sickness can all be seen as parts of a general stress response that includes an overall activation of the sympathetic nervous system (SNS). The stress response (it is assumed) enhances the chances of surviving poisoning, and it occurs in the absence of any stress on the body, for the same reason that vomiting occurs without any poison in the stomach: a poison-response mechanism has been activated. It should be noted that most motion sickness environments do not stress the body; babies sleep in these environments. Only if the poison response is provoked does the stress response appear (in the absence of stress).

It is unlikely that pharmaceuticals selected to counteract the autonomic signs of motion sickness would be satisfactory, because no one suffering from motion sickness cares about the autonomic signs; only pharamaceuticals that counteract the nausea and vomiting are relevant to the sufferer's desires.

Pallor is a result of vasoconstriction in the skin, probably in response to an increase in the SNS activity to the blood vessels of the skin (6), and it is a sign of motion sickness that is almost invariably (3) seen before vomiting. It has been found recently, however, that in some individuals there is flushing as part of the skin's reaction in motion sickness (11). Sweating (eccrine sweating) similarly results from an increase in activity in the SNS supply to the sweat glands, although the postganglionic SNS supply to the sweat glands is cholinergic (6). The cardiovascular changes of motion sickness are also consistent with an overall activation of the SNS: the pulse rate increases slightly (2,3) and the blood flow to skeletal muscle increases (21).

The endocrinological changes with motion sickness also resemble a stress response. Recent studies have found that humans in motion sickness have increased circulating levels of AVP, ACTH, EPI, NE, GH, and PRL (5,6,10,22). AVP means antidiuretic hormone, ACTH means adrenocorticotrophic hormone, EPI means epinephrin, NE means noradrenalin, GH means growth hormone, and PRL means prolactin.

Although this particular collection of autonomic and endocrinological responses is typical of a stress response, it is not clear exactly how it would improve the chances of surviving a poison. Perhaps this would be clear if it were known what poison the body thinks it has ingested in motion sickness; or perhaps the response is appropriate for several, or even most, poisons.

To the extent that motion sickness is nausea and vomiting, the "other" signs and symptoms are peculiar epiphenomena, but if it is assumed that only one peculiar thing happens in motion sickness (a poison response is provoked by unnatural motion), then all the signs and symptoms must be part of that poison response. If the autonomic effects were peculiar epiphenomena, it would be difficult to understand how their manipulation by cortical control, using autogenic feedback, could influence the nausea and vomiting: it would be like pushing on a string. However, if the autonomic effects are part of a poison response it is immediately understandable that a cortical input to the autonomic effectors could take a route (for example) through the poison-response mechanism and affect also other parts of the poison response, such as nausea and vomiting. Autogenic feedback training does, of course, influence nausea and vomiting, but how it does so is perhaps easier to understand if it is assumed that all signs and symptoms of motion sickness are part of a poison response.

If cortical input to the autonomic effectors of motion sickness can exert a desired influence on the nausea and vomiting, then perhaps (with training) a cortical influence on the sleepiness and malaise might be beneficial also; the cortex might convince itself (by way of the poison-response mechanism) that it feels fine, that it is in excellent health, and that it is wide awake; in doing so it might attenuate the nausea and vomiting.

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MOTION SICKNESS AND ITS RELATION TO SOME FORMS OF SPATIAL ORIENTATION: MECHANISMS AND THEORY

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INTRODUCTION

The mechanisms of motion sickness fall under three component topics, which separately involve distinctive subject matters and together challenge the entire scope of neuroscience disciplines. The components are: 1) The process involved in the sensorimotor and perceptual-motor adjustment to the sustained experience of unusual motion. 2) The neurochemical link whereby the neurochemical processes and byproducts of sensorimotor adjustment accumulate to a threshold level that when exceeded elicits the sickness syndrome. 3) The sickness syndrome, which includes emesis and all of the autonomic and psychological accompaniments that degrade performance.

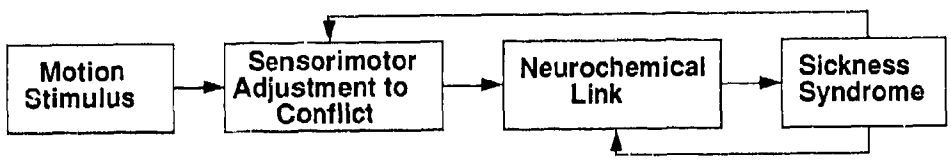

Figure 1. Motion sickness involves three component topics.

The three components, though distinctive, are interactive. Emesis often gives relief so that unusual motion can again be tolerated, at least for a while, but the malaise associated with the sickness syndrome may reduce willingness to remain in the motion environment long enough to achieve adaptation. A quick summary of where we stand today is that much remains to be understood under each of these topics but fortunately for the newcomer to this field, excellent reviews and overviews are available (Crampton 1990, Benson 1988b, Davis et al., 1986, Graybiel 1969, Money 1970, Reason and Brand 1975, Tyler and Bard 1949).

OVERVIEW

A verbal picture by Crampton (Carr and Fisher 1973) several years ago, presented here as Figure 2, provides an overview of the mechanisms of motion sickness. An homunculus, who reportedly lives in the cerebellum, has the job to receive messages from the motion sensors, the vestibular nuclei and higher levels, and to operate a switchboard appropriately to initiate fast automatic sequences of motor reactions that will improve the quality of motion control. For voluntary motion, he receives advance information concerning the goal of the movement and he also is aware of the state of the muscles, in advance. When messages from motion sensors are either in conflict with one another or occur in an unfamiliar pattern, the homunculus is confused and with confusion of sufficient length or intensity, he will sweat or vomit and contaminate the cerebro-spinal estuary so much that chemical sensors on the floor of the fourth ventricle signal the environmental protection agency to commence disposal actions.

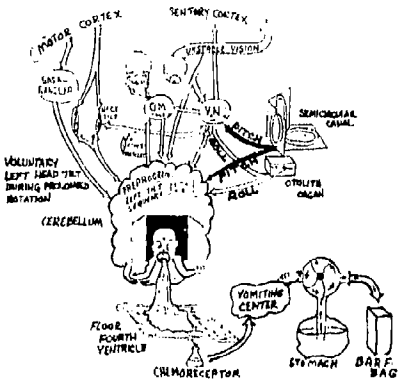

Figure 2. A cartoon illustrating the mechanisms of motion sickness. Above the homunculus are the sensory motor mechanisms involved in motion sickness while below is the neuro chemical link to the vomiting center. The pump represents the 'all or none' vomiting reflex which is part of the complex sickness syndrome. Recently, the locus of Chemoreceptor concept and the concept of a single vomiting center have been challenged.

SENSORY SYSTEMS INVOLVED MOTION SICKNESS

The sensory-motor systems involved in motion sickness are those that detect motion relative to the earth and, by their central neural processes, generate motor responses that serve to maintain balance, to stabilize vision through oculomotor control and in general serve to improve the quality of the control of motion relative to the earth. The vestibular system, the visual system, and the proprioceptor system (the pressure sensing properties of the skin and the sensory processes of the muscles and joints) are the principal systems.

Of these, only the vestibular system is exclusively dedicated to: 1) detecting motion of the head and body relative to the earth, and 2) generating reflexive motor activity that improves motion control while motion is in progress. The vestibular system is more like a sensorimotor system than a simple sensory detector. Vision, audition, olfaction and touch often serve to locate and detect motion of objects relative to the body. The vestibular system does not detect objects in the environment but rather serves to provide 3-dimensional earth-reference to visual and auditory images of objects, slopes and paths as the head changes orientation relative to the environment. From this perspective, sensory messages from muscles and joints and tactile information from weight-bearing surfaces are allied to the vestibular system in the business of controlling motion relative to the earth and in detecting the direction of gravity in order to maintain balance and control motion relative to the earth.

A cornerstone fact in the present fund of knowledge on motion sickness is that a functional vestibular system is necessary for the occurrence of motion sickness. Efforts to evoke motion sickness in man and animals deprived of vestibular function, have consistently failed (James 1882, Kennedy et al., 1968, Reason and Brand 1975, p.86, Sjoberg 1929, Wang and Chinn 1956). Even partial destruction of different vestibular endorgans confers a degree of immunity, at least until central compensation for the partial peripheral loss has occurred (Igarashi, 1990). Because of its central role in motion sickness, understanding of the vestibular system is necessary to the appreciation of the stimuli that initiate motion sickness.

THE VESTIBULAR SYSTEM

It is beyond the scope of this chapter to provide a detailed description of the vestibular system which has been done many times before (Wilson and M. Jones, 1979), but an overview of some principal characteristics is necessary for discussion of the mechanisms of motion sickness. Figure 3 provides a summary overview of vestibular connections to the systems involved in motion sickness.

The vestibular sensory endorgans consist of two kinds of sensors: the semicircular canals that detect angular acceleration of the head and the otolithic membranes that detect tilt relative to gravity. These two sets of acceleration detectors are located in fluid-filled tubes and sacs that are firmly attached to and encased in a labyrinthine cavity in the petrous portion of the temporal bone of each ear. The receptors in either ear are capable of detecting and setting off reactions to all of the directions of angular and linear motion that are normally detected by both ears.

SEMICIRCULAR CANALS

The semicircular canals detect angular acceleration and are not affected by linear acceleration or by different orientations relative to gravity. The semicircular canals respond in exactly the same way irrespective of whether they are at the center of rotation or at some radial distance from the center of rotation.

A simplified diagram of the structure of these receptors within one ear is shown in Figure 4. The planes of the semicircular canals lie approximately at right angles to one another. Each canal is filled with a fluid, endolymph, which, by virtue of its inertia, is displaced slightly relative to tube walls whenever the head experiences an angular acceleration in the plane of that canal. Displacement of the endolymph deflects the cupula, a structure which behaves like a damped drum because it seals an expanded portion of each canal, the ampulla. The amount by which the cupula is deflected is communicated to the vestibular receiving areas of the brain by sensory hair cells lying at its base because hairs from these cells extend upward into the gelatinous cupula.

As well as conveying information about the rate at which the head is being turned, these signals also generate reflex eye-movements, whose primary function is to maintain stable retinal images of the visual world. Though stimulated by angular acceleration, the dynamics of the cupula-endolymph system are such that the sensory signal from semicircular canal afferents is proportional to angular velocity of the head. The semicircular canals thus act as angular speedometers capable of sensing angular velocity in any direction as the head is rotated, when the frequency of head angular velocity approximates frequencies of natural voluntary movement.

OTOLITH ORGANS

The otolithic membranes, in the utricle and the saccule, function like density-difference linear accelerometers. The specific gravity of the calcite crystals in the utricular and saccular membranes is much greater than that of the surrounding gelatinous and fluid medium. When the head tilts to the right, the utricular membrane slides right due to the weight of the embedded crystals thereby bending cilia of underlying hair cells. The pattern of cilia deflection signifies rightward tilt of the head. A different pattern of haircell activation would signify different directions of head tilt.

A primary function of the otolithic system is to indicate the orientation (tilt) of the head (and body) relative to gravity. Notice however that if the head and body were accelerated linearly to the left, the utricular otolith membrane would again slide toward the right ear, even through the head remains upright relative to gravity. The otolith system can respond in the same way for tilting of the head or for a horizontal linear acceleration so that it does not always signify tilt of the head (and body) relative to gravity. Recordings from different otolith primary afferents show that some units respond maximally for particular tilt directions and angles whereas others appear to be influenced by movement rather than position of the otolithic membrane (Fernandez and Goldberg, 1976).

When horizontal linear accelerations are sustained (as can be done by maintaining fixed head position relative to a constant centripetal acceleration vector on a centrifuge) tilt is perceived as would be expected by the "steady-state" displacement of the otolithic membranes. But when the linear acceleration is brief and changing, then the person perceives linear movement. Movement-sensitive otolithic afferents may provide information on the linear velocity of movement during changing linear accelerations if the frequency characteristics of the change yield continual movement rather than "steady state" displacements of the otolithic membrane. However, how would the vestibular system immediately discriminate horizontal linear oscillation of the head from rapid to and fro tilts of the head when both would involve continual movement of the otolithic membrane? Here the absence of semicircular canal angular velocity signals coupled with the presence of otolith signals could discriminate linear movement from dynamic tilt; dynamic tilting produces both semicircular canal signals and otolith signals.

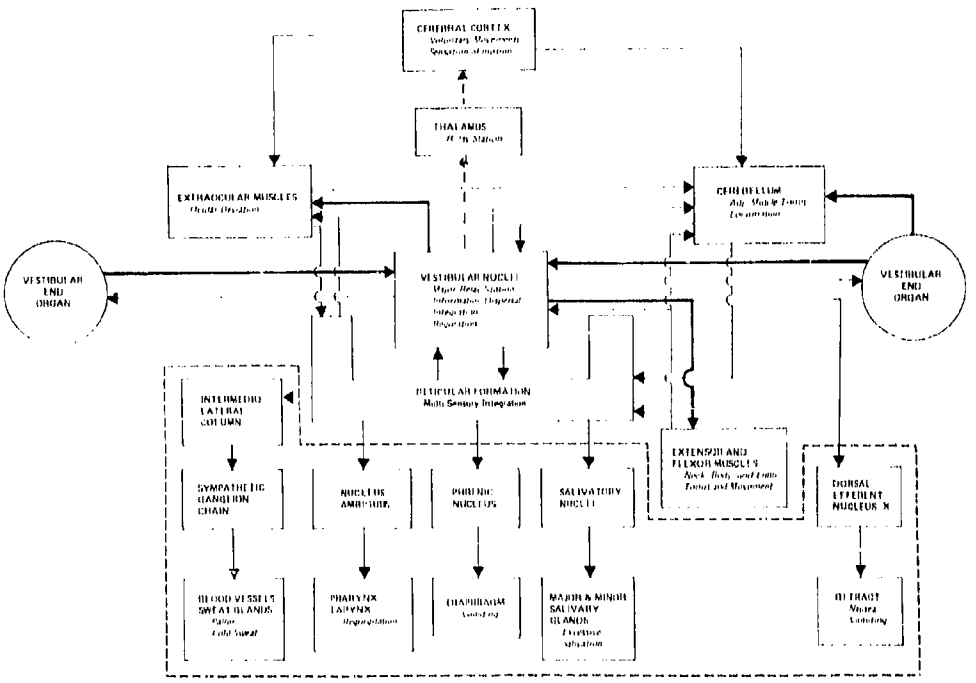

Figure 3. Interrelations of various neural structures associated with the vestibular system. Structures involved in reflexive and purposive behavior elicited by physiologic vestibular stimulation are indicated by heavy dark arrows. Structures associated with vegetative effects elicited by unusual vestibular stimulation are bounded by the dashed line. Modified from Correia and Guedry (1978).

CANAL/OTOLITH INTERACTIONS

Functionally, the semicircular canals tell the brain about the rate of rotation of the head relative to the earth when head movements approximate the temporal (frequency) characteristics of natural head motions. At much lower frequency of angular oscillation, the canal signal can be considerably phase displaced relative to the head velocity so that a turning motion may be perceived as stopped and even reversed in direction before the head movement actually stops. The canals locate the axis (or plane) of turn relative to the head but they do not provide information on how that plane of head rotation is oriented relative to the earth. For example, when the head is turned from leftward gaze to rightward gaze, the horizontal canals provide rate of turn information throughout the turn and the brain can integrate the velocity information to know how far the head has turned (Bloomberg et al., 1991, Guedry 1974). The predominant horizontal canal signal indicates that the head turn is in the yaw plane of the head. If the gaze is change . by movement of the head and eyes from extreme upward gaze to downward gaze toward the feet, the vertical canals indicate the rate of head turn throughout the turn and the velocity information can be integrated by the brain so that the angular extent of the pitch plane movement is known (Guedry et al , 1971). But the plane of rotation relative to the earth is not known from the canal information; sensory information from the canals would be the same irrespective of whether the person were standing upright, lying on the side, or supine or prone.

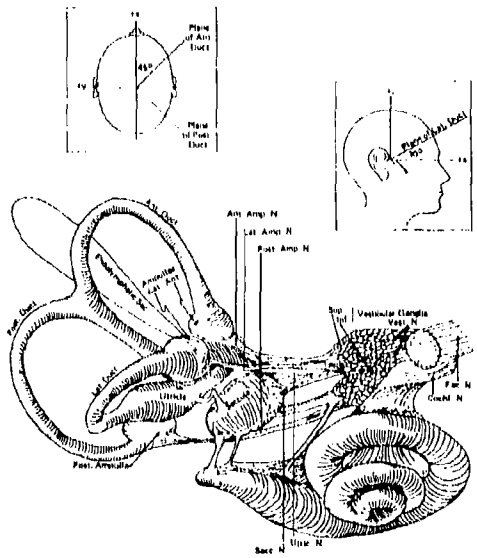

Figure 4. Orientation of the semicircular canals and otolith organs (utricle and saccule). Inset figures illustrate planes of the semicircular canals and also the x, y, and z head axes (cf Hixson, et al., 1966). Modified from Correia and Guedry (1978).

The otolith-bearing sensors provide information that locates the plane of the movement of the head relative to the earth while the canals provide angular rate information throughout the movement. Whenever pitch (or roll) head movements are made while the person is standing or walking in normal upright posture, the otolithic system produces a sequence of angular position signals (relative to gravity) that match the integrated velocity information from the canals. During pitch forward head movement from upright posture, if at some point the otolithic sensors indicate pitch-forward head position of 30 degrees relative to gravity, the integrated rate information from the canals indicates 30 degree displacement from start position and gaze can be redirected back to start position if the person so chooses, even in the dark (Bloomberg 1991, Guedry et al., 1971).

Effective vs. Aberrant Interactions

Assume now that, through biochemical intervention or an inflammatory process, the gain of semicircular afference is increased to twice the usual signal. At the end of a 30 degree head pitch forward from upright, the otolithic sensors would signal a 30 degree pitch forward position but integrated semicircular canal information would indicate 60 degree angular displacement from start position. Gaze control would be problematic, to say the least. Moreover during the head movement the rate of head movement experienced would be much too great for the angular positions being achieved. Interestingly, exactly this kind of canal/otolith neural mismatch is produced during a simple centrifuge run (Guedry and Oman 1990) and subjects are confused, disturbed and deplore centrifuge training because of the repugnant experience (Guedry and Rupert 1991/a).

If this hyperactive canal condition were to continue during daily activities, the brain could impose a reduction in gain of vertical canal neural inflow at some level before the perceptual-motor reactions are generated, returning motion control toward a state of normalcy but the neural-mismatch would induce motion sickness during the adaptive process and then again when the adapted individual returns to his normal environment.

Whereas all head movements made from upright posture involve a tight coupling between otolith information and vertical canal information, many yaw head movements made from upright posture involve very little angular position change relative to gravity. However, the axis of head turn is also frequently inclined relative to gravity during yaw head turns (even during upright posture) and so the CNS, which is adapted for tight canal/otolith coupling for the vertical canals, may be adapted for more varied interactions from the horizontal canals. This may explain differing sickness incidence observed for vertical and horizontal canal stimulation on earth (Benson and Guedry 1971, Guedry et al., 1990) and during micro-gravity missions (Lackner and Graybiel 1984).

Assume now that two pitch head movements are made through 90 degrees from upright posture and that the two movements are identical in total angular displacement and in the angular velocity profile during the movements. But one is made by bending at the neck while the other is made by bending from the hips. Semicircular canal information would be identical but otolith information would be different. Initial and final pitch positions indicated by the otolithic receptors would be the same but transitional information would be different. Tangential acceleration of the head (in one direction during starting and in opposite direction during stopping) reaches much higher magnitudes during the trunk movement. The head traverses a greater path. Backward displacement of the utricular otolith by tangential acceleration at the beginning of the trunk movement soon gives way to forward displacement as gravity components increase in the utricular shear plane. The more rapid movement of the utricular otolith membrane during trunk movement may reflexively influence the differing muscle actions required in stopping the two movements. Such head movements initiated voluntarily are substantially influenced by feed-forward preprogrammed mechanisms but if the gain of the otolith velocity information were to be altered so as to be discrepant with the preprogrammed message, then the movement might be less well-controlled and accompanied by a report of dizziness and confusion. Head movements made in hyper-G environments yield both transient (Gilson et al., 1973, Guedry and Rupert 1991/b) and steady-state (Correia et al., 1968) effects that are disorienting and the transient effects are confusing and nauseogenic. Head movements made in the Hyper-G environment that occurs during the reentry profile of space shuttle following adaptation to the Hypo-G orbital environment--these head movements are likely to produce transient disorienting effects.

Canal/otolith interactions range from those that are clearly functionally effective, to many that may constitute either conflict or functionally effective interactions (advance in vestibular neuroscience is needed to decide), to those, such as the cross-coupled angular velocities, that are unmistakably sensory mismatches.

Interestingly, the clear canal/otolith conflict from cross-coupled angular velocities can be resolved into benign concordant stimuli by subtle changes in accompanying stimuli. The "Coriolis cross-coupled" stimulus is produced by tilting the head during sustained whole-body rotation. This stimulus has played a major role in popularizing sensory conflict as a source of motion sickenss. For example, if the head is tilted forward during sustained clockwise whole-body rotation at 15 rpm, the semicircular canals immediately signal a roll-right head velocity due to cross-coupled angular velocities (Guedry and Benson 1978, Guedry 1974, Guedry 1970). At this instant otolith afferents signal a pitch-forward head position change, a clear mismatch with the roll-right angular velocity information from semicircular canals. The brain has been asked to set-off oculomotor and other postural reflexes to compensate for motion in two different directions simultaneously. Perceived orientation-change is paradoxical, confusing and unpleasant.

The subtlety of canal/otolith interactions is nicely illustrated by the fact that this extreme disorientation-stress reaction gives way to an accurate and benign motion perception if the head movement is made during the angular acceleration of the rotator (or immediately after 15 rpm angular velocity is attained). The accurate perception is explained by simple physics. During angular acceleration, the horizontal canals sense angular velocity in the yaw-axis head plane. Thus the brain has an accurate yaw-axis angular message when the head pitch is initiated. When the head is pitched forward, cross-coupled angular velocities produce a roll-axis velocity signal to the vertical canals which combines with the already present yaw axis signal to yield a vector that remains perfectly aligned with gravity (Guedry and Benson 1978). Thus the canal/otolith mismatch is avoided, the VOR is appropriate in plane to compensate for the plane of head motion relative to the earth and the motion perceived is essentially accurate, not confusing or disturbing. When the head-tilt is delayed until the yaw-axis canal signal has dissipated, then the highly disturbing canal/otolith conflict is fully appreciated. The immediate after-effects of optokinetic stimulation can also be used to cancel the conflict (Guedry, 1978) because visual motion can simulate, in the vestibular nuclei, horizontal canal (yaw-axis) stimulation (Waespe and Henn 1977, 1978).

Interactions between the canals and otolith systems that have common (everyday) functional value is a topic that has been relatively neglected by vestibular neuroscientists and it is critical to any motion sickness theory that rests on the presumption that sensory conflict is important to the provocation of motion sickness. Fortunately interest in this topic is developing (Benson 1974, Curthoys et al. 1991, Gresty et al., 1991, Guedry 1991/c, Paige and Tomko 1989, Raphan and Cohen 1986, Raphan et al., 1979).

Whereas the semicircular canals react identically to angular acceleration, , irrespective of their distance, r, from the center of rotation, the otolithic receptors, which are not responsive to angular acceleration, per se, are nevertheless stimulated during angular accelerations whenever the head is displaced from the center of rotation because rotation at a radius yields tangential linear acceleration. For example, with the head fixed in forward-facing tangential heading, but displaced radially from rotation center, angular acceleration, yields tangential acceleration, r. With continued angular acceleration another vector, $\Omega^2 r$, that was negligible initially, increases rapidly as angular velocity, Ω, increases.

These two vectors are at right angles to one another and with the head positioned as shown in Figure 4, they would lie approximately in the utricular plane. During angular acceleration, the resultant of these two vectors is a vector that rotates in the utricular plane. Here we are not really ignoring gravity, as it might seem, but we are attending to that component of the total linear acceleration (gravity, tangential and centripetal components) that acts in the mean "shear plane" of the utricular otolithic membrane.

This resultant vector rotates in the shear plane during the course of the angular acceleration in a counterclockwise direction, depicted in Figure 5, while the semicircular canals signal (in this example) counterclockwise rotation of the head. During deceleration of the head the resultant vector rotates in the utricular shear-plane in the same direction as during the acceleration but now the semicircular canals, indicate rotation in opposite direction. Whether or not these different canal-otolith interactions have functional utility in natural movement is not known but perceptual effects on centrifuge runs suggests that such differences in canal/otolith interactions may serve to differentiate turning-at-a-radius from turning about a body-axis (Guedry, 1991/c). This interpretation differs considerably from assuming that one configuration represents neural mismatch while the other represents functionally effective interaction.

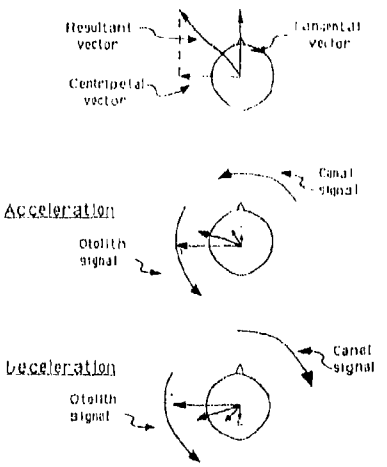

Figure 5. Centripetal and Tangential vectors form a resultant vector that rotates in the mean utricular shear plane. During acceleration, this vector rotates in the direction signaled by the semicircular canals. During deceleration, it rotates opposite the direction signalled by the semicircular canals.

SENSORY CONFLICT, NEURAL MISMATCH

A generally accepted hypothesis today is that conflicting messages from sensory detectors of motion produce motion sickness. This idea, which is not new (Guedry 1970, Irwin 1881, Lansberg 1960, McIntyre 1944, Oman 1990, Reason 1978), has gained favor over the notion that vestibular

overstimulation is responsible for motion sickness because very mild vestibular stimuli produce sickness when sensory conflict is present whereas some strong vestibular stimuli can be repeatedly tolerated without sickness. A partial melding of these ideas may be called for, viz., very strong vestibular stimuli, in the absence of apparent sources of exteroceptor conflict, may produce sickness when their continued presence threatens the subconscious but pervasive imperative to control whole-body motion relative to the earth. Nevertheless, sensory conflict is a significant factor in stimuli that induce sickness.

Some examples from experiences of everyday life will serve to clarify the nature of sensory conflict and its relation to control of motion. While in the well of a small cabin cruiser that is moored to a dock in a boat shed, a person proceeds without pause to inspect instruments within the boat and to check the engine. Vestibular sensations from a gentle rocking of the boat are concordant with peripheral visual motion cues from surrounding dock and the visual/vestibular interaction aids balance control, even though the vestibular and the peripheral visual messages fail to reach conscious awareness. Suddenly to maintain balance and to allay a little alarm signal, the person grasps for support and shifts gaze to find the source of alarm. A fairly large boat has entered the adjoining slip and produced a large moving peripheral vision message, discordant with concurrent vestibular information. Balance control is threatened until the person's spatial model and motor control strategy are adjusted. A similar experience occurs when a person places the palm of the hand against a wall in order to relax by leaning against the wall, which happens to be a sliding panel, not an earth-fixed wall. Again a little alarm, a diverted gaze to understand and a postural adjustment serve to restore effective control of motion and orientation relative to the earth. Both situations involve sensory mismatches in the context of an operating spatial model and motion control objectives; they challenge built-in demand to maintain control of motion and orientation relative to the earth. In these examples, the sensory feedback associated with motion control would have remained below conscious awareness had the conflict not occurred and in both examples the conflict was resolved on a conscious level. In many motions that induce sickness, conflicts may be sensed only as slightly confused perceptions and the alarm reactions may remain subconcious. Even for alarm reactions that penetrate conscious awareness due to severe sensory conflict, the reason for the alarm reaction cannot be understood by quick inspection unless the observer is very well informed on the role of sensory conflict in motion sickness. This is the nature of sensory conflict in motion sickness.

CONFLICT VS. MISMATCH

The phrase sensory conflict carries implications that do not clearly fit a number of stimulus conditions that provoke motion sickness. While conflict is an appropriate term for "Coriolis cross-coupled" stimulation, a number of nauseogenic motions involve stimuli in which one sensory input persistently calls for a reaction while information from another sensor is "abnormally" absent. Such is the case for sustained off-vertical rotation in which a continual reorientation relative to gravity of the otolithic sensors is unaccompanied by semicircular canal information (Benson 1988/b, Benson 1974, Guedry 1965/a), information normally present when the head is voluntarily turned about the head's z-axis while the axis of head turn is off-vertical. A brief natural head turn about a tilted axis generates coordinated canal and otolith information whereas during sustained rotation the coordinated canal information disappears. Head movements made during earth-orbit missions generate normal semicircular without the usually coupled change-in-orientation information from the otolithic sensors (and here it would be nice to have much better basic information than is available on the dynamics of the mechanical response of the otolithic sensors to linear accelerations in "microgravity"). Head movements (pitch or roll) involving the vertical canals (that on earth are typically accompanied by otolith information) are more provocative in microgravity than yaw movements (Lackner and Graybiel 1984). More than 66% of astronauts are afflicted with space motion sickness for several days until adaptation occurs (Reschke, 1990). Here we have sensory messages not clearly in conflict with one another but rather sensory messages in combinations that can't be immediately interpreted by the brain networks that generate sequences of motor reflexes that ordinarily improve the quality of motion control. From this perspective we prefer the term neural mismatch to sensory conflict in characterizing stimuli that lead to motion sickness.

EMPIRICAL PROCEDURE FOR NEURAL MISMATCH ASSESSMENT

The vestibular-system always operates as a silent partner to improve the efficiency of goal-directed voluntary body movements relative to the earth. Normally vestibular sensations do not achieve conscious awareness. For example, as the gaze is changed by head and body movement to see the source of a sound, the person is aware of the sound and then of the visual object and may be aware of pain from an arthritic joint but is unaware of the vestibular message. This property of the vestibular system can be used as a clue to the presence of neural mismatch. Vestibular sensations achieve conscious awareness in voluntary ambulatory movement only when they are 'disorderly' in relation to concomitant information from other senses that participate in the control of head and body movement relative to the earth. The dizziness that accompanies vestibular disorders is poorly described because the afflicted person is confused and disturbed by mixed signals from the various senses involved in the control of motion. Immediate confusion and disturbance from motion stimuli are therefore useful signs that neural mismatch is present either due to dysfunction within the motion sensing processes or due to unusual motion, beyond the range encountered in daily activities.

Awareness of motion and inaccuracy of motion perceptions cannot be used as indicators of the presence of sickness-provoking stimuli, because vestibular sensations usually achieve conscious awareness during passive motion. Accuracy of motion perceptions cannot serve as an indicator because perceptions may be wrong but not at all confusing. An example is the post rotational sensation of spinning following a gentle stop from low speed whole-body rotation in darkness. The spin perceived is inaccurate but not confusing, disturbing or nauseogenic because the misleading message from the semicircular canals encounters minimal conflicting information from other motion sensors and passive body movement minimizes demand from compensatory postural reactions. This example may seem ill-chosen in the light of the sickness provoked by the "sudden-stop test" (Graybiel and Lackner, 1980). The conditions for the sudden-stop test involve an angular impulse that generates a spin sensation of sustained magnitude far beyond any commonly encountered together with an angular impulse to the muscles and joints that would awaken spino-vestibular interplay (Wilson and M. Jones, 1979) despite body restraint in the rotation device. The sudden-stop test provokes sensations that are immediately confusing and disturbing.

Until vestibular neuroscience advances to a complete understanding of what is sensory conflict and what is functional sensory interaction, the best available empirical clue to neural mismatch is the presence of confusion and slight repugnance (disturbance) in the overall immediate perception.

CONCEPTUAL GUIDES FOR IDENTIFYING PROVOCATIVE STIMULI

Benson (1988/b) has provided a conceptual classification of provocative motion stimuli, based on the neural-mismatch theory of motion sickness. We propose another category of neural mismatch in the last column of Table I, inferred from the very different reactions to active and passive motion. The perceptual responses, oculomotor reflexes, trunk and limb reflexes during and after sustained active turning differ markedly from their counterparts during and after passive turning.

A confused and reversed perception follows active ambulatory turning that is attributable to feed-forward messages initiated by volitional motor commands and concomittant vestibular afferent messages (Guedry et al., 1978). Prostrotation trunk and limb torsional activity, present following active ambulatory turning (Correia et al., 1977), is less apparent when the subject is seated and the arms provide the torsional driving force (Guedry and Benson 1983). It is not apparent at all following passive turning, seated or standing. Thus the orientation perception and perceptual-motor reactions differ from their counterparts following passive turning which is dominated by the semicircular canals. Perceptual reactions during goal-directed voluntary whole-body motion differ from perceptual reactions following kinematically identical passive motion.

TABLE I: Identification of Motion Cue Mismatch Engendering Motion Sickness

Type	Visual(A)/ Vestibular(B) Mismatch	Canal(A)/ Otolith(B) Mismatch	Vestibular(A)/ Proprioceptor(B) Mismatch
Type 1 (A) and (B) simultaneously contradictory information	(i) Reading hand-held map or text in turbulent flight	(i) Cross-coupled (Coriolis) stimulation Head movement during rotation about another axis.	(i) Phase difference during low frequency vertical linear oscillation
	(ii) Inspection through binoculars of ground or aerial targets from moving aircraft	(ii) Head movement in abnormal force environment which may be stable (for example, hyper- or hypo-gravity) or fluctuating (during linear oscillation)	(ii) Aftereffect of sustained ambulatory turn
Type 2(i) (A) signals without expected (B) signal	(i) 'Simulator sickness'. Piloting of fixed base simulator with moving external visual display (VFA)	(i) 'Space sickness'. Head movement in weightless environment	(i) Sudden Stop Test
		(ii) Pressure (alternobaric) vertigo	(ii) Turning point zone during low frequency high peak velocity angular oscillation
Type 2(ii) (B) signals without expected (A) signal	(i) Looking inside aircraft when exposed to motion	(i) Low-frequency (<0.5 Hz) linear oscillation	(i) Null Class
		(ii) Rotation about non-vertical axis	

Adapted from Benson (1988b)

Here we propose that vestibular-induced motor response of the trunk and limbs come into opposition with motor responses engendered by preprogrammed feed forward messages during voluntary movement and by forces imposed directly on the muscles and joints by the motion itself.

This class of neural mismatch is difficult to study relative to the ease with which controlled changes in visual-vestibular interactions can be imposed; its importance remains to be determined. All of the examples in this column of Table I may be Type 1 or Type 2, and Type 2(ii) may be a null class for this form of neural mismatch. The sickness produced in the "Sudden-stop" test and the immediate disturbance that accompanies this very strong semicircular canal stimulus may be partially attributable to postural discord with vestibular adjustment demands from this exceptional stimulus. During very low frequency high peak velocity sinusoidal angular acceleration, some subjects report a paradoxical and disturbing sensation near the zero velocity cross-over of the stimulus--where the semicircular canal turning signal approaches and crosses zero signal. During this interval the Weber ratio (Stevens, 1951) for comparison of canal and proprioceptor signals may be optimal--canal signals approach zero while proprioceptor signals from torsional effects on the body approach maximum, although they are never strong in the seated subject. Similarly during low frequency vertical linear

oscillation, the perceived reversal point is phase advanced relative to the true point of reversal so that subjects perceive 'hitting bottom' before maximum force on the subject's 'bottom' occurs.

STOTT'S RULES

In an effort to provide simplifying principles that subsume the many particulars of motion stimuli that provoke sickness, Stott (1986) suggests some rules that the brain uses in evaluation of match/mismatch, based on the fact that the overall visual scene is usually stable and gravity doesn't change in direction or intensity (at least in the perceptual world of man).

> Visual-Vestibular rule: Angular or linear head motion in one direction must result in corresponding angular or linear motion of the overall visual surround to the same extent in opposite direction. Here adequate phase or planer matches must occur or else visual-vestibular sickness provoking mismatches are present.
>
> Canal-otolith rule: Rotation of the head, other than in the Earth-horizontal plane, must be accompanied by appropriate angular change in the direction of the linear acceleration due to gravity.
>
> Utricle-saccule rule: Any sustained linear acceleration is due to gravity, has an intensity of 1 g (9.81 m/s^2) and defines "downwards."

This latter rule does not overlook the fact that linear acceleration of the head in the earth-horizontal plane yields a resultant force vector that is tilted relative to gravity but rather assumes that the sum total of body linear accelerations including gravity, over any short period of natural voluntary locomotor activity, average to the direction of gravity and 1.0 g magnitude. One obvious deviation from this rule is running or walking in a circular path where the average centripetal acceleration yields a tilted average linear vector. However when this occurs the vestibular system assists in aligning the "average" vertical axis of the body with the average direction of resultant force vector, while the vertical dimensions of trees and buildings in visual space serve through visual/vestibular interaction to level the head, maintain clear vision and preserve the constancy of visual space.

Frequency-Effects and Stott's Utricle-Saccule Rule

The utricle-saccule rule, used to explain sickness induced by vertical linear acceleration, appeals to the fact that frequencies of linear oscillation that induce sickness are considerably below those normally encountered in locomotion activity so that for example the mean 1 g intensity rule would be violated on the high side for almost 2 s and then on the low side for almost 2 s in each cycle of a 0.25 Hz vertical linear oscillation. This interesting explanation relates to one of the more perplexing facts of motion sickness that must be dealt with by any comprehensive model of motion sickness. Motion sickness is dependent upon the frequency of motion stimuli, for some and perhaps all forms of sickness producing motion stimuli.

Data available suggest that 0.2 Hz is maximally provocative (Guignard and McCauley 1990, O'Hanlon and McCauley 1974) and that higher and lower frequencies are less provocative, at given peak vertical acceleration levels. The fact that frequencies above 0.5 Hz are less provocative fits neatly with the fact that the utricle and saccule are routinely stimulated at frequencies above 1.0 Hz during walking and running. However, if frequencies of vertical linear acceleration significantly lower than 0.2 Hz are in fact less provocative, then Stott's interesting 1 g explanation doesn't hold. Unfortunately, data on low frequencies of vertical oscillation is weak because low frequencies require very great linear displacement to reach the peak acceleration magnitudes evaluated at 0.2 Hz. Magnitude as well as frequency is important (Lawther and Griffin, 1986). Amplitudes required at very low frequencies exceed amplitudes that can be attained with existing motion devices.

Data from other forms of cyclic stimulation help but do not clear-up this issue. Cyclic stimuli that violate Stott's visual-vestibular interaction rule dramatically produce sickness at frequencies of .02 Hz, far below 0.2 Hz, but do not produce sickness at a frequency of 2 Hz (Guedry et al., 1982).

Whether or not cyclic vestibular interference with vision at 0.2 Hz would provoke more sickness is not known, but here, matching stimulus magnitudes is conceptually difficult. High peak angular velocities (120°/s) are required to produce visual blurring for head-fixed targets at 0.02 Hz whereas low peak velocity (20°/s) produces equivalent visual blurring at 2.5 Hz. With .02 Hz the peak acceleration was only 7.5 °/s² whereas with 2.5 Hz the peak acceleration was 314°/s². The investigators chose to match frequencies and peak velocities that induced equivalent degradation of visual performance.

Indications that flight simulators particularly conducive to simulator sickness have "high energy" of display motion centering around 0.2 Hz are interesting observations that require more data for analysis than is available from simulator studies. The Guedry, Benson, Moore (1982) experiments illustrate the need for analysis. The 0.02 Hz visual-vestibular interaction that induced sickness required the subjects to visually suppress vestibular nystagmus to obtain visual information from the head-fixed visual display. The cyclic waxing or waning of the VOR in each stimulus cycle is associated with a waning and waxing of difficulty in visual suppression, ranging from a maximum effort to suppress VOR of about ~70°/s in one direction to minimum effort to suppress a VOR of 0°/s and then to maximum effort to suppress a VOR of ~70°/s in the opposite direction. If cyclic variation in suppresion effort is the significant element, then the relevant stimulus frequency was 0.04 Hz, since suppression effort peaked twice in each cycle, whereas if direction of VOR suppression is relevant then 0.02 Hz was the relevant stimulus frequency.

However if the visual display used is simple, a set of 3 single digits changing ach second, then very little sickness occurs, whereas if the display is more complex, such as a matrix of numbers from which numbers must be retrieved when coordinates are given (Gilson et al., 1977, Guedry and Ambler 1973, Moore et al., 1977), then sickness incidence is high. Perhaps cyclic variation in vestibular interference with a pattern of voluntary gaze-control is the provocative attribute of this motion stimulus. When cyclic VOR is visually suppressed for even a short time, a VOR gain reduction (not attributable to arousal reduction) occurs. Recent neurophysiological data indicate cerebellar involvement in visual suppression of vestibular nystagmus and cerebellar 'circuitry' in the adaptive modification of the VOR. Moreover these same cerebellar components appear to be critical to provocation of motion sickness (Brizzee 1990, Crampton 1990/b, Wang and Chinn 1956).

Sustained off-vertical rotation is a form of sustained cyclic otolith stimulation that induces sickness and here the peak magnitude can be held constant. During whole body rotation about a rotation axis that is tilted relative to gravity, the otolithic membranes should be displaced in something approaching an orbital path about some central point in the sensory macula. The component of gravity directed in the mean shear plane of the otolithic membrane is the effective stimulus. Irrespective of rotation speed, the direction of this component is continuously changing. Rotation speed determines the frequency of this cyclic stimulus, 12 rpm being a 0.20 Hz stimulus. With this stimulus form it appears that about 0.25 Hz is maximally provocative whereas 0.10 Hz is not, and higher frequencies, e.g., .37 Hz, and above, are less provocative (Kokin et al., 1991, Miller and Graybiel 1973). The OVR data fall approximately in line with the vertical oscillation data.

Visual motion stimulation without whole-body movement may be useful in clearing up this perplexing question as to whether there is a maximally provocative frequency, irrespective of the type of motion stimulus. Perhaps cyclic otolith stimulation, as opposed to cyclic canal stimulation, is maximally provocative at 0.2 Hz. It is possible with visual stimuli to simulate whole-body motions that would stimulate the otolith system exclusively, or the semicircular canals exclusively or the canals and otoliths simultaneously. Simulated vertical linear oscillation at frequencies less than 0.2 Hz could be generated without the excursion limits that are imposed by devices that actually move the body. Some interesting information may be generated that would relate to whether or not there is in fact a common maximally provocative frequency across different forms of motion.

To explain frequency effects, one looks for something that might be tuned so as to have maximum gain at the most provocative frequency. The literature on frequency effects doesn't provide good rationale for deciding where to look. Referring to Figure 1, do we look in Stage 1, 2, or 3?

PERCPETUAL-MOTOR ADAPTATION TO UNUSUAL MOTION ENVIRONMENTS

Knowledge has been developed in recent years on mechanisms of adaptation to motion conditions that involve consistent mismatch among the orientation sensors (Berthoz and M. Jones, 1985). Human and animal subjects wearing optical devices that reverse, displace, or change the velocity of visual motion feedback during head and body movements (actively or passively generated) have been extensiveley investigated. These studies consistently show adaptive changes in the vestibulo-ocular reflex that serve to optimize stablization of retinal images during head motion. For example, VOR adjustment to optical right-left reversal requires a stimulus-response phase shift of almost 180 degrees. A phase-shift approaching 180 degrees developed in about 3 days in human subjects who wore reversing prisms continuously, even when tests of phase relations were made in the dark during passive motion (Melvill Jones,1985). Before phase shift was manifest, gain reduction was prominent.

Subjects living in an enclosed rotating room (Graybiel et al., 1960) show VOR gain reduction fairly early (Guedry, 1964). The confusing motion perceptions that accompany each head movement in a rotating room also diminish. After several hours of rotation exposure the gain of the VOR induced by head-tilt movements during room rotation is greatly reduced (Guedry et al., 1964; Guedry et al., 1962). And for several hours after rotation stopped, head movements produced confused perception, e.g., a roll-right head movement produced pitch-forward motion perception and postural instability. After this short exposure, change in the plane of the VOR was not apparent in electro-oculographic tracings. After three days (or longer) in the rotating room, for several hours after the room was stopped, a roll-right head movement produced a few beats of VOR nystagmus appropriate in direction for pitch forward perception of head and body movement (Guedry and Graybiel 1962, Guedry 1965). The effect that was perceptually evident after short exposure (2 hours), became evident in the VOR after longer exposure. A 90 degree change in plane of the VOR in response to voluntary headmovement can be seen for several hours after leaving the rotation room, when the exposure is of sufficient duration. The sequence of adaptive VOR changes in the optical reversal studies and the rotating room studies is first gain reduction and second "recoding" that shifts the phase or the plane of the VOR appropriately for the exceptional environment. The fact that only VOR gain reduction was reported during rotation in the rotating room studies is due to the fact that in the enclosed rotating room, the only VOR component that needed to be suppressed during x-axis roll was the y-axis component; the x-axis component was still needed during head roll movements in the rotating room. In other words, visual requirements for change did not demand a VOR change in the plane of the head-movement; rather visual requirements for change demanded suppression of the VOR in a plane 90 degree displaced from the head movement plane. Also in the adjustment sequence revealed by the post-rotation results, the perceptual plane was changed substantially before the VOR plane change was evident, at least with the measurement available at that time. The spatial orientation perceptual model may change before the control strategy initiates recoding of the VOR.

Along with adaptive changes in the reactions (VOR and perceptual) induced by head movements in these motion environments, changes in motion sickness symptoms also occur. Early in motion exposure, symptoms begin to appear and then increase (at different rates in different subjects) and with continuing exposure, symptoms diminish (again at different rates in different subjects). Thus it is reasonable to believe that initiating perceptual-motor adjustment induces motion sickness and that achievement of perceptual-motor adjustment serves to prevent motion sickness in the novel motion environment.

While changes in the sensorimotor, perceptual and sickness reactions parallel one another, they probably do not follow exactly the same course. Metrics to characterize each of these reactions lead to comparative uncertainty. Consider that VOR gain change did not proceed at the same rate as 'recoding changes' in the rotating room studies or in the optical left-right reversal studies and recovery rates occur in reverse order; recoding recovers faster than gain.

Individual reactions during the course of adaptation reveal individual differences in adaptive strategy. In a rotating room, one subject in the course of a series of prescribed head movements may stop, vomit and then continue on with head movements as though nothing exceptional had happened. Recovery is rapid. Just before emesis, the VOR often becomes disorganized and "unscorable" but it may return after emesis. Another subject, who may "tolerate" the head movement sequence much longer before emesis, finally vomits but is unable to continue for a long time. Recovery is slow. Some subjects try to resist emesis more than others, some recover faster than others and these are a

few of many differences between people that complicate efforts to model the mechanisms of motion sickness.

NEUROPHYSIOLOGICAL MECHANISMS OF PERCPETUAL MOTOR ADJUSTMENT

Most research aimed at evaluating the neurophysiological mechanisms of adaptive adjustment to unusual motion has focused on motion conditions that yield "neural mismatch" but very consistent neural mismatch. Optical distortion of visual feedback always yields the same visual-vestibular mismatch for particular head movements. Pitch or roll head movements from upright posture in a rotating room always yield the same canal/otolith mismatch irrespective of heading in the room (Guedry, 1965/b). Many recent studies have been described in Berthoz and Melvill Jones (1985) and a convenient succinct summary is provided by Stott (1990).

> The Marr-Albus model of cerebellar function suggests that the cerebellum learns to perform motor skills both for voluntary movements and for reflex maintenance of posture and balance. Essential to this learning capacity are the climbing fiber inputs from the inferior olive. The model proposes that the synaptic connections of parallel fibers to Purkinje cells are modifiable in their transmission efficiency and that this modification is brought about by conjunctive activation of a parallel fiber and a climbing fiber converging on the same Purkinje cell. This mechanism has received experimental confirmation in a series of experiments by Ito (reviewed in Ito) which substantiate the concept that the climbing fiber inputs constitute the instruction signals that reorganize the relationship between mossy fiber inputs and Purkinje cell output.
>
> Purkinje cell axons project to oculomotor control areas within the vestibular nuclei. Whether the plasticity within the cerebellum provides the sole basis for gain modulation of the vestibulo-ocular reflex is not certain. Miles and Lisberger, from studies in the monkey, argue for the gain modifiable elements to be located in the brain stem.
>
> The ability in experimental studies to control visual and vestibular motion stimuli and to make precise measurements of eye movement responses are important factors that have made the study of vestibulo-ocular mechanisms such a profitable area of research. The multiplicity and variability of the physiological correlates of motion sickness render the underlying neural mechanisms of this response more difficult to elucidate. Nonetheless, it seems likely that neurophysiological mechanisms similar to those involved in vestibulo-ocular gain plasticity are involved in adaption to nauseo-genic stimuli. The further elucidation of the mechanisms that enable recognition of spatial sensory patterns and bring about adaptive changes within this system may in turn yield principles that underlie the higher mental capacities of learning and memory.
>
> As stated by Berthoz and Melville Jones, "...the adaptive phenomenon rather than being a mere curiosity of brain function ... constitutes a fundamental property of the nervous system, responsible for active matching both between related sub-systems within the CNS and between them and their daily encounter with the external physical world."

A CONCEPTUAL MODEL

Characteristics of VOR alteration may provide clues to vestibular sensorimotor changes relevant to motion-sickness modelling. VOR gain reduction occurs naturally in darkness during any

passive sustained oscillation or repeated step function or any other repeated velocity waveform (Collins 1963/1964, Collins and Guedry 1962). VOR gain can be immediately reinstated by any of a variety of alerting tasks and can be manipulated, up or down, by imagined visual reference (Baloh 1984, Barr et al., 1976, Melvill Jones et al., 1984, Jell et al., 1988), but without manipulation, VOR diminishes. This everpresent tendency toward VOR gain reduction during passive stimulation may signify an early and ever-ready level of adaptive change that provides some defense against motion sickness in more variable (and perhaps less stressful) motion conditions than are encountered in rotating rooms or while wearing right-left reversing spectacles, variable conditions in which adaptive rearrangement of sensorimotor response would not be functionally appropriate.

ASSUMPTIONS

From this gain reduction propensity and the early gain reduction in the vision reversal and rotating room studies, we propose that gain reduction is an early adaptive change to sustained unusual motion that may be significant in highly variable motion environments. Following are additional assumptions underlying our conceptual model:

From the presence of optokinetic nystagmus, optokinetic after-nystagmus, arthrokinetic nystagmus (Brandt et al., 1977) and evidence for arthrokinetic after-nystagmus (Guedry and Benson 1983), we propose that the standard operating gain of the VOR is less than 1.0 during voluntary active motion which is the raison d'etre for the VOR. Voluntary initiation of motion increases VOR gain briefly. Then an optokinetic component, a lesser arthrokinetic component and possibly a weak audiokinetic component contribute to a gain of 1.0 for the overall response in sustained voluntary motion. This theoretical perspective concerning the brain's standard (desired) VOR gain may be important (if true) in motion sickness. In short, the vestibular preferred operating gain is less than 1.0 and how much gain is contributed by the other sources of gain augmentation may depend upon preprogrammed feed-forward messages related to movement objectives. From this perspective OKN, AKN, OKAN and AKAN are manifestations of processes whose presence is meant to achieve gain in the kinds of motions involved in natural voluntary movement. Responses generated by sustained off-vertical rotation or sustained active body turning by the limbs are manifestations of auxillary contributions to VOR gain in natural movement. The walk-in-place nystagmus of Bles, et al. (1984, 1979), may play a role in gaze stabilization during sustained ambulatory turns in circular paths.

From the absence of awareness of vestibular sensation in natural voluntary movement, we infer that optimal adjustment to novel motion environments provides control of motion in which there is minimal awareness of vestibular sensations.

Based on the belief that control of whole-body motion has been critical to survival of species and the fact that unexpected motions generate alarm reactions very early in life (Smith and Smith 1962, Guedry and Correia 1978) we assume that a demand (or drive) to maintain control of whole-body motion is present in passive as well as active motion conditions.

Finally we assume that during initial exposure to a novel motion environment, a spatial model of the motion environment forms. To permit optimal overall commerce with the environment, control of motion must proceed toward automaticity wherein the task of motion control is not a distraction. During either voluntary movement in a novel environment or passive exposure to novel motion, motion control objectives and strategies are part of the adaptive process. These are fundamental assumptions underlying the conceptual model illustrated in Figure 6.

In motion environments, it is important to distinguish between 1) voluntary motion relative to a motion platform that is moving (or perceived to be moving [visual vection]) relative to the earth, 2) motion relative to the earth by voluntary control of a motion platform that is moving or perceived to be moving (simulation) relative to the earth, and 3) completely passive whole-body motion with the head and body fixed to a moving motion platform. The different situations involve different relations between intention to move and motion experienced. However, herein we assume the presence of a constant internal demand to maintain effective control of orientation-change relative to the earth, irrespective of motion conditions. Thus, a detected mismatch in any motion environment begets arousal and corrective actions.

The first stage in the model comprises the motion stimulus which elicits immediate perceptual-

motor reactions which are compared with preprogrammed sets of perceptual-motor paradigms that in turn depend upon the spatial model, motion control objectives, and previous experience. Even during completely passive motion stimulation postural and motion control objectives are in operation due to the imperative to maintain control of control of body posture and motion relative to the earth. Figure 6 presents this conceptual model.

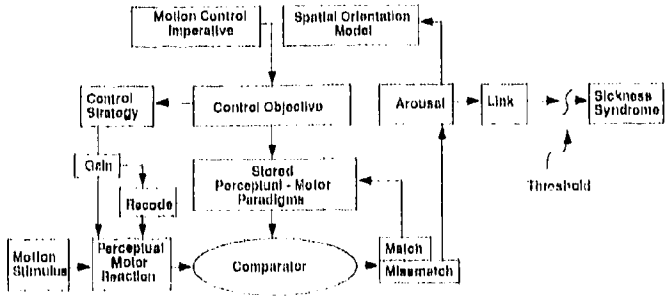

Figure 6. Conceptual Model of the mechanisms of adaptation to unusual motion and of the relationship of adaption to the onset of sickness.

When match is detected, the preprogrammed perceptual-motor (PM) paradigms are reinforced, the store remains the same, and the selection rate of appropriate sets of Perceptual-motor paradigms is improved. The spatial model and control objectives determine which paradigm sets are in immediate access for comparison. When mismatch is detected, arousal mechanisms operate through the spatial model and control strategy, calling for response change first through gain-control and then if necessary through gain-control coupled with sensory-motor recoding. Mismatch begins to alter the spatial model so that new sets of perceptual-motor sequences can be stored for comparison with subsequent reactions. But mismatch engenders arousal and continued arousal commences the build-up of the neurochemical link to the sickness syndrome. As the matches improve, the model and neural store develop permitting more rapid selection of preprogrammed perceptual-motor engrams that enable the automaticity required for efficient control of motion without arousal. As long as mismatch induces arousal, the neurochemical build-up for sickness-onset continues; big arousal contributing to faster sickness onset.

Adaptation Through Gain Control

The literature on adaptation to consistent mismatches induced optically or in rotating rooms provides examples of motion conditions in which recoding of sensory motor responses serves to repair highly consistent mismatch. Most of the effort to elucidate underlying physiological mechanisms is centered on optically induced neural mismatches. Our emphasis on gain change was partially due to our perception that different mechanisms may be involved in adaptation to novel motions that are not completely consistent in the neural mismatches they generate. Now we must provide some examples of situations in which gain control would appear (intuitively) appropriate for the job.

Ship motions are fairly variable despite the natural frequencies of the roll and pitch of the ship and they certainly induce seasickness. Individuals moving about on ships increase the complexities and variability of the motions they experience. When on deck there is some motion-control advantage for stabilizing the eyes relative to the earth, considering the horizon, distant ships and even slow moving clouds. As indicated earlier, the vestibular system stabilizes the eyes relative to the earth automatically with some optokinetic and arthrokinetic assistance, in a normal environment. On deck, high VOR gain is needed but arthrokinetic gain for oculomotor control may need some reduction because the leg, arm and trunk motions are appropriate for the ship's deck but not for the horizontal plane of the earth. Even the ambient visual information is playing tricks because some very large visual fields (the deck and superstructures of the ship) are moving and tilting relative to the earth's horizon but are the relevant visual frames of reference for control of motion of the body. Laboratory visual/ vestibular interaction studies suggest that earth-reference motion cues in ambient vision will

improve focal vision for shipboard objects when in accord with vestibular information; discord yields degradation of focal vision (Guedry et al., 1979). When the individual enters the ship's compartments, it would be nice for VOR gain to go down while optokinetic gain goes up; VOR effort to stabilize vision relative to the earth is not helpful inside a ship's compartment. Adjustment of the various contributors to oculomotor gain appears (intuitively) advantageous for at least part of the adaptation to some forms of complex motions. Similarly, vestibulomotor adjustment considering the limbs, neck and torso must require gain adjustment as well as directional adjustment. That gain control can be highly adaptive is nicely illustrated by a recent study in which, through adaptive conditioning, gain was increased or decreased, depending upon whether subjects gazed upward or downward (Shelhamer et al., 1991).

Expressed in terms of the conceptual model, adaptation to ship motion would involve development of a spatial model that when coupled with the immediate control objectives and strategy would permit quick selection of perceptual-motor paradigms including relative gains for VOR, OKN and AKN for the interior of the ship. Vestibular sensing of pitch and roll of the ship would be necessary for automaticity of limb and trunk control--some recoding of directional function would accompany dynamic changes in gain control.

DEVELOPMENTAL STRUCTURING OF ADAPTIVE CAPACITY

According to the model, past experience with interaction among motion sensors during whole-body motion plays a role in the state of adaptation to those interactions and hence influences whether or not particular interactions engender sickness. With this in mind, it is appropriate to consider the range of experience that occurs in everyday life with varied interactions between a) the semicircular canals and the otolithic sensors, b) visual and vestibular sensors and c) the vestibular and proprioceptor sensors. We include vestibular sensors in each of the interactions because of the central role in motion sickness of the vestibular system.

The coupling between semicircular canals and otolithic sensors remains very tight throughout life. Any particular voluntary head motion initiated from any particular position relative to gravity always generates the same combination of stimuli to these two different sensors. A particular head-motion relative to the body can produce different canal/otolith interactions depending upon the state of whole-body motion relative to the earth but a particular head motion relative to the earth always generates the same combination of semicircular canal and otolith stimuli.

Here we might ask how the coupling of canal/otolith interaction for particular motions of the head relative to the earth could ever be altered in the course of development. One mechanism is vestibular dysfunction. Vestibular dysfunction, for whatever reason, could yield unaccustomed sensory patterns. Sustained or repeated dysfunction could increase the range of experience with sensory-neural mismatches.

Another curious source in the development of man lies in early childhood experiences. The sustained ambulatory turning that most children enjoy generates a semicircular canal signal that produces the Purkinje effect when the head is tilted, an effect that is disturbing later in life. This childhood activity repeatedly induces incoordination that eventually ends with a fall, which the child finds amusing. Functional value of such activity is a matter of speculation. Is the child increasing the range of adaptive processes that will serve him later in life, should vestibular dysfunction arise? Certainly reinstatement of ability to control whole-body motion has survival value, if vestibular dysfunction should arise in later life. The passive motion experienced before birth and the vigorous tossing and turning that is generated by parents may also be relevant.

In contrast to the tight otolith/canal coupling in the course of natural life are the more varied combinations of interactions between the vestibular and visual systems and between the vestibular and proprioceptor systems. Coupling between visual and vestibular systems is varied in everyday life. Particular head movements relative to the earth can be generated with eyes open or closed. The eyes may be fixed on a point in space as the head moves so that retinal images of the surround is retinally stabilized, or the eyes may pursue a moving target during head movement so that the target image is fixed on the retina but background images move on the retina. During linear or slightly curvilinear head motion, the direction of retinal image sweep depends upon direction of gaze; fixation of far targets produces high speed sweep of near objects. In modern man, optical lenses demand visual-

vestibular adjustments. Everyday life demands central processes that handle a variety of visual-vestibular interactions, suggesting availabilty of CNS processes for handling varying combinations of visual-vestibular interaction. This may facilitate adaptation to visual-vestibular mismatches when they are imposed in modern transportation systems. Alteration of VOR gain begins in a matter of minutes, even during passive motion.

Similarly the coupling between vestibular and somatosensory inputs from weightbearing surfaces and from muscles and joints is variable in everyday life. The same forward pitch trunk movement can be made with the head turned to the right, left, up or down relative to the body. Turning movements about a body axis or in a circular path can generate similar vestibular messages produced by very different body movements. During walking, head and neck and vestibular systems are involved in gaze stabilization and their roles change for near and far targets (Reschke et al., 1991).

From this perspective we speculate that visual-vestibular mismatches can be very disturbing but that adaptive change should be relatively fast. Vestibular-proprioceptive mismatches can be disturbing but perceptual-motor adjustment to mismatch may also be relatively fast. This is a neglected area of study. Finally, intra-labyrinthine mismatches are more provocative and disturbing than the others and perhaps most resistant to perceptual-motor adaptive resolution of mismatch.

THE NEUROCHEMICAL LINK

Neural mismatch, recognized somehow somewhere, initiates the onset of sickness. A hypothetical sequence is a chemical agent which enters the cerebrospinal fluid in the third ventricle (Crampton 1990, Crampton and Daunton 1983), is tranpsorted to the fourth ventricle where it reacts with a chemically receptive zone in the Area Postrema (Brizzee and Neal 1954, Wang and Chinn 1954) on the surface of the floor of the fourth ventricle. This reaction triggers a nearby vomiting center (Wang and Borison 1950). This sequence has recently been challenged (ablation of the Area Postrema did not prevent vomiting (Borison 1986, Wilpizeski 1986) and a discrete vomiting center (Miller 1988, Miller and Wilson 1983) may not exist), but some kind of neurochemical link near these centers is likely (Brizzee, 1990).

In considering this link, we must account for the fact that sickness onset may be very sudden (emesis occurs in some individuals after 1 or 2 cross-coupled stimuli-- within say 30 seconds--and nausea onset can be almost immediate) or sickness may be very gradual in onset requiring several hours for build-up with some forms of stimulation (Homick et al., 1984). Analogy to titration procedures in a quantitative chemical analysis seems appropriate--the chemical endpoint is approached very gradually and slowly with very small droplets but the endpoint can be overshot with one big drop. Recovery may be very fast or very slow. An alternative to the titration analogy, expressed as the model (Oman, 1990) illustrated in Figure 7, proposes parallel slow and fast pathways to account for extremes in onset rates.

The fast and slow dynamic elements in the model may relate either to "orientation-emetic linkage mechanisms or the dynamic properties of the emetic centers themselves." Oman suggests that extremely fast nausea and vomiting is neurally mediated, whereas slower onset dynamics is suggestive of humoral mediation. He suggests physiological mechanisms that might be involved:

> The dynamic operators may approximate the action of some humoral diffusion or active transport process, or could be the intrinsic dynamics exhibited by a network of vomiting center neurons to direct neural or humoral conflict signal stimulation. For example, vestibular conflict neurons might work in synergistic pairs, somewhat as vestibular afferent neurons do, and project to neurons (perhaps in area postrema, AP) associated with nausea perception and vomiting. If the synapses were excitatory and had the character that a burst of conflict neuron activity produced a prolonged (e.g., 1 min) depolarization, "leaky integrator" dynamics would result. Carpenter et al. (1990) have described a class of AP neurons that respond qualitatively in this fashion to a variety of directly applied neurotransmitter agents. They attribute the long lasting depolarization of these neurons to a second messenger mechanism. Slow path dynamics might be associated with a

> potentiation of the fast path synaptic pathway by a third agent circulating from the hypothalamus - pituitary or diffusing through the ventricles. This slow path is presumably characterized by much slower response times. (Oman, 1990 p 300)

On the 'output side,' the mechanism for fast emesis is in place. Vomiting is a complex reflex that in surgery can be immediately elicited by pulling the gut or by lightly scraping the surface of the floor of the fourth ventricle near the Area Postrema. However physical traction on the gut seems unlikely to be part of the natural eliciting mechanism (Wang and Chinn 1956b).

Considering now the initiation of the linking process, is it the recognition of neural mismatch or the beginning of the sensorimotor adaptation process that triggers the sequence of events that culminate in nausea and vomiting? Triesman (1977) proposed that toxins induce neural mismatch among motion sensors and that vomiting provoked by neural mismatch serves to protect species. Moreover, the unpleasant experience associated with the source of the toxin would provide beneficial avoidance conditioning. Thus motion sickness is a biological accident attributable to the fact that unusual motions generate neural mismatches that simulate mismatches that toxins generate (cf Money 1990).

Money (Money and Cheung, 1983, Money 1990) provided inferential support for this idea when they found that labyrinthectomy in dogs reduced emesis from several toxins. Based on this idea, it is detection of neural mismatch in the motion control system and not the beginning of the adaptation process that triggers the onset of the motion sickness syndrome.

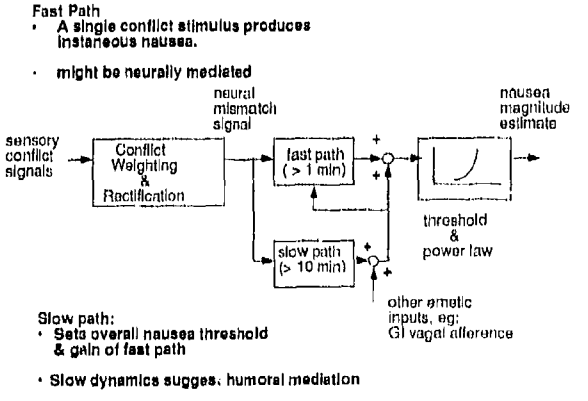

Figure 7. Schematic diagram of model for dynamics of nausea symptoms. Adapted from Oman (1990).

Onset of sickness may be slow because of a slow passive process or in some situations individuals may actively 'titrate' exposure. For example, in earth orbit or in rotating rooms, the individual may restrict head and body movements that are the sickness-inducing stimuli. In such situations, adaptation may develop at a rate sufficient to prevent the onset of serious symptoms of motion sickness. In studies of exposure to continuing passive motion, emesis incidence levels (Guignard and McCauley, 1990) for passive vertical oscillation reached 80% for the provocative frequencies around 0.2 Hz in about 2 hours and only about 30% for a higher frequency. Some individuals who did not reach emesis in this study undoubtedly experienced nausea, nevertheless lengthy exposure to some motions is required before awareness of symptoms occurs in many individuals.

THE MOTION SICKNESS SYNDROME

The motion sickness syndrome consists of autonomic nervous system reactions associated with motion stress, somatic nervous system reactions involved in emesis and a range of complex mental reactions including fatigue, drowsiness, poor motivation for work, and depression.

AUTONOMIC-STRESS REACTIONS

It is hard to imagine any perceptual-motor function more crucial to survival of the species than the control of body orientation and motion relative to the earth. From this perspective, any threat to this function must elicit alarm reactions. The perceptual-motor mismatches involved in initiating motion sickness are signals of threat to the control of motion relative to the earth. However, the magnitude of the stress would be related to the spatial model of the motion environment and the control objective. Stress and control strategies would differ between a disoriented pilot suddenly perceiving imminent impact with the earth and a ship's passenger whose perceptual-motor mismatches are detected but not perceived as threats to survival--except perhaps subconsciously.

"The general neuroendocrine response to stressful motion and sensory-rearrangements include elevated levels of adrenocorticotropin (ACTH) cortisol, prolactin, growth hormone, antidiuretic hormone or vasopressin (ADH or AVP, respectively), thyroid hormone, epinephrine, and norepinephrine" (Harm 1990, p158). However, individuals considered susceptible to motion sickness do not necessarily manifest greater autonomic activity than individuals more resistant to motion sickness. From a review of findings, Harm (1990 p165) found support for the idea that sympathetic nervous system activation provides some protection against motion sickness and that shifts in the balance of sympathetic and parasympathetic function underlies motion sickness symptomatology.

EMESIS AND THE SOMATIC NERVOUS SYSTEM

Nausea with its visceral concomittants and the somatic act of vomiting are distinct functions of the emetic control mechanism, (Borison, 1986 p4). Vomiting is usually preceded by nausea from autonomic actions but can occur without nausea. Curious characteristics of motion sickness are 1) the quick relief from nausea that emesis often brings as though whatever the neurophysiological accumulative process that accounts for feeling sick were suddenly set back to a 'better' state and; 2) the very slow recovery that may occur following brief exposure to a highly provocative stimulus. Nausea, stomach uneasiness, fatigue, headache, and drowsiness may persist for 12 or more hours following 60 seconds off-vertical rotation at 15 rpm, or following 2 head movements while rotating.

Rapid relief may partially be attributable to the fact that many of the autonomic concomittants of nausea such as salivation, gastric stasis, respiratory and heart rate changes are also part of the organized reflexive chain of events that comprises the somatic act of vomiting. The reflexive demand by the somatic vomiting sequence for change in these physiological concomittants of autonomically generated nausea may account for the sudden change in perceived well-being after vomiting. A sequential model (Davis et al., 1986 p73) as opposed to a "parallel" activation model has been proposed to explain the effector sequence that makes up the all or nothing vomiting mechanism--mouth opening, salivation, gastric relaxation, respiratory control, abdominal muscle contraction...controlled by the somatic nervous system.

Ideas relating vomiting to survival of the species center on levels of defense against toxins. Specific smells and tastes associated with nausea produce aversion and an initial level of defense. It is hypothesized that the first levels of defense are so effective in the rat that the rat has survived through avoidance of toxins even though the rat cannot vomit.

In considering changes in the physiological measures associated with motion sickness it is necessary to keep in mind not only that the vomiting sequence and the autonomic aspects are distinct functions of the emetic control mechanism but also the fact that changes in orientation and states of motion produce changes in respiration and heart rate associated with changes in energy demands of the body. The vestibular system is an efficient detector of these demands during natural movement. The paramedian reticular formation appears to be a site for the integration of cardiovascular and vestibular signals (Yates and Yamagata 1990). Vestibular function is apparently important in adjusting heart rate during Hyper-Gz exposure (Matsunami and Satake 1991). Frequent and sustained exercise, i.e. high levels of aerobic fitness, seems to increase susceptibility to motion sickness (Banta et al., 1987, Cheung et al., 1990) possibly related to neuro-chemical changes associated with exceptional exercise regimens.

Does willingness to vomit play a part in the magnitude of symptoms experienced in motion sickness? While the act of vomiting is reflexive and not under voluntary control, willingness to vomit plays a role in how different individuals adjust to unusual motion environments. Possibly quickness to vomit and hence feel better is partially idiosyncratic. Individuals who reflexively vomit with little effort and with little disruption of performance may be willing to vomit whereas those whose act of vomiting is a major event are less willing. Whatever the cause, willingness to vomit plays a role in the strategies individuals adopt in adjusting to a motion environment. Here we refer to strategies that are more on an intellectual level than the partially subconscious strategies that bring on the perceptual-motor adjustment in our conceptual model. Individuals in rotating rooms or in orbital flight can elect to avoid head movements that are not absolutely necessary, maintaining a sustained malaise that may never result in vomiting. Others may opt for a more active approach that may result in more emesis but perhaps more rapid adaptation.

COMPLEX SIGNS AND SYMPTOMS OF MOTION SICKNESS

The range of signs and symptoms that comprise the motion sickness syndrome, as described by Benson (1988/b), is presented here along with Figure 8, which depicts the neurophysiology of motion sickness.

> The cardinal symptom of motion sickness is nausea; the cardinal signs are vomiting, pallor and sweating. Other responses are frequently reported, but in general these occur more variably. Typically, the development of motion sickness follows an orderly sequence, the time scale being determined primarily by the intensity of the stimulus and the susceptibility of the individual (Money 1970, Reason and Brand, 1975). The earliest symptom is, commonly, the unfamiliar sensation of epigastric discomfort, best described as 'stomach awareness'. Should the provocative motion continue, well-being usually deteriorates quite quickly with the appearance of nausea of increasing severity. Concomitantly, circumoral or facial pallor may be observed, and the individual begins to sweat; this cold sweat is usually confined to those areas of skin where thermal sweating rather than emotive sweating occurs. With the rapid exacerbation of symptoms, the so-called 'avalanche phenomenon', there may be increased salivation, feelings of bodily warmth, a lightness of the head and, not infrequently, quite severe depression and apathy. By this stage, vomiting is not usually long delayed, though there are some individuals who remain severely nauseated for long periods and do not obtain the relief, albeit transitory, that many report following emesis.
>
> If exposure to the motion continues nausea typically increases in intensity and culminates in vomiting or retching. In the more susceptible individual this cyclical pattern, with waxing and waning symptoms and recurrent vomiting, may last for several days. Those so afflicted are commonly severely anorexic, depressed and apathetic, incapapable of carrying out allotted duties, or caring for the safety of themselves or others. Their disability is also compounded by dehydration and disturbances of electrolyte balance brought about by the repeated vomiting. Apart from the characteristic features of motion sickness--pallor, sweating, nausea and vomiting--other signs and symptoms are frequently though more variably reported. In the early stages, increased salivation, belching and flatulence are commonly associated with the development of nausea. Hyperventilation is occasionally observed, while an alteration of respiratory rhythm by sighing and yawning not infrequently precedes the 'avalanche phenomenon'. Headache is another variable prodromal symptom, usually frontal in distribution, though complaints of tightness around the forehead or of a 'buzzing in the head' are not uncommon.
>
> Drowsiness is an important, yet often ignored, symptom commonly

associated with exposure to unfamiliar motion, even if not necessarily an integral part of the motion sickness syndrome. Typically, feelings of lethargy and somnolence persist for many hours after withdrawal of the provocative motion stimulus and nausea has abated. However, in certain circumstances a desire to sleep may be the only symptom evoked by exposure to motion, especially when the intensity of the stimulus is such that adaptation occurs without significant malaise (Graybiel and Knepton, 1976). The soporific effect of a repetitive motion stimulus on infants has long been recognized. It may be that the drowsiness observed in the adult when exposed to appropriate motion is a manifestation of the same mechanism, though it must be acknowledged that the somnolence in an individual who has suffered overt motion sickness is frequently of abnormal intensity and persistence. (Benson p.319)

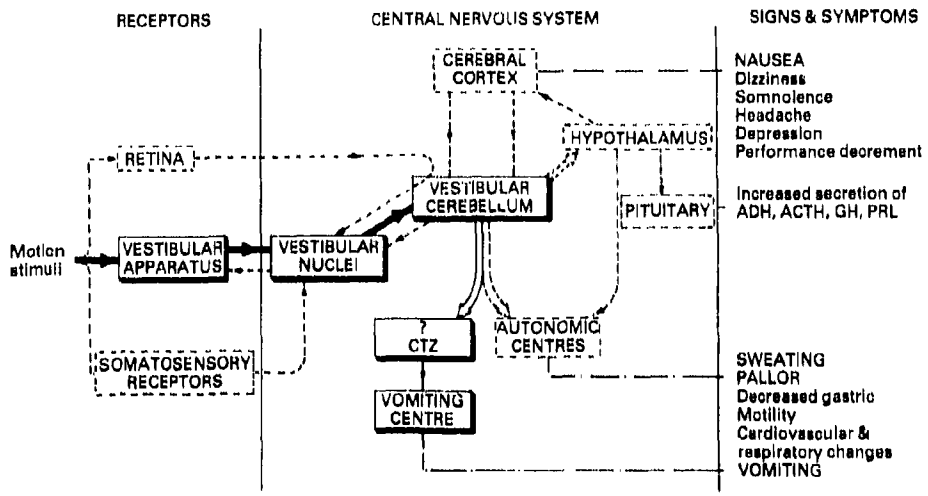

Figure 8. Neural structures in motion sickness. CTZ = Chemoreceptive trigger zone. (From Benson 1988b)

MOTION SICKNESS AND SPATIAL DISORIENTATION

The combination of sensorimotor systems involved in maintenance of spatial orientation awareness and the systems involved in bringing about the onset of motion sickness are the same. Motion sickness may increase the probability of failure to maintain adequate orientation awareness as a result of the distraction of coping with emesis or the depression and malaise associated with motion sickness. Enthusiasm and confidence of the student pilot in learning to control aerobatic maneuvers can be seriously degraded by the drowsiness, malaise and discouragement that are part of the motion sickness syndrome. If these symptoms are not accompanied by vomiting, the student and instructor alike may not appreciate that the student's 'poor attitude' is due to airsickness.

Many flight environments and especially aerobatics generate many mismatches that cloud the perception of the maneuver and that also tend to induce motion sickness. Trainees adjust to these mismatches and in so doing overcome airsickness. Indeed the motion perceptions of actual flight are so much a part of controlled flight that the experienced pilot disturbed and made sick by flying simulated maneuvers in a flight simulator. His perceptual-motor store for control of motion in the aircraft is violated by the feedback from his control actions in the simulator. The beginning flight student, without comparable stored perceptual-motor patterns, is undisturbed by flying the simulator (Guedry 1988, Kennedy et al., 1990, NRC conference 1990). From this perspective, motion sickness and spatial orientation reactions of pilots are related.

Many flight situations that induce pilot disorientation involve stimuli that are minimally nauseogenic. A pilot in formation flight and keeping station on a companion aircraft may be in a coordinated bank-and-turn but believe the aircraft to be in level flight. The turn sensation from the semicircular canals is absent because time since commencement of turn has allowed the cupula to recover to its null position and alignment of the resultant force vector with the 'vertical' axis of the aircraft elicits a zero-roll signal from the otolithic receptors. Thus the pilot can be dangerously disoriented but unaware of his disorientation and also undisturbed by nauseogenic sensory mismatches. In fact this is referred to as Type I disorientation (Benson 1988/a) which is responsible in the fixed-wing military aviation communities for a greater loss of aircraft and pilots than disorientation types in which pilots are aware of disorientation.

Type II disorientation, in which the pilot becomes aware of his disorientation, may involve mismatched and nauseogenic sensory input. Type II disorientation accounts for a higher incidence of aircraft and pilot losses in rotary wing aircraft than does Type I (Vyrnwy-Jones, 1988). However, incidence of airsickness is lower in rotary wing than in fixed wing communities. The frequency of occurrence of nauseogenic stimuli plays a role in the probability of sickness and pilots would obviously avoid repetitive Type II disorientation.

While the physiological mechanisms of spatial disorientation have a commonality with those of airsickness, practical solutions for the two problems and even research into those solutions involve divergent approaches.

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PHYSICAL CHARACTERISTICS OF STIMULI PROVOKING MOTION SICKNESS

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SUMMARY

The physical characteristics of motion stimuli responsible for motion sickness are reviewed in two parts. The provocative stimuli are categorised and their nauseogenic properties discussed qualitatively in terms of the sensory conflict theory of motion sickness. Quantitative information available from experimental studies with specific types of motion is then summarised. The motions of the body considered in this review include translational oscillation, swing motions, rotation about a vertical axis, rotation about an off-vertical axis, rotational oscillation and cross-coupled (i.e. Coriolis) stimulation. Conditions producing visually-induced motion sickness are also summarised.

1. INTRODUCTION

Motion sickness is studied primarily because of the problems it causes to people travelling in a wide variety of environments (see Table 1). The motions associated with sickness in the various environments are different and often complex and so it is not immediately apparent which motions are primarily responsible for sickness. Few studies conducted in real environments have made useful measurements of both the motion and the consequent sickness so as to establish cause-effect relationships.

Table 1 Some examples of environments, activities and devices which can cause symptoms of motion sickness (from Griffin, 1990).

Boats	Camel rides
Ships	Elephant rides
Submarines	
Hydrofoils	Simulators
Hovercraft	
Swimming	Fairground devices
Fixed-wing aircraft	Cinerama
Helicopters	Inverting/distorting spectacles
Spacecraft	Microfiche readers
Cars	Rotation about off-vertical axis
Coaches	Coriolis stimulation
Buses	Low-frequency translational oscillation
Trains	
Tanks	

Experimental studies have shown that relatively simple motions can cause sickness in laboratory conditions. These motions have been used to study physiological and psychological correlates with individual susceptibility to motion sickness. They have also been used to test alternative anti-motion sickness drugs. Some laboratory investigations have quantified a relation between the type of motion exposure and the consequent motion sickness.

Evidence of the causes of motion sickness comes from laboratory experimentation, field study, analysis of theoretical models and philosophical considerations. In the following sections the discussion is restricted to data from laboratory studies.

1.1 The causes of motion sickness

When suffering from motion sickness there is a tendency for people to attribute their condition to factors other than the motion. Food, temperature, smells, clothing and many other non-motion factors are blamed for causing sickness. Motion sickness in others is easily attributed to some psychological weakness.

The motion stimuli responsible for sickness cannot be easily identified by an exposed person. In transport environments there is motion in many directions and the traveller is unable to attribute the sickness to motion in any one direction. Some very gentle movements are capable of producing sickness - to succumb to their influence may seem a sign of weakness. For some environments, scientific study has yet to identify the motion principally responsible for sickness.

The loose relationships established between motion and motion sickness allow the false identification of cures. There are many unproven remedies offered for motion sickness.

1.2 Signs and Symptoms of Motion Sickness

A consideration of the physical stimuli causing motion sickness also involves a consideration of means of quantifying the effects. Vomiting is the most obvious sign of motion sickness but it is not the only effect; vomiting may be neither the most sensitive nor the most important consequence of motion sickness. Where the incidence of motion sickness is low, the recording of some less dramatic effect which may be present in greater quantity will be a more sensitive indicator. The various signs and symptoms of motion sickness may differ in their dependence on the characteristics of motion and other factors. However, studies at sea reported by Kanda *et al* (1977) and Lawther and Griffin (1987) suggest mild and moderate symptoms of sickness may be predicted from the relation between vomiting incidence and ship motion magnitude.

Investigators of motion sickness have often chosen to use rating scales which give some weight to effects other than vomiting. Terms such as 'nausea' and 'sickness' have different meanings in different studies so it is attractive to standardise on the rating scale. One of the most used scales is that presented by Graybiel *et al* (1968) in which the acute effects are given points (Table 2a) which are then aggregated and used to arrive at a level of severity of motion sickness on a five point scale from "slight malaise" to "frank sickness" (see Table 2b). This scale has been used to categorise the symptoms of motion sickness in some of the experiments summarised in Sections 3 to 9 below.

Table 2a Diagnostic categorisation of different levels of severity of acute motion sickness as defined by Graybiel *et al*, 1968. (Points accumulated from the signs and symptoms in this table are used to determine the relevant degree of motion sickness severity given by Table 2b).

Category	Pathognomonic 16 points	Major 8 points	Minor 4 points	Minimal 2 points	Additional Qualifying Symptoms 1 point
Nausea syndrome	Vomiting or retching	Nausea II, III	Nausea I	Epigastric discomfort	Epigastric awareness
Skin colour	-	Pallor III	Pallor II	Pallor I	Flushing
Cold sweating	-	III	II	I	-
Increased salivation	-	III	II	I	-
Drowsiness	-	III	II	I	-
Pain	-	-	-	-	Headache
Central nervous system	-	-	-	-	Dizziness; Eyes closed ≥II, Eyes open III

Table 2b Levels of severity of acute motion sickness as defined by Graybiel *et al*, 1968. (Points accumulated from Table 2a are used to determine the degree of motion sickness severity shown in this table).

Points	Level of severity	Sickness Category
≥ 16	Frank sickness	S
8 to 15	Severe Malaise	M III
5 to 7	Moderate Malaise A	M IIA
3 to 4	Moderate Malaise B	M IIB
1 to 2	Slight Malaise	M I

2. THEORETICAL CATEGORISATION OF PROVOCATIVE STIMULI

2.1 The role of theories

A complete theory of motion sickness would give a method of measuring both the cause and the effect and indicate how they are related. Various motion sickness theories have been proposed but none is yet capable of providing quantitative predictions of the degree of motion sickness to be expected from a range of different motion stimuli.

For a few types of stimuli, presented in restricted circumstances, it has been possible to determine empirical relationships between the motion and its effects (see Sections 3 to 9 below). These relationships may contribute to a general model of motion sickness but they are not, in themselves, motion sickness theories.

General theories of motion sickness are currently qualitative rather than quantitative. They provide a rationalisation of existing information but make no precise statements of what degree of motion sickness will arise in specific circumstances. The 'sensory conflict theory' described below provides a useful qualitative introduction to the characteristics of nauseogenic stimuli.

2.2 The sensory conflict theory

Several theories have been based on the idea that motion sickness arises from a conflict between the information received via two or more sensory systems. However, since the meaning of sensory information is being continually re-learnt, we vary our responses to stimuli. There is not conflict between the stimulation of the sensory systems, only conflict between the interpretation placed upon this stimulation. Any measure of conflict between the physical stimuli will be fixed and so such conflict will not explain habituation to nauseogenic stimuli or the after-effects of exposure to such stimuli. The extraordinary adaptive capabilities of the human body are involved in both causing and overcoming motion sickness.

A conflict between signals received from different senses is more easily considered a conflict with what is 'expected' than a conflict with what is 'correct'. The combination of signals which is expected will be largely that which has been learnt from previous experiences. The concept of sensory conflict has therefore been extended into a theory of sensory rearrangement (see Reason, 1970, 1978).

The sensory rearrangement theory of motion sickness states that: "*all situations which provoke motion sickness are characterised by a condition of sensory rearrangement in which the motion signals transmitted by the eyes, the vestibular system and the non-vestibular proprioceptors are at variance either with one another or with what is expected from previous experience*". Reason and Brand (1975) suggest that the conflict may be conveniently and sufficiently considered in two categories: inter-modality (between the eyes and the vestibular receptors) and intra-modality (between the semi-circular canals and otoliths within the vestibular system). For both categories it is possible to identify three types of situation in which conflict may occur (see Table 3).

Table 3 Types and categories of sensory conflict

Type of conflict	Category of conflict	
	Visual - Vestibular	Canal - Otolith
Type I	Visual and vestibular systems simultaneously signal different (i.e. contradictory or uncorrelated) information	Canals and otoliths simultaneously signal different (i.e. contradictory or uncorrelated) information
Type IIa	Visual system signals in the absence of an expected vestibular signal	Canals signal in the absence of an expected otolith signal
Type IIb	Vestibular system signals in the absence of an expected visual signal	Otoliths signal in the absence of an expected canal signal

Motion environments can involve more than one conflict: there is generally more difficulty in deciding where the various environments should be entered than whether they can be entered within this categorisation. Table 4 illustrates the division advocated by Benson (1984). The following explanations are derived from Griffin (1990).

Table 4 Type of motion cue mismatch produced by various provocative stimuli. (Adapted from Benson, 1984)

	Category of motion cue mismatch	
	Visual (A)/ Vestibular (B)	**Canal (A)/ Otolith (B)**
TYPE I A and B simultaneously give contradictory or uncorrelated information	Watching waves from a ship Use of binoculars in a moving vehicle Making head movements when vision is distorted by optical device 'Pseudo-Coriolis' stimulation	Making head movements whilst rotating (Coriolis or cross-coupled stimulation) Making head movements in an abnormal acceleration environment which may be constant (e.g. hyper- or hypo-gravity) or fluctuating (e.g. linear oscillation) Space sickness Vestibular disorders (e.g. Ménière's disease, acute labyrinthitis, trauma labyrinthectomy)
TYPE IIa A signals in absence of expected B signals	Cinerama sickness Simulator sickness 'Haunted swing' Circular vection	Positional alcohol nystagmus Caloric stimulation of semi-circular canals Vestibular disorders (e.g. pressure vertigo, cupulolithiasis)
Type IIb B signals in absence of expected A signals	Looking inside a moving vehicle without external visual reference (e.g. below deck in boat) Reading in a moving vehicle	Low-frequency (<0.5 Hz) translational oscillation Rotating linear acceleration vector (e.g. 'barbecue-spit' rotation, rotation about an off-vertical axis)

2.2.1 Visual-vestibular conflict

Type I

Watching nearby waves from a ship results in some vestibular and some somatosensory perception of the motion of the ship relative to the gravitational field of the Earth, while seeing only the uncorrelated motion of the waves relative to the ship. This situation may be neatly placed as a Type I conflict. It might then be assumed that the problem will be reduced by watching the horizon rather than watching the sea. It should not be concluded that this is the only cause of sickness at sea when inside a vessel with no outside view there is a Type IIb visual-vestibular conflict and there may also be a Type I or Type IIb canal-otolith conflict.

Various optical devices cause magnification or other distortion of the visual field so that natural head movements result in unexpected movements of the visual scene and a visual-vestibular conflict. The 'pseudo-Coriolis' effect arises when the head is moved while under the illusory sensation of body rotation induced by present or past movement of a surrounding visual field, such as a rotating patterned drum (Dichgans and Brandt, 1973; see Section 10.1).

Type IIa

Situations in which there is visual movement but no movement of the body can be highly nauseogenic (e.g. some films, fixed-base simulators and the haunted swing). Although some meaning to the motion may increase the provocation, this is not essential. Sickness can also be induced by illusions of self motion (circularvection) produced by the movement of patterned stimuli in the visual field (see Section 10.2).

Type IIb

Many forms of 'travel sickness' may be at least partially attributed to the vestibular perception of motion while not being able to see the motion. The sensory conflict for children in the rear seats of cars partially arises from them only being able to see their motion relative to the rear of the front seat rather than their true motion relative to the outside (i.e. distant) visual field. Reading a map in a moving vehicle may also be nauseogenic for this reason - though such visual tasks may be more provocative than observing a fixed point in a vehicle.

2.2.3 Canal-otolith conflict

Type I

The cross-coupled, or Coriolis, stimulation which occurs when the head is rotated about an axis other than the axis of rotation of the body is said to be a Type I canal-otolith conflict: it is usually assumed that the otoliths tend to signal the correct information while the canals provide a false signal - see Section 8 below.

A different conflict occurs if the head is moved during exposure to abnormal translational acceleration, such as high G aircraft manoeuvres, zero G in spacecraft, acceleration and deceleration in cars and oscillations aboard ships. In these cases a tilt of the head will not give rise to the changes in otolith signal which normally occur when stationary on Earth during exposure to 1G.

Space sickness has sometimes been considered to be a Type IIa canal-otolith conflict since head rotation will produce an appropriate canal signal without the normal changes in the signal from the otoliths which occur when they tilt within the gravitational field of the Earth (Reason and Brand, 1975). However, voluntary head motions which are associated with space sickness are thought to produce accelerations sufficient to stimulate the otoliths and so it may be more appropriate to identify space sickness as a Type I canal-otolith conflict (see Benson, 1977).

Some disorders of the vestibular system caused by disease give rise to symptoms of motion sickness due to unexpected contributions of signals from canals and otoliths, and so may also be classed as a Type I conflict. One example is Ménière's disease in which both balance and hearing can be affected.

Type IIa

A signal from the semi-circular canals in the absence of a signal from the otoliths can arise from the convection currents set up during caloric stimulation of the canals by irrigation of the outer ear with warm or cool water. Similarly, a difference in the specific gravity of the endolymph and cupulae which may arise from the consumption of alcohol, or 'heavy water', results in the cupulae being deflected by gravity. Both of these situations can produce nystagmus, dizziness and nausea with the greatest stimulation when recumbent with one ear down. In this position there is a strong feeling of not being moved from unchanging otolith signals and somatic senses but maximal stimulation of the, normally, horizontal semi-circular canals.

Type IIb

Constant speed rotation of the body about an off-vertical axis results in a change in otolithic stimulation with no change in the signal from the canals (see Section 6). This is said to be a Type IIb canal-otolith conflict. With rotation about an Earth-horizontal axis at about 10 rpm on a 'barbecue spit', nausea many be produced in a few minutes. A similar conflict occurs if the body is oscillated in translation at low frequencies, such as on a swing, in an aircraft, or aboard a ship (see Sections 3 and 4).

2.2.3 Significance of the theory

When providing a qualitative explanation of the causes of motion sickness, the sensory conflict theory is often sufficient: all known causes of sickness are accommodated by the theory and it suggests some useful preventive measures. Its greatest value appears to be the identification of the relevant sensory systems, their interactions, and the foundation that this provides for the concept of sensory rearrangement.

The sensory conflict theory does not indicate how sensory conflict can be measured and it therefore provides no quantitative information. It cannot be used to identify which of several possible conflicts in an environment is most significant. The sensory conflict theory might be used to anticipate whether some combination of stimuli is likely to be nauseogenic but it cannot be used to predict the extent of any symptoms, or how they depend on the magnitude of motion, the type of motion or the duration of motion.

A development of the sensory conflict theory, employing control theory and based on the means whereby the visual world appears to be stable despite movements of the head and eyes (i.e. 'efference copy'), has been elaborated by Oman (1982). He proposed that the site of visual perception receives a 'copy' of the efferent signals sent to the eyes and contains a model of how the sensory pathways operate. If the model fails to account for the received sensory information it eventually adapts but, initially, 'reports' conflict and produces signs and symptoms of motion sickness. A separate system model of motion sickness was evolved by Riedel (1980) and is also based on sensory conflict.

2.3 Other theories

Some motion sickness theories have incriminated somatosensory proprioception of joint or visceral movement while others have blamed the 'sloshing of the blood' or the fluctuating mechanical pressure of blood or stomach (e.g. Wollaston, 1809). Such suggestions are generally discounted by evidence that an impaired vestibular system imparts immunity to sickness and the tempting corollary that the vestibular system is 'responsible' for sickness. 'Overstimulation theories' attributed sickness to excessive stimulation of the vestibular system and orientated the discussion towards whether the otoliths or the semi-circular canals were responsible.

2.3.1 Otolith tilt reinterpretation theories

A 'tilt-translation reinterpretation' hypothesis (Young *et al*, 1984) and an 'otolith tilt-translation reinterpretation' hypothesis (Parker *et al*, 1985) have been advanced in the context of space sickness. The absence of a gravitational field in space means that

otolithic signals only arise from translational head movements - unlike on earth, where the most common cause is assumed to be the roll or pitch of the head through the gravitational field of the Earth. Roll and pitch head motions in microgravity will give signals from the semicircular canals, and relative movements of the perceived visual scene, without the expected signals from the otoliths. Astronauts have commonly reported that, early in a flight, pitch motions of the head provoke symptoms of sickness. Immediately postflight, astronauts who have adapted to microgravity exhibit decreased postural stability with their eyes closed (Young *et al*, 1984) and decreased ocular counterrolling during tilt (von Baumgarten *et al*, 1984). Both observations are consistent with adaptation to microgravity resulting in a failure to interpret roll motions of the head in a gravitational field as rotation. It has also been observed that immediately following spaceflight some astronauts perceive roll motion as horizontal translation and that roll motion induces more horizontal eye movements (Parker *et al*, 1985). A 'prophylactic adaptation training' procedure to encourage the appropriate microgravity relationships between otolithic and visual signals prior to spaceflight has been proposed by these authors. These hypotheses may be considered as lending further support to, or refining, the all-embracing sensory conflict theory.

2.3.2 Reflex-response theory of motion sickness

It has been suggested that the causation of motion sickness might be partially explained in terms of inappropriate (i.e. unnecessary, unsynchronised, delayed, or opposing) reflex responses to movements (Griffin, 1990). Reflex responses normally arise from the interpretation of signals from the various sensory systems capable of detecting motion. The standard interpretation of these signals will be incorrect in some situations and new reflex responses will need to be developed. Motion sickness is assumed to arise from the 'conflict' between inappropriate reflex responses. The theory is therefore centred on *response conflict* rather than *sensory conflict*. It is suggested that while sensory conflict cannot be measured it may be possible to measure indicators of response conflict. Nevertheless, the suggestion appears to be broadly compatible with both the sensory conflict theory and the otolith tilt reinterpretation theories. The reflex-response theory may contribute to understanding the time-dependant nature of the development and recovery from motion sickness. It may also suggest some measurable parameters of both the motion and human response. This theory does not discount a role for somatosensory perception in motion sickness.

2.4 Application of motion sickness theories

The sensory conflict theory cannot be tested by experiment since, while it provides a framework for what has been observed, it makes no precise predictions. Since the theory cannot be disproved, it may be assumed to be either broadly correct, or irrelevant. The theory does not make quantitative predictions. Indeed, it implies that such predictions cannot be made. The effects of changes to the quantity or quality of motion stimuli cannot, therefore, be inferred from the sensory conflict theory.

Any explanation of motion sickness in terms of the sensory conflict theory is therefore qualitative and does not provide an explanation of the physical causes of sickness. Although some later theories attempt to move the theory forward they are also insufficient to make confident predictions based on physical measurements.

In the following sections some of the available quantitative data are presented. The occurrence of motion sickness with the individual stimuli should be accommodated by the sensory conflict theory, and all other theories. However, currently, only the experimental data show quantitative relationships between the physical causes of sickness and the various signs and symptoms.

3. TRANSLATIONAL OSCILLATION

3.1 Vertical oscillation

Studies have shown that low frequency vertical oscillation can cause sickness in both man and animals.

3.1.1 Human studies

Morton *et al* (1947) report exposing five subjects to up and down motion in a lift over a distance of about 5 metres with an acceleration of approximately 2.7 ms^{-2} and a maximum speed of 4 ms^{-1}. The exact characteristics of the motion were not stated but it was reported that four out of the five subjects became sick in a period of 10 to 30 minutes.

The first major series of systematic laboratory studies of the production of motion sickness in man by vertical oscillation were conducted with several hundred Naval cadets and are sometimes referred to as the 'Wesleyan University studies'. An elevator, or lift, capable of 5.5 metres of displacement (peak to peak) was used to impart 20 minute exposures to alternating periods of constant acceleration and constant velocity so that the resulting displacement waveforms were, very approximately, sinusoidal. The first study investigated four different frequencies (0.22, 0.27, 0.37 and 0.53 Hz) and produced the conclusion that the higher frequencies resulted in less sickness (Alexander *et al*, 1945a). The second study showed that increasing the magnitude of the motion (at about 0.37 Hz) increased the incidence of sickness (Alexander *et al*, 1945b). The third study used similar frequencies to the first study but changes to the periods at constant acceleration allowed the generation of different magnitudes of motion. The data again showed greatest sickness with lowest frequencies. However, comparison of the results with those from the earlier studies resulted in the conclusion that moderate magnitudes of motion produced the most motion sickness and that the highest magnitudes produced least sickness (Alexander *et al*, 1945c).

The fourth study investigated the effect of unsymmetrical waveforms (Alexander *et al*, 1945d). A later study produced further data with the same frequencies (Alexander *et al*, 1947).

The above results from the Wesleyan University studies have been reanalysed so as to express the vomiting incidence as a function of r.m.s. acceleration for each frequency (see Figure 1 and Lawther and Griffin, 1987). While there is scatter in the results it is apparent that vertical acceleration at 0.53 Hz produced less vomiting than the same acceleration at 0.37 Hz, and both frequencies produced less vomiting than acceleration at 0.27 Hz. Other publications in the series report the absence of a reliable effect of time of day on sickness using the elevator, a correlation between sickness on the elevator and responses to a questionnaire history of motion sickness, an attempt to find effects of motion sickness on task performance and show the absence of a significant effect of temperature on motion sickness in the elevator. Studies of the efficacy of anti-motion sickness drugs were also undertaken.

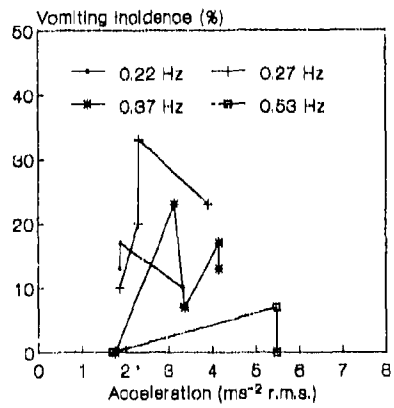

Figure 1 Variation in vomiting incidence with acceleration magnitude for four frequencies of distorted sinusoidal vertical motion. (Data from Alexander et al, 1947).

There has been only one other major series of laboratory investigations of the relation between physical characteristics of vertical motion and motion sickness in humans. The investigations were undertaken by Human Factors Research Inc using a 2.4 metre square closed cabin supported by an hydraulic motion system capable of 6.1 metres of vertical displacement, ± 15 degrees of roll and ± 15 degrees of pitch (the axes rotation were approximately 0.4 metres below the floor of the cabin). The first study exposed groups of 20 to 33 subjects to each of 14 experimental conditions involving various magnitudes of vertical sinusoidal motion at 0.083, 0.167, 0.333 and 0.500 Hz (O'Hanlon and McCauley, 1974). Seated subjects sat with heads on a headrest during exposures of up to 2 hours with no external view. Vomiting incidence ranged from 0% to 60% with higher magnitudes of acceleration being required to generate sickness at the higher frequencies; at each frequency the vomiting incidence increased with increasing acceleration magnitude. The frequency of maximum sensitivity to motion sickness was found to be 0.167 Hz. The authors proposed a formula for calculating motion sickness incidence (MSI) over 2 hours based on the integral of a log normal function of acceleration. The mean value of the function specified the acceleration required at a particular frequency to generate 50% vomiting (see Section 3.3.1 below).

McCauley *et al* (1976) extended the initial studies to frequencies above 0.5 Hz, investigated response to pitch and roll motion and examined habituation effects. Studies with 0.5, 0.6 and 0.7 Hz showed that motion sickness incidence continued to decrease as the

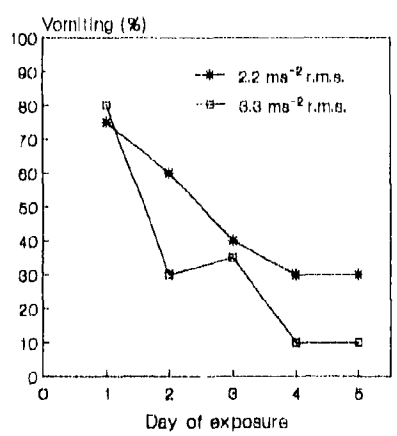

Figure 2 Habituation in two groups of subjects exposed on five occasions. One group exposed to 0.25 Hz at 2.2 ms^{-2} r.m.s. and the other group exposed to 0.25 Hz at 3.3 ms^{-2} r.m.s. (Data from McCauley et al., 1976).

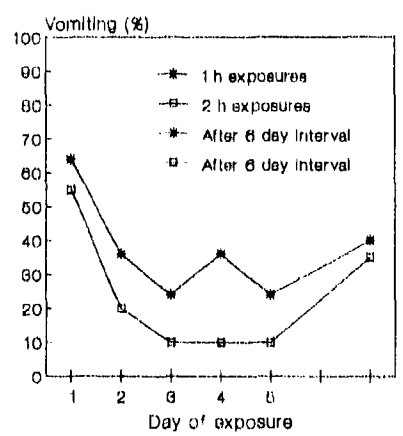

Figure 3 Habituation in two groups of subjects exposed to 0.25 Hz vertical sinusoidal motion at 3.3 ms^{-2} r.m.s. on 6 occasions. (Vomiting incidence shown after 1 h of motion exposure. Data from McCauley et al., 1976).

frequency was increased if the acceleration magnitude was unchanged. Only 1 person (i.e. 4%) became sick with 0.7 Hz vertical motion at 5.55 ms^{-2} r.m.s. With roll or pitch motion (at 0.115, 0.230 or 0.345 Hz with magnitudes of 5.5 to 33.3 deg s^{-2} r.m.s.) added to a vertical motion of 0.25 Hz at 1.1 ms^{-2} r.m.s. the incidence of sickness was not significantly different from sickness with the vertical motion alone. No subject vomited with roll motion alone (33.3 deg s^{-2} at 0.345 Hz) but two subjects vomited with this magnitude of pitch. The authors infer that the results support the conclusion that vertical motion is the cause of seasickness and imply that head motion during vertical oscillation may not contribute to sickness.

Three experimental studies of habituation are reported by McCauley *et al* (1976). The first experiment involved exposing 20 male subjects on five occasions to 0.25 Hz vertical motion at 2.2 ms^{-2} r.m.s. The subjects had all previously vomited when exposed to this motion. The incidence of motion sickness fell during the five experimental sessions from 75% to 30%. The second and third experiments with slightly different exposures involved small groups of males and females and showed a similar habituation effect but, after a period of 7 days without motion, susceptibility increased - suggesting that this interval was sufficient for subjects to lose some protection from motion sickness. Figure 2 shows that the rate of habituation was greater in experiment 2 (with 3.3 ms^{-2} r.m.s.) than in experiment 1 (with 2.2 ms^{-2} r.m.s.) - the authors suggested that this may be because habituation is greater when the stimulus is more nauseogenic. Figure 3 shows that the rate of habituation was greater in the first hour of experiment 2 (2 hour exposures) than in experiment 3 (1 hour exposures). The retention was also greater in the group exposed to 2 hours of motion per day. Again, the more nauseogenic exposure resulted in greater habituation.

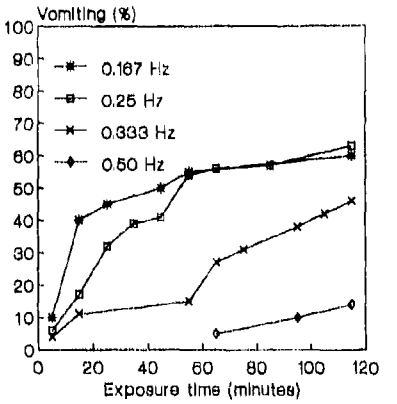

Figure 4 Increase in the numbers of persons to have vomited with increasing duration of exposure to four frequencies of vertical sinusoidal motion. (Data from McCauley et al, 1976).

Figure 4 illustrates the rise in the incidence of motion sickness with increasing duration of exposure to four frequencies of motion as reported by McCauley *et al* (1976). They report that the cumulative motion sickness incidence as a function of exposure time can be expressed in terms of a log normal distribution. An equation is offered to express motion sickness as a function of frequency, acceleration and exposure time (see Section 3.3.1).

The effect of oscillation frequency on motion sickness is shown in Figure 5 for 2 hour exposures to various magnitudes of motion. These data show a clear decrease in the vomiting incidence produced by vertical sinusoidal acceleration with increasing frequency from 0.25 to 0.7 Hz. Figure 6 shows that at frequencies between 0.167 and 0.60 Hz the increases in vomiting incidence with increases in the magnitude of the motion were nearly

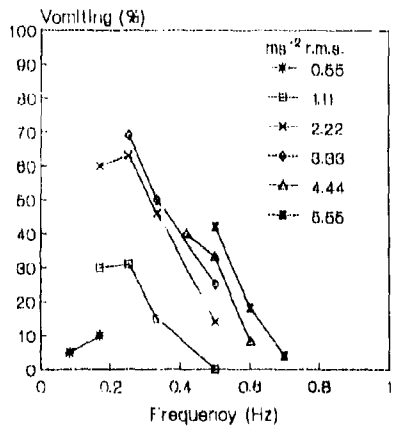

Figure 5 Variation in vomiting incidence with the frequency of vertical sinusoidal motion for 2 hour exposures to various magnitudes of motion. (Data from McCauley et al, 1976).

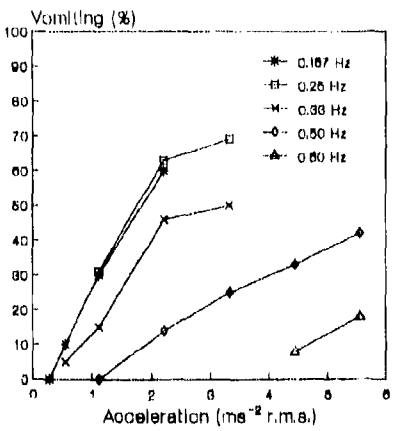

Figure 6 Variation in vomiting incidence with the magnitude of vertical sinusoidal motion for 2 hour exposures to various frequencies of motion. (Data from McCauley et al, 1976).

linear. The manner in which vomiting incidence varied with both the magnitude and the frequency of vertical sinusoidal motion is shown in three-dimensional form in Figure 7.

Guignard and McCauley (1982) investigated the effect of adding harmonics (at 0.33 or 0.50 Hz) to a fundamental frequency of 0.17 Hz vertical sinusoidal oscillation. The authors report no statistically significant difference in the incidence of vomiting produced by the five conditions studied. The motion sickness incidence varied from 50 to 78% in the five conditions and are reasonably consistent with predictions of 40 to 58% using the motion sickness dose value procedure defined in Section 3.3.3.

While studies with artificial stimuli have involved sinusoidal and pseudo-sinusoidal stimuli, several investigations have been performed with reproductions of recorded motions from vessels. For example, Malone (1981) and Anderson *et al* (1984) summarise results obtained with reproductions of motions predicted for a surface effect ship travelling at various speeds.

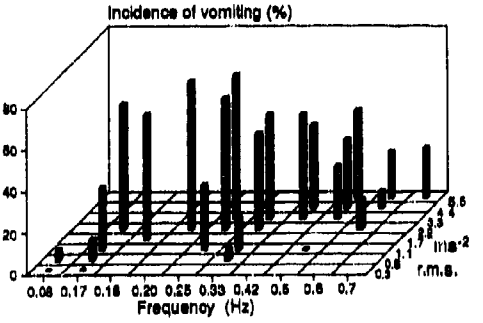

Figure 7 Variation in vomiting incidence with the frequency and the magnitude of vertical sinusoidal motion for 2 hour exposures. (Figure derived from Griffin, 1990).

3.1.2 Animal studies

Sjöberg (1931) made dogs motion sick using vertical oscillation generated by a seesaw arrangement, an elevator and a crane. Studies were also undertaken with human subjects (see Sjöberg, 1968). After trying a four-pole swing and a complex turntable giving continuous rotation and pitch oscillation, de Wit (1957) found that vertical oscillation of dogs in a cage suspended by a spring (0.33 Hz, 2.5 metre excursion, 4 ms^{-2} r.m.s.) was more effective in causing sickness in dogs. Exposures of 20 minutes were sufficient to cause sickness.

Using a means of oscillating cats vertically, McCabe and Gillingham (1964) were able to cause sickness in all animals with an approximately sinusoidal acceleration of about 4 ms^{-2} r.m.s. (frequency unknown). There was no apparent difference in sickness between those free to move, those who rested their heads on the floor and those whose heads were fixed by a 'tooth bar'.

Suri *et al* (1973) reported that 0.65 Hz vertical sinusoidal oscillation at 4.3 ms^{-2} r.m.s. produced sickness in cats: 22% vomited within 20 minutes. Borison and Borison (1986) produced sickness in cats with 3.5 ms^{-2} r.m.s. vertical oscillation at about 0.5 Hz. Twenty minutes vertical sinusoidal 0.6 Hz oscillation at 3 ms^{-2} r.m.s. has also been shown to produce vomiting in cats (Fox *et al*, 1987). These authors found that this frequency of oscillation with a displacement of 0.5 metres (2.5 ms^{-2} r.m.s.) also caused vomiting.

Using vertical sinusoidal oscillation in the range 0.17 to 0.33 Hz, with accelerations in the range 0.2 to 1.3 ms^{-2} r.m.s., Wilpizeski *et al* (1987a) found squirrel monkeys resistant to motion sickness. Of 11 monkeys highly susceptible to rotation about a vertical axis, only 1 vomited when exposed to vertical oscillation. The others were exposed to 2 hours per day for up to 10 days without signs of sickness. Wilpizeski *et al* (1987b) report that vertical sinusoidal oscillation at 0.2 Hz through a displacement of 2.08 metres (i.e. 1.2 ms^{-2} r.m.s.) produced sickness in only few squirrel monkeys within two hours.

The house musk shrew appears to be less susceptible to motion sickness with vertical oscillation than horizontal oscillation (Kaji *et al*, 1990). Fifteen minute exposures to frequencies between 0.25 and 2.0 Hz with a displacement of 40 mm resulted in little or no vomiting at 0.25, 0.5 and 2 Hz, but significant problems with 1 Hz vertical oscillation. Nevertheless, the overall effect was less than with 5 minute exposures to similar magnitudes of horizontal oscillation (see Section 3.2).

3.2 Horizontal oscillation

Any substantial studies of the extent to which horizontal oscillation of humans causes motion sickness have yet to be published. It has been reported that the house musk shrew (Suncus murinus) can be made to vomit by 5 minute exposures to horizontal oscillation of 25 mm displacement at 0.5, 1.0, 2.0 and 3.0 Hz (Ueno *et al*, 1988). About 20% of animals vomited at 0.5 Hz while 65% to 80% vomited at the three higher frequencies. With 1 Hz oscillation, a similar percentage vomited when the oscillation displacement was reduced to 10 mm but all animals vomited when the displacement was increased to 40 mm. There was some habituation to motion with repeated exposures and some drugs were found to reduce motion sickness in the animals. Other studies showed that with a displacement of 40 mm and a frequency of 1 Hz the house musk shrew was similarly affected by fore-and-aft motion, lateral motion and a circular motion (formed from the summation of the other motions with a 90 degree phase shift). The animal was more susceptible to horizontal oscillation than vertical oscillation (Kaji *et al*, 1990).

3.3 Standards for evaluating translational oscillation

All current standards for evaluating motion with respect to motion sickness are restricted to the assessment of vertical oscillation occurring in the z-axis of the body (i.e. in the seat-to-head or foot-to-head axis). Since translational oscillation in other axes, continuous rotation (with or without head movements), movements of the visual scene etc. can cause motion sickness it should not be assumed that the use of these standards is a sufficient means of predicting motion sickness in all environments. It is not clear which environments can be assessed with these standards but they are mainly applied to boats and ships.

3.3.1 McCauley et al studies

From the results of laboratory studies of motion sickness caused by vertical oscillation, a series of formulae for predicting motion sickness incidence (MSI) were proposed by O'Hanlon and McCauley (1974), McCauley and Kennedy (1976) and McCauley *et al* (1976). The method applies to motion in the frequency range 0.08 to 0.63 Hz with maximum sensitivity to acceleration at about 0.16 Hz. The assumption that MSI will vary with acceleration and with time in ogival form (i.e. a cumulative normal distribution) resulted in somewhat complex mathematical operations. Motion sickness incidence (MSI), expressed as a percentage, is assumed to be the product of a term representing the influence of motion magnitude and frequency, P_A, and a term expressing the effect of motion duration, P_T:

$$MSI = 100\ P_A\ P_T$$

The term P_A is calculated from a term, z_A, quantifying the effect of magnitude and frequency and a term describing the form of a cumulative normal distribution (in practice this may be obtained from statistical tables). The effect of magnitude and frequency was determined from a curve describing the acceleration required to produce vomiting at various frequencies in 50% of persons during two hour exposures:

$$z_A = 2.13 \log_{10} a - 9.28 \log_{10} f - 5.81 (\log_{10} f)^2 + 1.85$$

Where a is the r.m.s. acceleration in g, f is the frequency in Hz. The term P_T is calculated similarly to give a value for z_T:

$$z_T = 2 \log_{10} t + 1.13 z_A - 2.90$$

where t is the exposure time in minutes.

Values of P_A and P_T are obtained by consulting a table of the normal deviate z at the values of z_A and z_T respectively. For example, with a 60 min. exposure to an acceleration of 2.1 ms^{-2} r.m.s. (i.e. 0.21 g r.m.s.) vertical sinusoidal motion at 0.25 Hz, $z_A = 0.19$ and $z_T = 0.87$ so $P_A = 0.57$ and $P_T = 0.81$ giving a predicted motion sickness incidence of 46%.

3.3.2 International Standard 2631 Part 3 (1985)

Part 3 of this standard suggests the magnitudes of vertical oscillation in the range 0.1 to 0.63 Hz expected to produce a 10% incidence of sickness in sitting or standing fit young men over 30 min, 2 h and, tentatively, an 8 h exposure. The magnitudes and durations are in an inverse-square relationship so that the magnitudes for 2 hours are double those for 30 min. The magnitudes required for 10% vomiting in 30 minutes, 2 hours and 8 hours are shown in Figure 8. Sensitivity to acceleration is greatest from 0.1 to 0.315 Hz but falls at higher frequencies so that acceleration magnitudes required to produce sickness at 0.63 Hz are 3.15 times greater than those required to produce sickness at 0.315 Hz and at lower frequencies. The contents of this standard were first published as Addendum 2 to ISO 2631 in 1982.

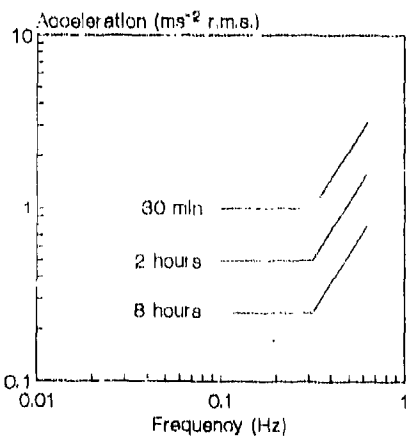

Figure 8 Magnitudes of vertical z-axis oscillation expected to cause 10% incidence of vomiting in exposures of 30 minutes, 2 hours and, tentatively, 8 hours according to International Standard 2631 Part 3 (International Organization for Standardization, 1985).

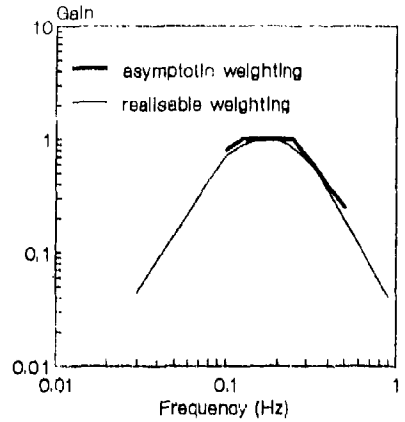

Figure 9 Frequency weighting W_f as defined in British Standard 6841 (1987). (Graph shows straight line 'asymptotic approximations' to the illustrated realisable weighting defined by the standard for use in instrumentation).

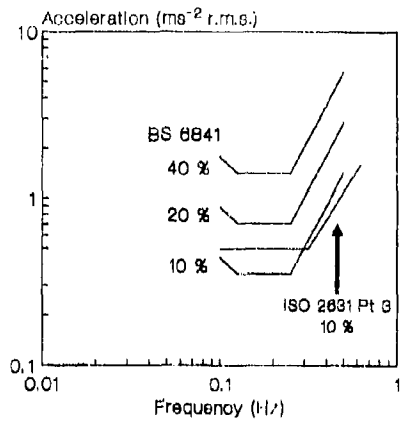

Figure 10 Vertical z-axis oscillation expected to cause 10%, 20% and 40% incidence of vomiting during 2 h exposures according to British Standard 6841. Incidence will double if magnitude is doubled or exposure duration is increased by a factor of four.

3.3.3 British Standard 6841 (1987)

British Standard 6841 (1987) defines a 'motion sickness dose value', MSDV, based on a frequency weighting (i.e. a filter) and a time dependency. The realizable frequency weighting, W_f, can be represented by an asymptotic approximation having an attenuation which varies with frequency at +6, 0, and -12 dB per octave. Table 5 lists the attenuations of this asymptotic approximation and Figure 9 compares the asymptotic and realisable frequency weightings. The standard fully defines the realisable weighting for implementation by analogue or digital filters. The greatest sensitivity to acceleration is in the range 0.125 to 0.25 Hz, with a rapid reduction in sensitivity at higher frequencies. The exposure duration, t (seconds), and the frequency weighted r.m.s. acceleration, a_{rms} (ms^{-2} r.m.s.), may be used to compute the motion sickness dose value:

$$MSDV_t = (a_{rms} \cdot t)^{1/2}$$

The percentage of unadapted adults who may vomit is then given by:

$$\text{percentage who may vomit} = {}^1/_3\ MSDV_t$$

This relation is based on exposures lasting from about 20 min to about 6 h with a prevalence of vomiting up to 70%. Figure 10 shows the predicted magnitudes of vertical oscillation required for 10%, 20% and 40% of persons to vomit within 2 hours.

Lawther and Griffin (1987) compared the motion sickness dose value ($MSDV_t$) procedure with the motion sickness incidence (MSI) method summarised in Section 3.3.1. It was shown that the frequency weightings were similar and that the dependence of vomiting on the magnitude and duration of motion were similar for magnitudes up to about 2.5 ms^{-2} r.m.s. and for durations up to about 6 hours.

Table 5 The filter gain of frequency weighting W_f used for assessing vertical acceleration with respect to motion sickness as defined by British Standard 6841 (1987). (Values shown are for the asymptotic approximation to a realisable filter)

Frequency, Hz	0.100	0.125	0.160	0.200	0.250	0.315	0.400	0.500
Gain	0.800	1.000	1.000	1.000	1.000	0.630	0.391	0.250

4. OSCILLATION ON SWINGS

Various investigators have decided that the motions of swings may be a convenient means of generating motion sickness. Two principal categories of swing have been used: (i) those secured so that the platform supporting the body swings as a simple pendulum and inclines under the action of the applied forces (e.g. 2 pole swings) and (ii) parallel swings which are secured at either end so that they remain horizontal during swinging (e.g. 4 pole swings). Figure 11 illustrates typical 2-pole and 4-pole swings. The motions experienced by observers on the swing are different in the two cases and neither imparts a unidirectional translational acceleration. For a simple swing of radius r, the swing

frequency, in Hz, is given by:

$$f_{swing} = \frac{1}{2\pi}\sqrt{\frac{g}{r}}$$

where g is the acceleration due to gravity (9.81 ms^{-2}).

On a two-pole swing it must be assumed that the person tilts with the platform - although this may not be true for the head unless it is restrained by a headrest. The subject is exposed to rotational acceleration at the swing frequency in addition to translational acceleration. In the absence of mechanical imperfections, the acceleration parallel to the platform (in the x- or y-axis of a seated subject) is zero since the acceleration in this direction is equal and opposite to the acceleration component due to gravity arising from the tilting of the platform. The acceleration perpendicular to the platform (in the z-axis of a seated subject) is primarily due to the radial acceleration: it always acts in the positive direction (keeping the subject on the platform) and oscillates at twice the frequency of the swing.

On a four-pole swing the platform remains horizontal and it must be assumed that the subject remains in the same orientation throughout the arc of swinging (although in practice this may not be the case without head restraint). There is therefore no rotational acceleration. In the horizontal direction (i.e. parallel to the platform and in the x- or y-axis of a seated subject) there is significant acceleration which is non-sinusoidal. The vertical motion is also non-sinusoidal.

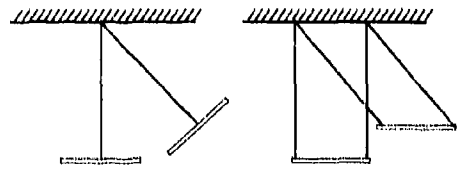

Figure 11 Diagrammatic illustration of 2-pole and 4-pole swings.

Reports of some studies do not clearly describe the form of swing used and it may not always be correct to calculate the motions experienced by subjects based on the categorisation and information presented below.

Studies in swings have been conducted for two main purposes: to assess the effectiveness of drugs or devise a procedure for eliminating susceptible persons when being selected for military service. A few studies have sought to determine the influence of the type and direction of motion on motion sickness.

4.1 Two pole swings

Using a swing with a radius of 4.3 metres (from the centre of swing to the seat) and a swing arc of 150 degrees, Hemingway (1945, 1946) investigated motion sickness in various groups of aviators and a control group during 20 minute exposures. In the control group 57% became ill and 2.5% of these vomited. Among three groups of aircrew known to be suffering from motion sickness there were 79% to 90% ill with 38% to 65% of these vomiting in less than 20 minutes. The sickness rates in aircrew not suffering from motion sickness were considerably lower: 28% ill with 11% to 15% vomiting. (It is inferred that the swing behaved as a simple pendulum and that the frequency of swinging was approximately 0.24 Hz). Similar results were obtained when administering placebos to control groups in drug studies with the same apparatus (Smith, 1946; Smith and Hemingway, 1946). Hemingway (1946) found that symptoms of motion sickness appeared to be unaffected by ambient temperature in the range 1 to 45 degrees Celsius.

Manning and Stewart (1949) report that the incidence and severity of 'swing sickness' was not dependent on time of day, room temperature or apprehension and was similar for those swung shortly before and those swung shortly after a meal. Using a two-pole swing with a radius of 4.29 metres (giving a frequency of about 0.23 Hz) and a swing arc of about 69 degrees, they investigated effects of body posture and visual conditions on the incidence of sickness during 30 minute exposures. With eyes closed, symptoms were least with subjects supine: 11% with nausea and vomiting compared with 51% when in a prone position and 51% when sitting facing the direction of swing. Opening the eyes reduced the problem to 5% when supine, 50% when prone and 27.5% when facing the direction of swing (there was poor vision when in the prone position). Wearing 'blackout goggles' resulted in a slightly greater sickness incidence (57.5%) than in the eyes closed condition, while sitting in a partially darkened room resulted in an intermediate incidence (39%). Covering the swing with a sheet, so as to remove the external visual cues and the feeling of the breeze, resulted in 64% with nausea and vomiting. When sitting sideways with the eyes open it was found that the incidence of nausea and vomiting was greater when sitting on the platform (24%) than when sitting supported on a chair placed on the platform (12%). This was attributed to the reduction of about 0.45 metres in the radius of swing to the vestibular system when using the chair. Standing on the swing facing the direction of motion resulted in the same incidence of nausea and vomiting as sitting facing the direction of swing (i.e. 27.5%) - even though the radius of the swing to the vestibular system was shortened by about 2 metres. Manning and Stewart attribute this to a different orientation of the head when standing. The authors conclude that a determining factor in

swing sickness is the orientation of the semicircular canals with respect to the direction of motion; sickness "*was greatest when accelerative forces were acting in the plane of the vertical semicircular canals*". They recommended the supine position, or any other position in which the 'vertical canals' are horizontal, for the transportation of air-borne troops.

Using a two-pole swing, Fraser and Manning (1950) systematically investigated the effect of swing radius and swing angle on motion sickness in seated men during 30 minute exposures. With a 90 degree angle of oscillation, increasing the swing radius from 1.8 metres (giving a swing frequency of about 0.37 Hz) to 3 metres (a swing frequency of about 0.28 Hz) increased the number of men with signs and symptoms of sickness from 4% to 50%. A further increase in the swing radius to 4.9 metres (~0.25 Hz) increased the sickness rate slightly further to 58%. With a 3 metre radius (a swing frequency of 0.28 Hz), increasing the angle of swing from 50 to 90 degrees increased the incidence of sickness from 22% to 50% while increasing the swing angle further to 130 degrees only produced sickness in 46%. The authors give some consideration to the accelerations received by subjects on the swing and conclude that rotational acceleration was not important since sickness increased when rotational acceleration decreased as the radius of the swing was increased. They conclude that the 'tangential' component of acceleration rather than the vertical acceleration may be necessary for sickness, or that at least two forms of motion are necessary for sickness. Using a simple swing with a radius of 4.6 metres swinging through 70 degrees at a frequency of 0.26 Hz, Johnson *et al* (1951) found that the greatest incidence of sickness occurred among subjects making the greatest head movements.

Other studies have been conducted on swings of various types. For example, Babkin *et al* (1946) produced motion sickness in dogs using a swing with a radius of 2.4 metres which swung through about 70 degrees at 0.33 Hz. This was reported to produce definite signs of motion sickness in about 60% of dogs tested; the use of a shorter swing radius produced a lower incidence of sickness. Babkin and Bornstein (1943) had earlier found that bilateral labyrinthectomy abolished symptoms of sickness in two dogs previously susceptible to swing sickness. Johnson (1951) reports that five dogs who all vomited on a swing within 30 minutes did not vomit when their heads were fixed. Chinn and Plotnikoff (1953) found that using a swing with a radius of 4.3 metres (a swing frequency of about 0.25 Hz) with a swing arc of 120 degrees, 60% of dogs vomited within 30 minutes and 25% of men vomited in 20 minutes. Some drugs were able to reduce the incidence of vomiting. Wang and Chinn (1954) used the same motion and showed a considerable reduction in vomiting among dogs after the surgical removal of their 'chemoceptive emetic trigger zone'.

Money and Friedberg (1964) produced vomiting in restrained dogs standing on a two-pole swing of radius 4.1 metres swinging through a 70 degree arc at 0.25 Hz. Of 57 dogs tested, 17 vomited within 25 minutes in their initial test and 16 of these vomited within 25 minutes during three subsequent tests. Surgical procedures to inactivate the semi-circular canals while leaving the otoliths intact, greatly reduced the susceptibility of the dogs to motion sickness on the swing. Later investigations with dogs involved surgical removal of the ampullae, the utricle and the saccule (Money and Cheung, 1983). The same two-pole swing test resulted in seven out of eight dogs vomiting within 25 minutes on four separate occasions prior to the operation. Following surgery none of the dogs vomited during the swing test. The emetic response to some drugs was also greatly diminished by the surgical removal of the vestibular system.

Using a two-pole swing with a radius of 3.7 metres oscillating through an arc of 90 degrees, Crampton and Daunton (1983) produced motion sickness in cats within a clear plastic box. Mean durations before retching or vomiting are given as approximately 18 minutes. Daunton *et al* (1984) report experiments on cats exposed to various motions including those generated by a two-pole swing with a radius of 1.8 metres swinging out about 60 degrees with a frequency of 0.37 Hz and a vertical displacement of 0.9 metres. The swing was placed within a stationary illuminated box so that during swinging there was combined visual and vestibular stimulation. Stimulation by visual movement only was provided by swinging the box (0.25 Hz, 60 degrees) over the stationary animals. With the combined visual and vestibular stimulation, 25% of cats vomited whereas only 10% vomited with the visual stimulation alone (see Section 10).

In comparison with the translational studies reported in Section 3 it can be shown that the above swing studies with seated persons have produced a greater incidence of motion sickness than would be predicted from the z-axis acceleration (i.e. radial acceleration produced by a combination of centripetal acceleration and a gravitational component). (Two-pole swings produce z-axis acceleration of seated subjects at twice the swing frequency so, for this axis, the frequencies quoted in the text above should be doubled.) The Wesleyan University studies and the Human Factor Research Inc. studies showed that sickness incidence decreased with increasing frequency. In order to obtain sickness incidence of 50% within 30 minutes, the z-axis acceleration would need to be greater than that occurring in the above studies. This could be achieved with larger angles of swing and lower frequencies of swing - both difficult to achieve. For example, the motion sickness dose value procedure indicates that a frequency-weighted acceleration of 4.5 ms^{-2} r.m.s. would be required in order to achieve 50% vomiting in 30 minutes. This would require a longer swing than has been used in the studies: a radius of 15.9 metres (swinging at 0.125 Hz so as to give a z-axis acceleration at 0.25 Hz) with an angle of swing of about 145 degrees would be needed. It seems reasonable to conclude that z-axis oscillations of acceleration are not alone the cause of sickness on two-pole swings.

For a person restrained to a two-pole swing, the only other motion is the rotational oscillation. The magnitudes of roll (or pitch) oscillation used in the swing studies are larger than those found to produce only minor motion sickness in the McCauley *et al* (1976)

studies (see Section 4.1 b). Evidence from the 'roll pitch rocker' studies of Morton *et al* (1947) suggests that motion sickness generated by a 6.6 metre up and down motion of a seesaw was not affected by the addition of roll motion of up to 39.5 degrees. (It was assumed that the vertical motion, not the pitch motion, of the seesaw was responsible for sickness - possibly because the angles of roll and pitch were similar and studies in an elevator confirmed that vertical motion caused motion sickness). It therefore currently remains uncertain which characteristics of the motions on two-pole swings are primarily responsible for sickness (see Section 4.2 below).

4.2 Parallel swings

A series of studies of drugs for preventing motion sickness in dogs and man was conducted on a parallel swing of radius 4.4 metres giving a frequency of oscillation in the fore-and-aft axis of 0.25 Hz (Noble, 1945a, 1945b, 1946, 1948). The heads of the men were 0.6 metres above the swing. In most studies, dogs and men were made sick by swinging through 90 degree angles; between 55% and 60% of 369 men vomited in the course of 30 minute exposures to this motion. It was found that lesser angles of swing were sufficient to cause vomiting among the more susceptible dogs and men. Eight out of ten susceptible men vomited (and the other two were very nauseated) when exposed to 30 degree swings with an average time to vomiting of 14.5 minutes compared to 10.5 minutes with the 90 degree swing.

Johnson and Taylor (1961) compared the sickness generated by 2-pole and 4-pole swings - both having a radius of 4.6 m (to the heads of subjects), an oscillation frequency of 0.27 Hz and a swing arc of 70 degrees. Using 800 young male subjects facing the direction of swing (so as to produce motion in the fore-and-aft axis) and up to six minutes of motion they investigated the differences between the swings, the effects of fixing the head and the effects of closing the eyes. The greatest percentage of sickness symptoms arose with the head free and the eyes closed (35% felt ill on the 2-pole swing and 20% felt ill on the 4-pole swing). Restricting the head, opening the eyes, or both restricting the head and opening the eyes resulted in a considerable decrease in sickness incidence for both swings. The authors concluded that sickness was caused by "*angular accelerations resulting from unrestrained movement of the head*". The beneficial effect of vision was attributed to subjects being able to use sight to assist in the maintenance of a steady head. The occurrence of up to 7% sickness with the head fixed was attributed to incomplete immobilisation of the head.

4.3 Other devices

A Ferris wheel device har been used to provoke motion sickness in cats (Crampton and Lucot, 1985; Fox *et al*, 1987; Lucot and Crampton, 1987; Lucot *et al* 1989; Crampton and Lucot, 1991). Cats were placed in two clear boxes which hung at either end of a frame which rotated about a horizontal axis through its centre. The radius from the centre of rotation to the point of suspension of each box was 0.445 m. Continuous rotation of the frame caused the suspension of each box to move in a vertical circle. With the cages freely suspended, they will have tended to swing outwards under the action of centripetal acceleration as they ascended and descended. The motion experienced by the cats was not merely vertical sinusoidal oscillation. Using rotation rates between 0.15 Hz and 0.58 Hz all stimuli caused some sickness but 0.28 Hz (17 rpm) produced the earliest onset of motion sickness and it appeared that 40% of cats would vomit within 30 minutes when exposed to this frequency.

5. ROTATION ABOUT A VERTICAL AXIS

5.1 Constant speed rotation

Corning (1904) describes some apparatus in which he was able to produce symptoms of sickness by continuous rotation about a vertical axis. He found that 30 rpm was sufficient to induce in himself "*a high degree of vertigo, giddiness and nausea being especially marked when the eyes, closed during rotation, were, upon its cessation, opened suddenly*". Corning used the chair to assess the efficiency of drugs in combating motion sickness and subsequently advocated the use of hyoscine.

Guedry (1968) reports that simple rotational acceleration at low rates does not produce symptoms of motion sickness. In an experiment with more than 100 men who received 176 accelerations to 10 rpm within a 4 hour period no subject exhibited symptoms.

Leger *et al* (1981) found that motion sickness was not common during 5 minute exposures to 20 rpm rotation about a vertical axis. Only one subject reported symptoms of more than trivial severity; most symptoms arose when subjects had an external visual frame of reference.

Daunton *et al* (1984) determined the incidence of motion sickness in squirrel monkeys exposed to constant speed rotation about a vertical axis with a view of the laboratory. With a rotation rate of 10 rpm, 70% of animals retched or vomited; with the rotation at 25 rpm all animals retched or vomited (see also Section 10).

Wilpizeski *et al* (1985) conducted three experiments with squirrel monkeys in which the animals were exposed to continuous rotation at 33 rpm. When allowed to see out through the rotating transparent cage and not restrained, all monkeys vomited on several occasions within 2 hours. Blindfolding greatly reduced the incidence of vomiting. Repeated exposure produced habituation in only a few animals (see also, Wilpizeski *et al*, 1987a,b).

Daunton and Fox (1985) reported the occurrence of motion sickness in squirrel monkeys exposed to body rotation or optokinetic rotation about a vertical axis with various viewing

conditions. With 10 rpm rotation of the visual surround around stationary animals, 82% became sick within 30 minutes. About 77% became sick when only the animals were rotated, 95% were sick when both the animals and the visual surround were rotated together and 45% became sick when the animals were rotated in the dark. With a higher rate of rotation (25 rpm) the sickness rates increased for all three conditions involving motion of the animals but decreased for the condition involving only movement of the visual field. The animals were free to move within a small cage during the motions.

The effects of various restraint conditions on motion sickness in squirrel monkeys exposed to 30 rpm for up to 120 minutes has been reported by Wilpizeski *et al* (1985). With a clear view of the laboratory all of the monkeys exhibited motion sickness within 120 minutes when they were allowed to move. When the torso was fixed, sickness fell to about 50%, and with both torso and head restrained the sickness fell a little more. With torso and head fixed and a blindfold, no animal exhibited signs of motion sickness.

During continuous constant velocity rotation about a vertical axis, the sensation of rotation diminishes and nystagmus does not persist after the onset period. On cessation of rotation there is a sensation of counter-rotation and some horizontal nystagmus. Motion sickness induced by the above exposures to constant speed rotation about the vertical axis may have been caused by the apparent movement of the visual scene (see Section 10), movements of the head (see Section 8) or the acceleration and deceleration before and after the period of constant velocity (see Section 5.2).

5.2 Rotational acceleration

Severe, or sudden, rotational acceleration about a vertical axis can produce motion sickness. A test for pilot susceptibility to airsickness developed in 1922 to 1924 has been reported by a former Director of Medical Research for the Royal Air Force (Flack, 1931). With eyes open, subjects were spun on a rotating chair through 10 rotations in 20 seconds (30 rpm). This exposure, which is reported to have been capable of causing vertigo, nausea and vomiting, involves acceleration and deceleration in addition to the period at constant speed rotation.

Lackner and Graybiel (1979) describe a 'sudden-stop vestibular-visual interaction test' in which subjects are accelerated at 20 deg s^{-2} to 300 deg s^{-1} (50 rpm), maintained at this velocity for 30 seconds and then brought to a stop in 1.5 seconds. The procedure was then repeated until a motion sickness end point was reached or 50 stops had been completed. For 10 subjects receiving their first exposures with eyes open during the test, an average of about 14 stops was required before slight nausea occurred. With eyes closed an average of 38 stops was required. A similar test with subjects viewing within a stationary striped drum was used by Graybiel and Lackner (1980). It has been found that susceptibility to sickness with this test increases if the gravito-inertial force level is decreased to zero G or increased to 2 G (Lackner and Graybiel, 1983).

McCabe and Gillingham (1964) placed cats within a mould, so as to restrict movement, and subjected them to rotation about a vertical axis followed by high rates of deceleration (up to 38 rad s^{-2}; 2160 degrees s^{-2}). None of the cats became sick with this stimulus but they were made sick by vertical oscillation (see Section 3.1.1). This led the authors to conclude that the otoliths rather than the semicircular canals were responsible for motion sickness.

Ossenkopp and Ossenkopp (1990) concluded that 20 minute periods of body rotation on a turntable at 70 rpm with 15 seconds on and 5 seconds off caused motion sickness in guinea pigs. Corcoran *et al* (1990) compared susceptibility to sickness of rhesus monkeys and squirrel monkeys when exposed to continuous rotation (45 minutes at 25 rpm) and when exposed to sudden-stop rotation (45 minutes with sudden stops every 30 seconds). None of the rhesus monkeys became sick in either condition but all of the squirrel monkeys became sick in both conditions. (The squirrel monkey is in the same taxonomic order as man and has been used as a surrogate for humans in many experimental studies.)

The motion sickness arising from rotational acceleration is greatly influenced by the visual field (see Section 10). Head movement may have also contributed to the reported signs and symptoms in some of the above studies (see Section 8).

5.3 Complex rotational motions

'Double rotation' has been used to study some behavioral responses of rats which are thought to be indicative of motion sickness (Morita *et al*, 1988 a, b). Rats are unable to vomit and so some other indication of sickness is required. The exposures to double rotation were generated by placing the rats on a small turntable 25 cm off the axis of a second turntable. While the smaller upper turntable rotated continuously at 80 rpm the lower turntable alternately accelerated to 25 rpm and then decelerated to a stop. The responses of the rats suggested that this double rotation was more nauseogenic than 80 rpm rotation of the upper turntable alone. Removal of the vestibular system appeared to prevent the rats from experiencing sickness.

Another complex form of motion used to provoke sickness in animals has combined constant speed rotation about the vertical axis with vertical oscillation (Ordy and Brizzee, 1980). Using a sinusoidal 0.5 Hz vertical oscillation with an excursion of 152 mm (about 0.5 ms^{-2} r.m.s.), squirrel monkeys were rotated at either 10, 25 or 50 rpm for 60 minutes. They had a clear view of the laboratory and were able to make small movements within their cage. The 25 rpm condition caused the greatest sickness: 89% vomiting at 25 rpm compared with 21% at 10 rpm and 71% at 50 rpm. Covering the cage to eliminate visual cues reduced sickness and there was some evidence that male monkeys suffered slightly more

from sickness than females. A separate experiment indicated that ablation of the area postrema of squirrel monkeys inhibited their motion sickness response to this type of motion (Brizzee et *al*, 1980).

6. OFF-VERTICAL ROTATION

Rotation about an off-vertical axis produces a 'rotating linear acceleration vector' from the force of gravity. Unlike constant speed rotation about a vertical axis, this produces sensations of turning and nystagmus which persists throughout the rotation, but little or no post-rotation effects. Rotation about an off-vertical axis can also be highly nauseogenic. When rotation occurs about a horizontal axis, the term 'barbecue-spit rotation' is sometimes used. Johnson (1954) found that the use of this type of rotation combined with head movements in the coronal or sagittal plane could produce sickness in up to 95% of persons within five minutes and that sickness could be induced within 15 seconds in some.

Benson and Bodin (1966) reported that rotation of subjects on a stretcher about a horizontal axis for 3 to 4 minutes was likely to produce nausea with rotation rates of 40 to 60 degrees per second (7 to 10 rpm). Using 120 second periods of rotation about a horizontal axis with rates of 10 and 30 rpm, Correia and Guedry (1966) report that twelve of 20 subjects were unable to complete the experimental sequence due to motion sickness.

Graybiel and Miller (1968) rotated normal subjects and some with labyrinthine defects on a chair tilted by 10 or 20 degrees from the vertical. All but six of 66 normal subjects reached moderate malaise A (M IIA) (see Tables 2a and 2b) with a 10 degree tilt and a sequence of rotations incrementally increased to 25 rpm over 65 minutes. More than half reached this end point by 12.5 rpm over about 30 minutes. None of five subjects with labyrinthine defects developed symptoms of motion sickness. Graybiel and Miller (1970) report similar results and show that there was a high correlation between susceptibility to sickness with this test and susceptibility to sickness as indicated by the Coriolis Sickness Susceptibility Index, CSSI (see Section 8.2.1).

The effect of varying the rate of rotation (from 2.5 to 45 rpm) about a 10 degree off-vertical axis, and the effect of varying the angle of tilt (from 2.5 to 25 degrees) at a rotation rate of 17.5 rpm was investigated in a small number of blindfolded subjects by Miller and Graybiel (1973). There was little or no sickness with either a 2.5 or a 5 degree tilt and some subjects were insensitive at rotation rates above about 30 rpm. Greatest susceptibility to motion sickness occurred in the range 15 to 20 rpm with all four subjects reaching 'moderate malaise A' (i.e. M IIA) within 15 minutes with a 10 degree tilt. The authors note that this range of frequencies is similar to that giving greatest susceptibility to vertical oscillation. As the angle of tilt increased from 2.5 degrees to 25 degrees an increased susceptibility to sickness was shown by reductions in the time to reach M IIA.

Graybiel and Lackner (1977) found that 60 minutes of rotation about an earth horizontal axis at 30 rpm caused sickness but that rotating the body 10 degrees head-up or 10 degrees head-down did not differ from rotation in the horizontal position.

Lentz and Guedry (1978) describe the 'Tilted-Axis Rotation Test' (TART) in which blindfolded standing persons are rotated about their z-axis while they (and the axis of rotation) are tilted at various angles. In the initial trial, a vertical subject was accelerated at 25 deg.s^{-2} to a speed of 60 deg.s^{-1} (10 rpm) in the clockwise direction. After 90 s at constant velocity the subject was decelerated to a stop at 25 deg.s^{-2}. In a second trial the rotation was the same except motion was in the counterclockwise direction. In the third and fourth trials the axis was tilted 30 degrees off-vertical. In the fifth and sixth trial the device was accelerated at 25 deg.s^{-2} to 102 deg.s^{-1} (17 rpm). There was an interval of 5 minutes between trials. Lentz and Guedry state that it was not uncommon for subjects to terminate the test prior to completion.

Leger et *al* (1981) exposed subjects to 5 minute periods of 20 rpm with rotation about a horizontal axis through the three axes of the body (the x-, y-, and z-axes) and with three visual conditions (blindfold, internal frame of reference and external frame of reference). There were reports of sickness during 95% of the 59 conditions experienced by 11 subjects. There was no significant difference according to whether the rotation about the horizontal axis occurred about the x-axis, the y-axis, or the z-axis of the subject. There was less sickness with the external visual frame of reference.

With rotation rates between 4 and 17 rpm, Mowrey and Clayson (1982) used the provocation provided by 6 minute exposures in an inclined rotating chair to assess the effectiveness of ginger as an anti-motion sickness agent. They state that none of the 36 subjects was able to stay in the chair for 6 minutes when given a placebo whereas half of the subjects survived 6 minutes exposures after consuming powdered ginger.

Off-vertical rotation (25 rpm for 30 or 60 minutes at an inclination of 20 degrees to the vertical) has also been used to study motion sickness in the rat (see Sutton et *al*, 1988).

The perceptions of body movement during rotation about an axis inclined to the vertical are discussed by Denise et *al* (1988). These authors reported that using a rotation rate of 7.5 rpm with inclinations between 5 and 30 degrees, most subjects reported sickness after a delay ranging from less than two minutes to more than 45 minutes.

Cheung *et al* (1990) tumbled subjects head-over-heels about an earth-horizontal axis at a rate of 20 rpm for up to 10 minutes to determine the effects of fitness training on motion sickness susceptibility. They found no difference between males and females but an increased susceptibility to sickness after fitness training.

7. ROTATIONAL OSCILLATION

7.1 Oscillation about a vertical axis

Moore *et al* (1977) describe a 'Visual-Vestibular Interaction Test' (VVIT) in which a seated subject is oscillated in yaw (i.e. about his z-axis) sinusoidally at 0.02 Hz with a peak angular velocity of ±155 deg.s^{-1}. (This corresponds to a rotational displacement of approximately ±3.4 rotations). During rotation, the subject identified the co-ordinates of numbers shown on a visual display within an enclosed cabin. Lentz and Guedry (1978) say that the motion itself, when experienced with simple visual displays or in darkness is not disturbing. They say the problem arises when the motion-induced nystagmus is superimposed on the saccades required during shifts of visual fixation and that nausea tends to build up slowly on successive cycles of motion. Lentz *et al* (1977) reported that a five minute exposure to the test was more provocative of sickness than the Brief Vestibular Disorientation Test (BVDT) - see Section 8.2.1. There were significant correlations between the results obtained from the VVIT and the BVDT. There was also a low, but statistically significant, correlation with results of a motion sickness questionnaire indicating experience of sickness in various forms of transport.

Guedry *et al* (1982) presented seated subjects performing a head-fixed visual search task with 5 minute exposures to two types of sinusoidal yaw oscillation: 0.02 Hz at ±155 deg.s^{-1} per second or 2.5 Hz at ±20 deg.s^{-1}. While oscillation at 2.5 Hz produced no convincing signs of sickness, the oscillation at 0.02 Hz was highly nauseogenic. Six of 51 subjects failed to complete the 5 minute exposure to 0.02 Hz and 4 vomited; 75% of the subjects reported some relevant symptom. The authors also exposed the subjects to cross-coupled (i.e. Coriolis) stimulation produced by ten 45 degree head movements during a 5 minute exposure to 15 rpm and found this somewhat less nauseogenic (see Section 8.2.1).

Igarashi *et al* (1986) provoked sickness in squirrel monkeys by oscillating them about a vertical axis (at 0.25 Hz with an amplitude of 90 degrees and velocity of 141 deg.s^{-1}) while they were within an optokinetic drum rotating with the same motion but a 45 degree phase lag. When the motion occurred about a vertical axis there was vomiting in 13.3% of the animals, whereas when the motion occurred about the horizontal axis there was vomiting in 50.8% of the animals.

Lackner and Dizio (1989) exposed subjects to 40 deg.s^{-1} constant velocity rotation about a vertical axis with the direction of rotation changing (at 300 deg.s^{-2}) every minute. When tested wearing a helmet which doubled the inertia of the head, all eight subjects experienced nausea, but when tested without the helmet there was no nausea.

7.2 Oscillation about a horizontal axis

Morton *et al* (1947) used a 'roll-pitch rocker' to expose subjects simultaneously to roll motion (through 25.5 degrees) and a combined pitch and vertical motion through 3.6 metres at the end of a 4.9 metre arm of a see-saw. Oscillation on the see-saw alone at 0.125 Hz resulted in 40% of subjects vomiting, whereas with this motion combined with roll motion at 0.08 Hz, 33% of subjects vomited. The illness rates were similar in both conditions and the authors concluded that vertical motion from the seesaw was the cause of the sickness. Assuming sinusoidal vertical motion, the motion sickness dose value procedure would predict about 16% of persons becoming sick during a 1 hour exposure to this motion. Sickness was slightly greater when the seesaw oscillated at about 0.10 Hz, and lower when it oscillated at about 0.09 Hz. If the full 3.6 metres of displacement was used at all frequencies, these changes with frequency are consistent with motion sickness being caused by vertical oscillation and possibly consistent the motion sickness dose value procedure.

Igarashi *et al* (1987) exposed squirrel monkeys to oscillatory motion about an earth horizontal axis while viewing within an optokinetic drum oscillating in the same axis and by the same amount but with a 45 degree phase difference. Oscillation at 0.25 Hz with an amplitude of 90 degrees provoked sickness in normal monkeys but was decreased in those with surgical removal of otolith end organs.

McCauley *et al* (1976) investigated response to pitch and roll motion at 0.115, 0.230 or 0.345 Hz with magnitudes in the range 5.5 to 33.3 deg.s^{-2} r.m.s. When the rotational motions were added to a vertical motion of 0.25 Hz at 1.1 ms^{-2} r.m.s., the incidence of sickness was not significantly different from the incidence of sickness with the vertical motion alone. No subject vomited with roll motion alone (33.3 deg.s^{-2} at 0.345 Hz) but two subjects vomited with this magnitude of pitch. The authors conclude that the results support the view that vertical motion is the cause of seasickness and infer that head motion during vertical oscillation may not contribute to sickness.

The effects of rotational oscillation about an earth-horizontal axis at 45 degrees to the x-axes and the y-axes of seated subjects was investigated by Kolnick and Bles (1989). Over 35-minute exposures to oscillations (10 degrees peak at 0.025 Hz, 0.1 Hz and 0.025 Hz + 0.1 Hz), subjects performed various tasks and rated subjective symptoms. No subject vomited but symptoms increased with exposure time and were greatest when there was no outside view. The provision of an artificial horizon reduced motion sickness symptoms. The tasks included a requirement to make 45 degree head movements and the authors speculate

that Coriolis effects may have contributed to sickness.

8. CORIOLIS, OR CROSS-COUPLED, STIMULATION

8.1 Principles of cross-coupled stimuli

If, while the body undergoes constant speed rotation, the head is rotated about an axis other than the axis of constant speed rotation, nausea and other classic symptoms of motion sickness may appear. Most commonly, the subject is rotated on a chair about the vertical axis and required to make pitch or roll movements of the head. In the sensory conflict theory this is assumed to arise from the otoliths sending the correct information on head movements while the semicircular canals give a false indication of head movement.

The 'false' indication of head motion produced by the semicircular canals arises when a canal is rotated so as to change the extent to which it is within the plane of the constant speed rotation. Consider, for example, a vertical canal with constant speed rotation occurring about the vertical axis (i.e. in yaw) so that the canal is not in the plane of the rotation. Assume the canal is then rotated forward in pitch, so as to become horizontal and in the plane of rotation. The rotational velocity of the canal will have changed from zero to the velocity of the constant speed rotation. The fluid within the canal (i.e. endolymph) will need to accelerate to reach the speed of rotation. Until this speed is reached the endolymph will deflect the cupula and so indicate that there has been a change of rotational velocity in the plane of the canal (i.e. in the yaw axis of the body but in the roll axis of the head, for pitch head movements). However, the only movement of the head containing the canal has been in the pitch axis. So, while the semicircular canals indicate pitch and roll rotation of the head, the otoliths and proprioceptive information indicate only a pitch motion of the head. In summary, when a person is exposed to continuous rotation about one axis and makes a rotary movement of the head about a second axis, there is a feeling of being rotated in a plane approximately orthogonal to the two true motions.

The above illustration is only a simplified introduction to the cross-coupled effect. For example, it is also necessary to consider the signals that arise from canals that leave the plane of rotation. For a canal orientated horizontally and exposed to rotation at constant speed for about 30 s or more the endolymph in the canal will be rotating with the walls of the canal and will then cause no deflection of the cupula. Suppose that the canal is now rotated in pitch while still undergoing constant speed rotation about the vertical axis of the body. When the canal becomes vertical there will be no rotation in the plane of sensitivity of the canal. The decrease in rotational velocity will result in the endolymph deflecting the cupula.

For pitch head motions while exposed to constant speed yaw rotation of the body, the behaviour of canals in the roll and yaw axes of the head may follow the above general explanation. A canal sensitive to motion in the pitch axis of the head would remain out of the plane of rotation at all times and would indicate head movement as if there was no constant speed rotation. If the head were to be rotated in roll while undergoing constant speed yaw rotation, the 'erroneous' signals would arise from canals sensitive in the pitch and yaw axes - a canal sensitive in the roll axis would give the same roll signal as in the absence of constant speed rotation. In practice, the semicircular canals are not truly orthogonal and aligned in the pitch, roll and yaw axes of the head. Consequently, some change will be indicated by all three canals during most head motions. It is assumed that the adaptive processing of signals from canals allows for the resolution of the signals into pitch, roll and yaw motions of the head (see Section 8.2.2).

The sensory conflict explanation of sickness produced by cross-coupled stimulation usually assumes that the otoliths give a signal dependent on their orientation with respect to gravity and are unaffected by the constant speed rotation. However, as the otoliths move away from the centre of rotation there will be centripetal forces and translational acceleration. These might be significant in some cases.

The direction of the illusory sensation introduced when the semi-circular canals enter and leave the plane of rotation can be envisaged without mathematical derivations. Suppose the constant speed rotation is in the clockwise direction (looking down on the subject) and the head is rolled to the left. An 'idealised semi-circular canal' orientated in the roll plane of the head is not in the plane of constant speed rotation at any time during the head movement. The signals given by this canal are therefore the same as in the absence of constant speed rotation. A canal orientated in the pitch plane of the head enters the plane of rotation when the head is rolled to the left and will indicate an increase in pitch velocity. Signals from this canal would not occur with this head movement in the absence of the constant speed rotation. The yaw canal which leaves the plane of rotation will indicate a decrease in velocity - again, a change which would not occur in the absence of constant speed rotation. The overall impression caused by a pure roll movement of the head is a combined movement involving roll, pitch and yaw. Figure 12 provides a diagrammatic illustration of the signals that may be expected from canals orientated in the roll, pitch and yaw axes of the head when making a roll movement of the head during constant speed rotation. The semi-circular canals are not capable of detecting constant speed rotation: in Figure 12 it is assumed that the head has been held stationary during constant speed rotation long enough for the effect of any previous head movement (and the original acceleration of the turntable) to have decayed. The decay in turning sensation after the roll movement is shown in the figure.

8.2 Nature of practical cross-coupled tests

Various purpose-built devices and proprietary rotating chairs, often designed for vestibular function tests, have been used to generate cross-coupled stimuli. The test is usually conducted with seated subjects rotated about their vertical axis (i.e. yaw rotation about their z-axis). The principal variables involved in the test are:

(i) rate and direction of rotation;
(ii) direction and angle of head movement;
(iii) frequency and duration of head movement;
(iv) visual conditions;
(v) gravitational force;
(vi) end point of test.

The full influence of all variables has not been exhaustively studied but some general conclusions appear possible.

8.2.1 Effect of rate and direction of rotation

The nature of the cross-coupled stimulation is such that greater rates of rotation would be expected to produce greater magnitudes of 'illusory motions' and, presumably, a higher incidence of sickness.

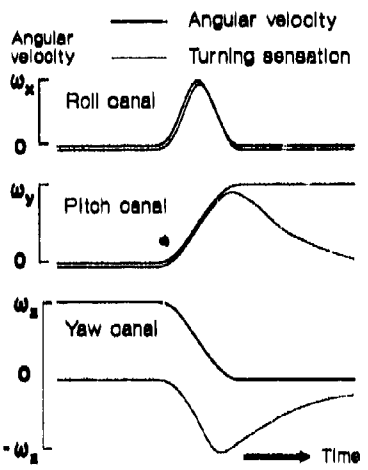

Figure 12 Angular velocity of semi-circular canals and sensation of turning produced by roll head motion during constant speed rotation about the z-axis of the body. (Idealised canals in the roll, pitch and yaw planes of the head. After Benson, 1984).

Using 90 degree movements of the head, Miller and Graybiel (1969, 1970a, 1970b, 1970c, 1974) investigated sickness as a function of rotational velocity from 1 to 30 rpm (i.e. 0.0166 to 0.5 Hz; 6 to 180 deg.s^{-1}). Blindfolded subjects made movements to the front, right, back, left, front returning to the upright between each movement and holding each head position for 1 s. After completing this sequence of 5 head movements in 14 s they paused for 20 s before repeating the same series of head motions. There were large inter-subject differences with some subjects giving useful results in the range 1 to 10 rpm while others could be tested over the range 5 to 30 rpm. Nevertheless, for all subjects the number of head movements required to reach both 'severe malaise' (M III) and 'moderate malaise' (M IIA), decreased as the rate of rotation increased. (The system used to identify and rate the symptoms of motion sickness was that developed by Graybiel *et al*, 1968; see Tables 2a and 2b). Presenting the data on logarithmic scales showed that there was at least a 10:1 difference in the number of head movements required for moderate malaise by the most and least sensitive subject at a fixed rate of rotation. Different subjects showed similar rates of decrease of the logarithm of the number of head movements with the logarithm of the increase in the rate of rotation. The average slope appears to have been such that there was an inverse relationship between the number of head movements, N, before moderate malaise and the rate of rotation, R, raised to the power of 1.7.

Miller and Graybiel determined the 'average stressor effect' (E factor) of a single head movement at each rotational velocity for both moderate malaise (M IIA) and for severe malaise (M III). Values for other degrees of malaise were also determined (Miller and Graybiel, 1970c, 1974). The logarithmic relationship between the E factors and chair velocity for five levels of motion sickness severity are shown in Figure 13.

The Coriolis Sickness Susceptibility Index, CSSI, was defined as the product of the average stressor effect of a single head movement (E factor) with the number of head movements required to reach the desired motion sickness endpoint:

$$CSSI = E \times N$$

The value of E can be obtained from Figure 13. Alternatively, CSSI values can be calculated from the following equations:

Slight malaise (M I): $CSSI_{M\,I} = 0.00536\ R^{1.67} \times N$

Moderate malaise B (M IIB): $CSSI_{M\,IIB} = 0.00304\ R^{1.65} \times N$

Moderate malaise A (M IIA): $CSSI_{M\,IIA} = 0.00206\ R^{1.70} \times N$

Severe malaise (M III): $CSSI_{M\,III} = 0.00104\ R^{1.67} \times N$

Frank sickness (FC): $CSSI_{FC} = 0.00045\ R^{1.65} \times N$

The index was arranged so that practical CSSI values would fall within the range 0 to 100, with scores increasing for less susceptible subjects. A value greater than 100 would be obtained by a subject who required more than 150 head movements to reach moderate malaise at a revolution rate of 30 rpm. High CSSI values indicate that a subject is resistant to motion sickness while low CSSI values indicate high susceptibility.

The Coriolis Sickness Susceptibility Index provides a means of quantifying individual

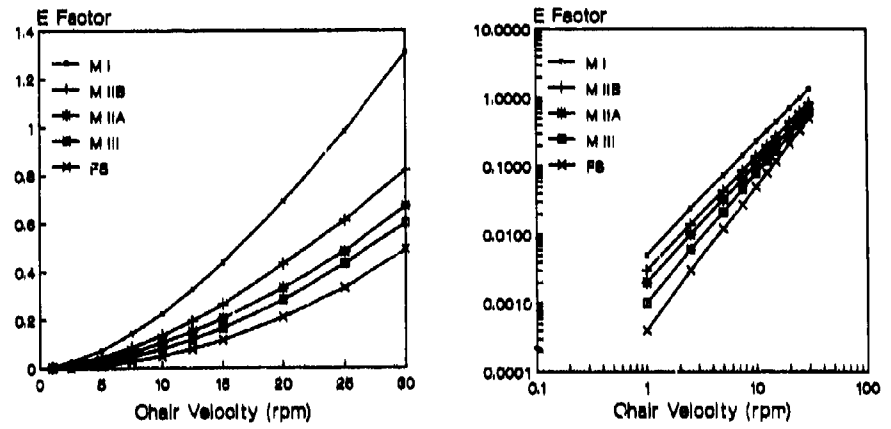

Figure 13 Relationship between the E factor and chair velocity for five levels of motion sickness severity. The E factor indicates 'average stressor effect' of a single head movement at each velocity. (see Miller and Graybiel, 1970c).

susceptibility to sickness by determining the number of head movements required during rotation at any rotation rate between 2.5 and 30 rpm to cause a defined degree of malaise (on the scale shown in Tables 2a and 2b, Graybiel *et al*, 1968). If the CSSI value for a subject is known it becomes possible to predict the rate of rotation that will be required to cause the required motion sickness endpoint within a defined number of head movements. Miller and Graybiel (1970c) suggested that moderate malaise (M IIA) yielded the best balance between subject acceptability and test confidence.

The CSSI values reported by Miller and Graybiel (1969, 1970a, 1970b, 1970c) were determined with blindfold subjects and the pattern of 90 degree head movements described above. Other visual conditions or other types or patterns of movement may not provide the same values.

For severe malaise (M III), Miller and Graybiel (1969) found a high test-retest reliability when CSSI values were obtained on two occasions. Using 250 normal subjects (mainly aviation students and flight crew) they found that the distribution of CSSI values was highly skewed such that while the mean value was 15.3, the median was 10.0 and the mode 7 to 8. Figure 14 illustrates the cumulative distribution of CSSI values in the group. Miller and Graybiel (1974) report almost identical distributions for all four levels of malaise (M I, M IIB, M IIA, M III). Calkins *et al* (1987) used a logarithmic transformation to reduce the skew in the distribution of CSSI values. They also showed that after the application of this transformation there was a 'respectable reliability' in CSSI values as indicated by two successive exposures of subjects to the test. Miller and Graybiel (1970c) showed that there was a high correlation between the CSSI values of subjects across five motion sickness end points.

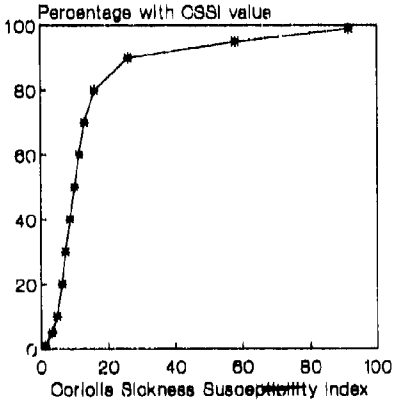

Figure 14 Cumulative distribution of CSSI values in a group of 250 aviation students and flight crew. (The distribution is highly skewed - mean: 15.3; median: 10.0; mode: 7 to 8. Data from Miller and Graybiel, 1969).

The CSSI index implies that a doubling of the revolution rate will have a greater effect than doubling the number of head motions. For example, a three fold increase in the rate of rotation is approximately equivalent to a six-fold increase in the number of head movements.

Kohl *et al* (1986) defined a procedure for calculating the value of the CSSI when subjects have been exposed to a sequence of motions in which the rate of rotation has been systematically increased.

The application of cross-coupled stimulation requires prior judgement of the rate of rotation which will result in the desired symptoms with a reasonable number of head movements. Miller and Graybiel (1970) explain how responses to a questionnaire may be used to determine both motion experience and susceptibility to motion sickness and hence select

an appropriate revolution rate for each subject. They state that when M III symptoms (severe malaise) occurred within 40 to 166 head movements, the signs and symptoms developed gradually without the hazard of provoking frank sickness (vomiting). Consistent with the data in Figure 14, they found that 50% of subjects reached severe malaise with a CSSI of 10.

The dependent variable in cross-coupled vestibular stimulation studies can be either the degree of nausea produced by a predetermined stimulus or the stimulus required to produce a predetermined degree of nausea. When the stimulus is predetermined, some scale such as the scale shown in Tables 2a and 2b (Graybiel *et al*, 1968) may be used. If the endpoint is predetermined, some measure of the stimulus severity is required. This could be the number of head movements required when exposed to a constant rate of rotation. However, with the wide inter-subject variability in susceptibility to sickness, it is often convenient to increase the rate of rotation as the test proceeds so as to be sure that most persons will produce the desired symptoms. A cumulative measure (such as total number of head movements executed before reaching the endpoint) can still be used so long as all subjects receive the same sequence of conditions. Another advantage of this type of test is that it is possible to prevent subjects developing severe symptoms by terminating the exposure when the signs or symptoms are less severe. Consequently, some studies have chosen to use fixed rates of rotation while others have varied the rate according to subject response.

Some studies suggest that the magnitude of the effect (e.g. perceived tilt) when making head movements in a rotating environment are linearly related to the rotational velocity (Guedry and Montague, 1961). Studies in a slow rotation room showed that more head movements were required before subjects habituated to Coriolis stimulation at higher rotation rates if the rate of rotation was increased by fixed steps (see Section 8.3 and Reason and Graybiel, 1970). More head movements were required to habituate to the change from 9 to 10 rpm than the change from 3 to 4 rpm. Reason and Graybiel (1970) recommend the percentage of total head movements appropriate for each rotation velocity from 2 to 10 rpm when using 1 rpm increments in rotational velocity. Using this information, Benson *et al* (1971) devised adaptation schedules in which the increment in rotation rate decreased as the rotation rate increased so that the number of head rotations required was similar at each new rate. They provided experimental support for the method for rotation rates from 3 to 5.95 rpm. Golding (1989) compared results obtained with fixed and variable increments as proposed by Benson *et al* (1971). He concludes that the findings from both methods are similar if the severity of the stimulus is expressed as the integral over time of the rotational velocity experienced (equivalent to the product of the number of head movements and the rotational velocity).

The Brief Vestibular Disorientation Test (BVDT) described by Ambler and Guedry (1966, 1968, 1971, 1978) and subsequently employed in many studies, used a fixed rotation rate of 15 rpm (90 degrees per second). After 30 s at constant velocity, a seated subject with closed eyes makes 45 degree head movements every 30 s in the order: right, upright, left, upright, right, upright, left, upright, forward, upright. On completion of the sequence (330 s) the motion is stopped and the subject opens his eyes after sensations of movement have ceased. Guedry (1968) says that the results of testing 500 student pilots with this test showed that observers judged that only 5% of subjects remained unaffected after six head movements. Ambler and Guedry (1971) show that the test could also be conducted with a lower rotation rate (10 rpm) with the benefit of less subject disturbance. Banta *et al* (1987) describe a modified version of this test: the subject is exposed to constant clockwise rotation at 18 rpm with eyes closed and the head tilts relative to the plane of rotation every 30 s for a total of 10 minutes or until the subject terminates the exposure due to symptoms of motion sickness.

Studies using variable rates of rotation include a series of experiments in which the responses of the autonomic nervous system reaction to motion sickness have been studied (e.g. Cowings *et al*, 1977, 1986, 1990). The procedure is similar to that proposed by Miller and Graybiel (1969): blindfold subjects are initially rotated at 6 rpm and incremented by 2 rpm every 5 minutes up to a maximum of 30 rpm. At 2 s intervals throughout each 5 minute period, head movements are made at 45 degrees in each of four directions in response to recorded voice instructions. A subjective scale of motion sickness severity was administered at the end of each five minute period while the head movements were stopped but the chair continued to rotate. The time to reach severe malaise (M· III), or the number of rotations to reach this degree of severity were used as the dependent variable. It appears that the exposure durations before reaching this endpoint ranged up to 55 minutes with a median of 19.5 minutes among a mixed group of 127 males and females (Cowings *et al*, 1986). Stott *et al* (1984) required subjects to make 45 degree head movements while exposed to a rate of rotation which increased at 0.1 deg.s^{-2} until 15 rpm was reached (after 15 minutes); this rate of rotation was then maintained for a further 5 minutes. Subjects who had not taken active drugs required the motion to be stopped after an average of 11 minutes.

There appears to be no particular virtue in using clockwise or anticlockwise rotation. Varying the direction of rotation has sometimes been used to reduce the chances of subjects habituating to the motions (e.g. Miller and Graybiel, 1970). Guedry (1965) exposed nine men to 10 rpm of counter-clockwise rotation in a slow rotation room for 12 days. The subjective effects (vertigo, nausea and stomach awareness) caused by head movements while exposed to counter-clockwise rotation decreased. One hour after the end of the 12 day continuous exposure, the effects of head movements while exposed to clockwise rotation were unchanged from those while exposed to clockwise rotation before the 12 day exposure to counter-clockwise rotation. Two days later, responses to both directions of rotation were

diminished, but 3 months later responses were similar to those before the 12 day exposure.

8.2.2 Effect of frequency, direction and angle of head movement

As the semicircular canals are not truly orthogonal and aligned in the pitch, roll and yaw axes of the head, the effect of roll and pitch movements of the head do not have equally provocative effects and the greatest effect is not produced by a 90 degree head movement.

Whereas the Brief Vestibular Disorientation Test (BVDT) uses 45 degree head movements at 30 s intervals, the Coriolis Sickness Susceptibility Index (CSSI) involves 90 degree head movements at 1 s intervals. In some tests the angles of head movement have been measured precisely whereas in many they have been based on loose verbal instructions.

Lentz (1976) described a chair in which headrests positioned the head either in a normal upright position - with the lateral semicircular canals in the plane of rotation - or with the head rotated 90 degrees forward and 45 degrees to the left so as to place a pair of vertical semicircular canals in the plane of rotation. This chair was used to study turning sensations (not to produce motion sickness). Subjects maintained their heads in one of the two positions throughout a 2 minute exposure to 18 seconds of acceleration (5 deg.s^{-2}), followed by constant velocity rotation (90 degrees.s^{-1}/ 15 rpm), followed by deceleration (at 5 deg.s^{-2}).

Taylor *et al* (1957) describe a test in which blindfold subjects were strapped in a supine position on a turntable rotating in the horizontal plane at 30 rpm. The head was moved mechanically through a vertical arc of 36 degrees and back with a cycle time of 24 seconds. The authors state that this test produced nausea and vomiting within 2 or 3 minutes in subjects unaccustomed to the motions of aircraft.

8.2.3 Effect of gravitational force

Lackner and Graybiel (1984, 1986b) found that the strength of symptoms induced by cross-coupled stimulation were dependent on the gravitational force. When this was reduced (during flight manoeuvres or in space) the stimulation was less effective. When the steady-state force field was increased the effects were increased. They suggest that the effect may be partially due to reduced effectiveness of semicircular canal function when there is reduced otolith signals. Additionally they suggest that the changes in motor control and sensory feedback required with different acceleration fields may be relevant.

8.3 Slow rotation room

Various studies have been conducted in which subjects have been placed in rotating rooms rather than on a rotating seat. 'Slow rotation rooms' 15 or 20 feet in diameter and 7 or 10 feet high allowed the provision of accommodation and the ability to expose subjects for periods of several days. The rooms were constructed to provide information for manned orbiting satellites. Graybiel (1969) said that rotation at 1 rpm gives little or no disturbance of vestibular origin but "*at 10.0 rpm it is comparable to exposure on rough seas*".

It has been found that there can be adaptation to the oculogyral illusion in a slow rotating room over a few days (Graybiel *et al*, 1961). There is evidence that habituation to motion sickness acquired while standing parallel to the axis of rotation transfers to the orientation with the subject at right angles to the axis of rotation (Graybiel *et al*, 1968). Graybiel *et al* (1969) found that incrementing the rate of rotation by 1 rpm per day up to 10 rpm resulted in no overt symptoms of motion sickness whereas sudden exposure to 10 rpm invariably led to motion sickness among subjects performing set tasks. Attempts to habituate to 10 rpm over shorter periods were unsuccessful. Using rotation rates of 7.5 and 20 rpm Graybiel (1970) found no difference in susceptibility to motion sickness between blind and normally sighted persons - although sighted persons tended to be more susceptible with their eyes open. Oosterveld *et al* (1972) found that 19 subjects were more susceptible to sickness when making head movements in a rotating room with eyes open, three were more susceptible with eyes closed and two were equally susceptible in the two conditions.

The slow rotation room has also been used to evaluate the effectiveness of some anti-motion sickness drugs (e.g. Wood *et al*, 1966; Wood and Graybiel, 1968, 1970; Graybiel *et al*, 1976, Graybiel *et al*, 1981). In a typical experiment, subjects seated away from the centre of rotation, were required to perform tasks requiring head movements in roll, pitch and yaw. In a control condition, the rate of rotation was increased (from 1 rpm up to 27 rpm in 1 rpm steps) until subjects experienced severe malaise (M III) within about 40 head movements. The number of additional head movements possible with this rate of rotation after the consumption of drugs was used as the measure of drug effectiveness. Graybiel and Knepton (1978) reported mixed success in providing habituation to motion sickness among a small group of aircrew in a rotating room.

9. OTHER COMPLEX MOTION CONDITIONS

The motions in many forms of transport are causes of motion sickness (see Table 1). In most transport environments there are motions in several axes of translation and rotation and the motion occurs at more than one frequency. For many environments the directions and frequencies of the motion primarily responsible for sickness have yet to be proven beyond reasonable doubt.

Seasickness is often primarily caused by low frequency vertical motion (see Griffin, 1991). However, the vertical motion is partially caused by the pitch motion of ships so the incidence of vomiting and illness is usually highly correlated with both the vertical

motion and the pitch motion of vessels. Since sea conditions which increase the pitch and the vertical motions of vessels also increase the motions in other axes, there can be statistically significant correlations between the incidence of motion sickness and the vessel motions in all axes. The traveller suffering from seasickness may not be able to identify the axis of motion causing the problem.

The 'ride' in road vehicles, and efforts to improve the comfort of person in road vehicles, is dominated by consideration of the effects of vibration at frequencies in excess of 1 Hz. However, there is no evidence that oscillation of the human body at frequencies greater than 1 Hz is a significant cause of motion sickness in such vehicles. The motions causing sickness must be at lower frequencies. It seems unlikely that vertical motion is normally sufficient to produce the high incidence of sickness that occurs in road vehicles. Accelerations in the horizontal directions are more likely causes of motion sickness but there are currently no dose-effect relationships.

The physical causes of airsickness have received less investigation than the physical causes of seasickness. The complex motions on aircraft vary greatly between aircraft and depend on the flight condition. It seems probably that some forms of airsickness are due to low frequency vertical oscillation, but dose-effect information is not available.

Various studies of motion sickness in gravito-inertial force fields other than 1 G have been undertaken. In studies during parabolic flight in aircraft it has been found that at zero G, movements of the head in roll, pitch or yaw increase motion sickness and that the symptoms are greatest with the eyes open (Lackner and Graybiel, 1984). Pitch movements of the head and trunk through 90 degrees with the eyes open had the most severe effects. Similar results were obtained when the study was repeated at 2 G but the symptoms tended to be more severe (Lackner and Graybiel, 1986a, 1987). 'Space motion sickness' is often provoked by head motion and was not a problem in small spacecraft which allowed little or no movement.

10. VISUALLY-INDUCED MOTION SICKNESS

Movement of a large visual scene gives the illusion of self-motion in the opposite direction. The illusion may be produced by translation of the visual field past the observer (linear vection) or rotation of the visual field around the observer (circular vection). These illusions may not, in themselves, cause sickness but the occurrence of other motions (especially head motions) while experiencing an illusory motion can be nauseogenic. Circular vection is more easily simulated and has been most studied.

10.1 Pseudo-Coriolis effect

The Coriolis effect occurs if, while the body is rotating about one axis, the head is rotated about another axis (see Section 8). Dichgans and Brandt (1973) used the term 'pseudo-Coriolis effect' to describe the similar consequences that arise if head movements are made during circular vection.

In the Dichgans and Brandt experiments subjects sat at the centre of a 1.5 m diameter cylindrical drum with its axis vertical and having inner walls with alternate white and black vertical stripes each subtending 7 degrees at the eye. (This apparatus is sometimes called an 'optokinetic drum'). Using 45 degree head movements to the right and left during rotation about the vertical axis at 15 rpm, they compared the number of head movements required to produce motion sickness in three conditions: rotation of the drum only, rotation of the chair only and combined rotation of the chair and drum. All three conditions produced sickness with the 'drum only' rotation tending to require most head movements and the combined rotation of chair and drum requiring the least head movements to cause sickness. The same sensations were experienced by subjects in all three conditions.

Lackner and Teixeira (1977) found little difference in the motion sickness symptoms experienced when subjects held the head stationary or moved the head from shoulder to shoulder while viewing within an optokinetic drum with rates of rotation increased incrementally from 1.66 to 13.33 rpm. There was a trend towards fewer symptoms when the head was held stationary. They report that symptoms were not the same as those experienced during Coriolis stimulation - possibly because the incremental changes in the rotation velocity resulted in only a few subjects experiencing the pseudo-Coriolis effect when moving the head.

Using randomly positioned black dots on the interior of a white sphere, Yang and Pei (1991) investigated the severity of motion sickness in seated subjects arising with various directions of sphere rotation (at 7.5 rpm) and head rotation (±20 degrees). During roll motion of the drum, head movements in roll reduced motion sickness while pitch and yaw head movements caused no significant change relative to the condition with no head movement. During pitch motion of the sphere, head movements in roll, pitch and yaw significantly reduced motion sickness symptoms. During yaw axis rotation of the sphere, head movements in roll and pitch significantly increased motion sickness symptoms while yaw axis head motions had no effect. The effect of head motion was most detrimental with yaw rotation of the sphere and most beneficial with pitch rotation of the sphere. This may be explained in terms of the strength of the illusory sensation of turning (circular vection) being strongest with yaw rotation of the sphere and weakest with pitch rotation of the sphere (see Section 10.2).

10.2 Circular vection

Various studies have produced motion sickness without head motions using a drum similar to that devised by Dichgans and Brandt.

Daunton *et al* (1984) compared the incidence of motion sickness in 27 squirrel monkeys exposed to constant speed rotation about a vertical axis with a view of the laboratory with the sickness that occurred within a rotating optokinetic drum. With a rotation rate of 10 rpm, 70% of animals retched or vomited when they were rotated, while 74% retched or vomited in the rotating optokinetic drum. At 25 rpm all animals retched or vomited when they were rotated and 81% retched or vomited within the optokinetic drum.

Using drum rotation at 10 rpm, Stern *et al* (1985) found that 14 out of 21 subjects developed symptoms of motion sickness in a 15 minute exposure. Five subjects requested the drum motion to be stopped before 15 minutes. Hu *et al* (1989) found that symptoms of motion sickness increased appreciably as the rotation rate of the drum increased from 2.5 to 10 rpm but fell when the speed was increased further to 15 rpm. Using a rotation rate of 10 rpm, Stern *et al* (1990) found that symptoms of motion sickness could be greatly reduced by restricting the subjects to a 15 degree circular field of view or providing them with a fixed visual target. Hu *et al* (1991a) found that exposure to a circular vection drum rotating at 2.5 rpm and 5 rpm for periods of 4 minutes reduced susceptibility to sickness during subsequent exposures to 10 rpm for 16 minutes. Using 10 rpm rotation of a circular vection drum 0.76m in diameter with alternating 5.7 degree black and 9.3 degree white stripes and a fixed head position, Hu *et al* (1991b) found that symptoms of sickness decreased over successive exposures of 16 minutes duration.

In studies reported by Doble *et al* (1987, 1989a, 1989b) most subjects tolerated appreciably less than 20 minutes of exposure to drum rotation at 10 rpm before symptoms of motion sickness resulted in withdrawal of exposure. Doble and May (1990) report mean tolerance times to this test in the range 3 to 6 minutes compared to mean tolerance times in the range 2 to 3 minutes for subjects given Coriolis stimulation (chair rotation at 10 rpm with 45 degree tilts in the frontal and lateral planes every 40 seconds).

In a study of optokinetic and vestibulo-ocular reflex responses to pseudo-random stimuli, Peterka *et al* (1987) found that no subject complained of sickness when the seated subjects were oscillated in yaw in a darkened room but 20% of subjects produced symptoms when a visual stimulus was rotated about the stationary subjects. The provocation was greater with the optokinetic stimulus even though the magnitude of the visual motion was half the magnitude of the rotation when the subject was oscillated. The two motions had similar, though not identical, spectra with all energy below 1.5 Hz.

Using randomly positioned black dots on the interior of a white sphere, Yang and Pei (1991) investigated the severity of motion sickness in seated subjects arising with roll, pitch and yaw rotation of the sphere at 7.5 rpm. The strength of the illusory sensation of turning (circular vection) was strongest with yaw rotation of the sphere and weakest with pitch rotation of the sphere. However, in the absence of head movements, symptoms of motion sickness were strongest with pitch rotation and least with yaw rotation. The findings are explained in terms of otolithic signals reducing vection for rotation about horizontal axes, at the expense of introducing 'conflict' between visual and vestibular information. Pitch head motions are assumed to be worse than roll head motions because pitch head motions are more frequent and so more closely associated with otolithic stimulation - consequently pitch rotation of the sphere produced the strongest conflict.

10.3 Other motions

From studies involving the swinging of cats and the rotation of monkeys, Daunton *et al* (1984) concluded that these animals could be made sick by visual stimulation alone and that animals most susceptible to optokinetic stimulation tend to be those most susceptible to motion sickness with bodily movement. The two optokinetic motions involved swinging a box (at 0.28 Hz) over stationary cats and placing stationary squirrel monkeys within a rotating optokinetic drum (see Section 10.2).

10.4 Distortion or degradation of the visual field

Benfari (1964) obtained subjective reports of motion sickness symptoms while subjects viewed four types of film projected with a wide angle lens within a 165 degree dome. It was found that the combination of peripheral flicker with a poorly structured visual field gave most symptoms of motion sickness. The presence of flicker alone or a poorly structured visual field alone were not sufficient to cause significant problems.

The wearing of left-right or up-down reversing spectacles while walking or making other voluntary or involuntary head movements can induce symptoms of motion sickness (e.g. Kottenhoff and Lindahl, 1960; Bock and Oman, 1982). Lackner and DiZio (1991) report that susceptibility to sickness produced by 0.5 Hz pitch head movements with visual inversion was less in a 0G environment (a parabolic flight profile in an aircraft) than in a terrestrial 1G environment. Ambulation while wearing left-right reversing goggles has been reported as considerably more nauseogenic than ambulation with up-down reversing goggles (Takahashi *et al*, 1991).

10.5 Simulator sickness

There are many reports of motion sickness in simulators, some of which may be attributed to distortion or other deficiencies of the visual simulation. The presence of inappropriate lags between subject responses and movements of the visual scene and the motion simulator may also be responsible.

11. OTHER NON-MOTION STIMULI CAUSING SICKNESS

Symptoms indistinguishable from some motion sickness symptoms can be produced by various non-motion stimuli. These include caloric stimulation of the outer ear, the excessive consumption of alcohol, the consumption of heavy water (deuterium oxide), some physiological conditions and physiological disorders, radiation and various drugs, including poisons. Some of these may also accentuate susceptibility to motion sickness.

12. DISCUSSION AND CONCLUSIONS

Motion sickness arises from a wide variety of physical stimuli. Experimental results provide a growing body of data from which predictions can be made. General theories of motion sickness offer qualitative explanations of why motion sickness occurs in each circumstance but they do not allow quantitative predictions of the physical characteristics of stimuli required for sickness. General theories do not indicate which of two stimuli will be more nauseogenic or provide predictions of the extent of sickness with any form of motion.

Head motion appears critical to the development of sickness with some motions. The failure to control, or adequately monitor, head motions in many experiments limits the interpretation of some results. The motion imparted to a person at the floor or seat may not be the same as that occurring at the head. The transmission of motion through the body may be attenuated or amplified and there may be phase lags. Motion in one axis at the seat (e.g. fore-and-aft) may induce other axes of movement at the head (e.g. pitch). The subject may make voluntary or involuntary movements of the head in response to the motions, with the extent of these depending on the type of restraint and the visual field. The interaction between these head motions and the imposed motion may be a cause of sickness.

Studies of the effects of head position and body orientation on sickness induced by the drug apomorphine, revealed that vomiting was far less when subjects were supine (Isaacs, 1957). The benefit was shown to arise from the supine posture of the body and not from the changed orientation of the head. While head movements may be the cause of sickness with some motion stimuli it does necessarily not follow that the often reported benefits of a recumbent posture in transport arise from the changed orientation of the head. These studies also suggested that the action of apomorphine was additive, or synergistic, with the sickness induced by vertical motion, horizontal motion and caloric stimulation.

Visual conditions are also critical to the development of motion sickness in some situations. Different degrees of sickness can arise with eyes closed, eyes open viewing a moving environment and eyes open viewing the outside world. When viewing the moving environment (e.g. the interior of a moving cabin) the effect may depend on the visual scene. A task which requires eye movements may have a particularly detrimental effect on subject well-being. The presentation of a visual scene which moves within a cabin (as with some simulators) may be expected to increase or decrease sickness depending on the eye and head movements that arise and how both the extent and the phase of movements of the projected visual scene correspond with movements of the cabin or the position of the external world.

Future research will extend the set of relevant experimental data. This will allow increased understanding of the effects of individual physical, physiological and psychological variables. It may then be possible to offer more confident predictions of the physical causes of motion sickness in specific environments.

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SIGNIFICATION OPERATIONNELLE DES CINETOSES POUR L'AIR, L'ESPACE ET LA SURVIE EN MER.

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RESUME: Sur un plan purement médical, le mal des transports ou cinétose ne pose généralement que des problèmes bénins dans la vie courante. En revanche, les opérations aériennes et spatiales donnent une toute autre dimension à cette affection relativement fréquente. De même, le syndrome de cinétose a une influence sensible sur les conditions de survie en mer.

La perspective historique issue des recherches menées lors du dernier conflit mondial permet de mieux situer le problème. Cette démarche conduit ensuite à analyser les relations complexes qui existent entre les cinétoses et la performance psychomotrice des opérateurs.

L'impact des cinétoses sur les opérations militaires aériennes comporte des aspects classiques, relativement bien étudiés sur le plan épidémiologique. Les problèmes liés à la formation initiale et à l'entraînement des pilotes constituent toujours un point important. De même, les cinétoses peuvent éventuellement contribuer à accroître la vulnérabilité de mise à terre des combattants héroportés. On relève par ailleurs des aspects nouveaux, résultant de l'évolution technologique des aéronefs et des systèmes. Ainsi, l'introduction des systèmes d'aide à la vision nocturne et du suivi de terrain automatique amène des conditions de vol propices au développement des cinétoses.

Le retentissement du mal de l'espace sur les opérations spatiales ne se pose pas du tout de la même façon selon que sont considérés des séjours courts (navette) ou longs (station spatiale habitable). Les résultats de l'exploitation du Système de Transport Spatial (STS) constituent la meilleure source de données sur ce sujet.

Enfin, les conséquences physiologiques et psychologiques du syndrome de cinétose, telles qu'aggravation de la déshydratation, apathie, sont les principaux facteurs à prendre en compte pour la survie en mer.

1. INTRODUCTION

Les conséquences opérationnelles des cinétoses ont été particulièrement étudiées dans la communauté militaire au cours du second conflit mondial et dans les années qui ont suivi. Pour comprendre cet intérêt, on peut citer un extrait du rapport de la première réunion du sous-comité US sur les cinétoses, d'après Reason (22):

> " The testimony of representatives of the Ground Forces, the Army Air Forces, and the Navy and reports from England and Canada suggested that the incidence of seasickness in amphibious training and combat operations had been high, resulting at time in serious losses of efficiency; that airsickness had become a problem of some magnitude in the selection, training and proper elimination of air force personnel particularly navigators, bombardiers and gunners; and that operations then anticipated, in which very large numbers of troops would be carried by air and by small surface craft, might be attended by a serious degree of incapacitation from motion sickness" Bard, 1948.

Ceci témoigne bien des préoccupations qui pouvaient exister à cette époque. La question que l'on peut légitimement se poser est: "qu'en est-il maintenant?". Le but essentiel de ce document sera donc de tenter de faire le point sur les aspects actuels de la signification opérationnelle des cinétoses.

Il faut bien se rendre compte que, dans la vie courante, l'apparition du syndrome de cinétose est certes déplaisante, mais que son retentissement sur la conception des moyens de transport et leur mise en oeuvre demeure généralement assez limité. On peut, par exemple, se demander quel sera l'impact des cinétoses sur la décision des passagers de choisir le bateau, l'avion ou le tunnel sous la manche pour se rendre au Royaume Uni, mais il est probable que cette question demeure du second ordre par rapport aux considérations tarifaires. On peut également voir certains constructeurs automobiles s'interroger, lors de la conception des véhicules, sur les interactions visuo-vestibulaires, toutefois, cette démarche demeure habituellement relativement ponctuelle. Il est pourtant bien connu que le "mal de voiture" constitue un fléau pour les parents de jeunes enfants mais qui , généralement et fort heureusement, affecte plutôt la moyenne horaire des déplacements que leur sécurité.

En fait, ces quelques réflexions anodines, associées aux considérations historiques, ont pour but de souligner que le problème de la signification opérationnelle constitue un moteur extrêmement puissant pour toutes les études menées dans le domaine des cinétoses. L'"impact sur l'efficacité des opérateurs et la sécurité des opérations, donc le coût humain et matériel, constitue une clé fondamentale de la progression des connaissances générales dans ce domaine (comme dans beaucoup d'autres d'ailleurs!!).

Ainsi, dans les années 60 à 70 la perspective des vols spatiaux et des problèmes d'orientation spatiale pouvant en découler a sans aucun doute largement contribué à l'essor important des études de physiologie vestibulaire, en particulier aux Etats-Unis.

Comme le note Benson (1), la perte de bien-être résultant du mal des transports, même modérée, peut constituer une source de problèmes pour un pilote dont la tâche est par avance complexe. Il faut cependant reconnaître que cette perte d'efficacité est souvent extrêmement difficile à quantifier, surtout chez des pilotes entraînés, habituellement peu portés sur l'introspection. De plus, les relations entre la performance, prise au sens général du terme, et les cinétoses ne répondent pas à des lois simples et sont connues pour être bidirectionnelles.

Après avoir brièvement évoqué l'historique du problème, il convient donc d'envisager les interactions qui existent entre cinétose et performance afin de poser les bases d'une réflexion sur la signification opérationnelle actuelle du syndrome.

2. PERSPECTIVE HISTORIQUE

Comme le souligne Reason (22), avec quelques anecdotes depuis Ciceron jusqu'à T.E. Lawrence, les cinétoses ont de longue date préoccupé la communauté militaire. Ces préoccupations ont très certainement culminé au cours du second conflit mondial, avec la nécessité de transporter de très larges contingents de troupes, appelés à être rapidement engagés dans des opérations de grande envergure. Ce souci, bien traduit par la citation de Bard présentée plus haut, concernait essentiellement l'entraînement aéronautique, les opérations maritimes et le transport de troupes. Les travaux effectués très tôt dans le cours du second conflit mondial ont surtout donné lieu à une estimation de l'incidence des cinétoses sans toujours répondre directement au problème de la signification pour les opérations. La revue de cette question figurant dans l'ouvrage de Reason (22) constitue une base de données particulièrement intéressante à cet égard.

Citons pour mémoire des chiffres concernant les opérations amphibies, qui vont de 11% par petit temps jusqu'à 60 à 70% par gros temps. Pour ce qui concerne les opérations aériennes, trois thèmes essentiels sont distingués, l'entraînement au pilotage, les opérations de combat aérien et les opérations aéroportées.

L'incidence pendant l'entraînement, rapportée dans les études du temps de guerre, est relativement modeste pour les pilotes (10 à 13% selon Hemingway), mais beaucoup plus importante pour les navigateurs (jusqu'à 65% selon une étude de McDonough publiée en 1943). Le retentissement opérationnel le plus évident des cinétoses lors de l'entraînement en école de pilotage est le taux de disqualification qui peut leur être attribué. Selon Birren (1949), ce taux varie en fonction des sources d'information entre 0,15 et 6,5 %. D'une manière plus qualitative, des sources alliées diverses (Afrique du Sud, Etats-Unis, Australie,...) font état du problème qui semble particulièrement aigü pour les navigateurs.

Beaucoup moins de données sont disponibles pour les opérations de combat aérien. Les estimations varient entre 5 et 19 % selon les sources, mais l'interférence des cinétoses avec l'exécution des tâches opérationnelles est habituellement relatée comme très basse, puisque ne dépassant pas 0,3 pour 1000 homme/mission. Il existe par ailleurs des interactions reconnues entre les conditions du combat et la performance pouvant participer à ces taux relativement bas par rapport à l'incidence globale du syndrome.

Les études du temps de guerre font une place importante au problème du transport de troupes par voie aérienne. (Tyler et Bard, 1949; Birren, 1949; Hemingway, 1945; Chinn et Smith, 1953). Tous s'accordent pour constater que l'incidence des cinétoses dans les troupes aéroportées est particulièrement élevée lors des phases de transport tactique. On relève ainsi des incidences de l'ordre de 25 à 80 % selon le type de vol. En revanche, il ne semble pas exister de mention particulière pour l'interférence avec les opérations de combat immédiatement après l'aéroportage.

En fait, dans l'ensemble de ces études de terrain en temps de guerre, l'influence des cinétoses sur la performance des combattants, en dehors des phases aigües du syndrome, semble extrêmement difficile à quantifier, ce qui est assez facilement compréhensible. Cette constatation amène donc à considérer les études de terrain ou de laboratoire qui ont été spécifiquement consacrées aux interactions entre les cinétoses et la performance de l'opérateur.

3. CINETOSE ET PERFORMANCE

Plusieurs revues de la littérature abordent les relations existant entre cinétoses et performance (11, 20, 22). Comme Reason (22), on peut également évoquer l'aspect bidirectionnel des interactions entre cinétoses et performance, à savoir les effets des cinétoses sur la performance et les effets de la performance sur le syndrome de cinétose.

Pour ce qui concerne les études de terrain, la plupart se sont intéressées aux cinétoses provoquées par la navigation maritime, mais quelques résultats sont disponibles pour des situations aéronautiques. Il existe par ailleurs de nombreuses études de laboratoire consacrées à l'effet de diverses stimulations provocatrices sur la performance psychomotrice et intellectuelle. Comme le souligne Money (20), les études de laboratoire introduisent un biais inévitable dans l'évaluation de la performance, car le sujet est conscient d'être observé et connaît généralement la durée de l'épreuve. Ceci est donc fortement susceptible de modifier son comportement et sa motivation par rapport à une situation opérationnelle prolongée. Ces deux aspects de l'évaluation de la performance seront successivement abordés.

3.1. Etudes expérimentales de terrain

Bien qu'obtenues avec des stimulations et un environnement relativement éloigné de ce qui est rencontré en aéronautique, les données provenant des études à la mer sont intéressantes à considérer. Des résultats anciens avaient déjà souligné la différence entre la performance de routine et la performance de pointe. Ceci signifie que, même étant en proie au syndrome, un individu qui trouve une motivation suffisante dans l'accomplissement de sa tâche est capable de l'effectuer pratiquement sans dégradation. Les dégradations observées sur différents tests de performance sont essentiellement présentes dans les premières heures d'exposition à l'environnement. On a ainsi pu observer une diminution des performances sur des tâches d'extraction de données, de raisonnement grammatical et de tracking et, d'une manière plus prolongée, une dégradation de capacités de tracé de la route (tâche de navigation). Ce type de résultats est retrouvé dans plusieurs études (11), en condition réelle ou en simulation.

Parmi les rares études réalisées en environnement aéronautique, il faut citer le travail de Kennedy et coll. dans des vols de pénétration de cyclone (14). Des dégradations significatives de la performance ont ainsi été observées sur une tâche de vigilance auditive.

Certaines études ont également été menées en milieu spatial, mais rarement avec des données quantitativement établies. Ainsi, Reason (22) rapporte certains éléments d'analyse subjective sur la performance de cosmonautes soviétiques montrant une réduction de la performance en vol et un accroissement de la difficulté à effectuer les tâches demandées. Hettinger (11) mentionne des résultats expérimentaux récents révélant une altération du temps de réaction et de la capacité d'estimation du temps, semblant liés à l'existence d'épisodes de mal de l'espace (21).

3.2. Etudes en laboratoire

Les études sur la dégradation de la performance réalisées dans de sconditions de laboratoire sont extrêmement nombreuses. Au regard des différents environnements utilisés, les études classiques de Wendt sur l'effet des accélérations verticales, ainsi que celles qui ont suivi sur ce type de stimulation, ont largement contribué à augmenter les connaissances dans le domaine de la performance. La chambre de rotation lente de Pensacola tient également une place particulière quant à la qualité et à l'étendue des expérimentations qui y ont été menées. Enfin, d'autres environnements rotatifs (orientés sur les études en milieu spatial) ont été utilisés (11). Il faut aussi citer d'assez nombreuses études en simulateur, dont certaines liées à la simulation de combat aérien (15).

Effectués à l'issue d'épreuves provocatrices, différents tests physiques et de coordination motrice, comme la course, le lancer de fléchettes, le tir à la carabine, ne sont révélés non significativement affectés par le syndrome de cinétose. En revanche, surtout dans la phase aigüe de l'exposition aux stimulations nauséogéniques, différents tests de performance intellectuelle ou psychomotrice se sont révélés altérés. C'est le cas pour des tâches de calcul mathématique (bien que cec soit controversé), d'estimation du temps et pour différentes tâches de poursuite ou de tracking. On note aussi l'existence de dégradations lors d'épreuves mettant en jeu la vigilance auditive.

3.3. Effet de la performance sur les cinétoses

Il existe un certain nombre de données, la plupart anecdotiques, qui établissent un lien entre la nécessité vitale d'accomplir une tâche et la sévérité du syndrome provoqué par des stimulations nauséogéniques. Il est habituellement observé une forte diminution, voire une disparition complète des symptômes, lorsque le sujet se trouve brusquement dans une situation critique ou exerçant une forte demande sur le plan psychomoteur. Plusieurs études expérimentales effectuées sur ce sujet (22), tendent à montrer que les tâches dirigeant l'attention vers le monde extérieur diminuent la cinétose, alors que la concentration sur les symptômes a plutôt tendance à les accroître. La question est cependant loin d'être totalement tranchée, puisque Lentz (19) observe un accroissement de la sévérité du syndrome provoqué par un test d'interaction visuo-vestibulaire en fonction de la complexité de la tâche visuelle.

4. ASPECTS ACTUELS

L'ensemble des données issues des observations effectuées en situation de guerre et les diverses études sur l'effet des cinétoses sur la performance permettent d'établir qu'il existe un risque potentiel, tant au niveau des opérations militaires que dans le domaine spatial.

Dans le premier cas, c'est véritablement le problème de l'efficacité, en terme de réussite et de sécurité des opérations, qui est en jeu dans des situations où la survie du combattant va dépendre de sa capacité à mettre en oeuvre des armements de plus en plus complexes et coûteux. Dans le second, le problème n'est habituellement plus aussi vital, mais, compte tenu du coût extrêmement élevé des opérations spatiales, il s'agit plus d'un aspect de rentabilité que de sécurité. Il faut cependant noter que, dans les deux cas, on se trouve bien confronté avec un problème qui, sur le plan de la signification opérationnelle, implique la notion du coût humain et matériel des cinétoses.

En gardant en mémoire les données existantes, il convient maintenant d'effectuer une analyse du problème de la signification opérationnelle des cinétoses dans le contexte aéronautique et spatial actuel. Les caractéristiques propres aux opérations aériennes, au vol spatial et aux problèmes soulevés par la survie en mer seront successivement abordées.

4.1. Opérations aériennes

Dans le cadre général des opérations aériennes, il convient de distinguer des aspects classiques, comme l'entraînement et le transport aérien tactique, mais aussi des éléments

nouveaux liés au contexte de l'évolution des armements et des techniques de combat et d'entraînement.

Les deux premières questions peuvent être abordées comparativement à des situations connues antérieurement. En revanche, les éléments nouveaux, comme le suivi de terrain automatique ou les problèmes liés à la visionique de casque, doivent être évoqués en terme de perspectives et de potentialité, puisque, en fait, il existe très peu de données disponibles sur ce sujet.

4.1.1. Apprentissage et entraînement

Traditionnellement, l'école de pilotage a toujours présenté un contexte privilégié pour le développement des cinétoses. A quelques différences près, liées à l'évolution des matériels et des techniques d'entraînement, ceci reste relativement exact. En fait, à l'heure actuelle, la signification opérationnelle des cinétoses en école est sans doute à considérer en termes de coût direct et indirects, mais aussi relativement à la situation vis à vis du recrutement des élèves-pilotes.

Parmi les études modernes concernant l'incidence des cinétoses lors de l'entraînement au pilotage, les résultats rapportés par Reason en 1968 (22) donnent une estimation très élevée, puisque, selon cet auteur, 76 % d'un échantillon d'élèves-pilotes de Cranwell a souffert du mal de l'air pendant l'entraînement au vol. Dans ce groupe, 18 % ont été atteints au point d'interrompre prématurément un vol.

En 1972, Leguay et Coll. rapportent, d'après une enquête effectuée dans une école de l'Armée de l'Air Française, une incidence globale de 30 %, seulement 5 % des élèves ayant consulté le médecin du Personnel Navigant (18). A la même époque, Dowd (5) émet le jugement que les cinétoses demeurent encore un problème très important dans l'US Air Force, particulièrement dans les manoeuvres d'avions à haute performance. Il cite les résultats de Tucker (1965), rapportant que 10 à17 % des élèves-pilotes ont été atteints au moins une fois durant leur entraînement, 5 % d'une manière répétitive, avec un taux d'élimination dû aux cinétoses de 0,7 %.

Dobie (4), en se fondant sur les commentaires des pilotes instructeurs portant sur une population de 577 élèves, détermine une incidence de 24,1 % de mal de l'air modéré et de 14,6 % de mal de l'air sévère. Il est intéressant de considérer que, dans cette étude, le jugement de sévérité du syndrome était établi en tenant compte de la dégradation des performances observée durant le vol. L'auteur souligne que cette estimation est sans doute assez prudente, puisque l'incidence des malaises légers ou même modérés a pu être sous-estimée du fait de la méthode d'évaluation employée. D'un autre côté, les chiffres résultant de cette étude ont une valeur importante sur le plan de la signification opérationnelle des cinétoses, puisque évaluant directement l'effet sur la performance. En 1977, avec une méthodologie relativement identique, Galle-Teyssoneau (8) estime que 30 à 50 % des élèves-pilotes souffrent du mal des transports à des degrés divers durant leur entraînement en école. Cependant seulement 5 élèves sur 492 ont été éliminés pour mal de l'air, soit 1 %, alors que le taux d'échec global du cours est de 33 %. L'auteur note toutefois que parmi les 40 élèves qui ont consulté pour mal de l'air, le taux d'élimination (toutes causes confondues) atteint 70 %. Le chiffre de 1 % rejoint également le taux d'attrition avancé par Tribel en 1972, sur la base de 400 élèves-pilotes de la Force Aérienne Belge (251).

Enfin, pour terminer cette revue des données récentes sur l'incidence des cinétoses lors de l'entraînement, notons que Hixson et coll. (12) ont rapporté les résultats d'une enquête longitudinale portant sur une population de 796 élèves, à partir des données collectées lors de 28.483 sorties effectuées sur différents types d'aéronefs pendant le cours d'Officier de l'Aéronautique Navale (personnel navigant non-pilote). Selon le type d'appareil et d'exercice, l'incidence des cinétoses va de 9 à 23 %. Les vomissements surviennent dans 4 à 11 % des cas et la performance est considérée comme significativement dégradée dans 3 à 15 % des cas.

L'examen de ces chiffres montre bien que le problème posé, en temps de paix, par les cinétoses dans les écoles de pilotage est loin, dans l'absolu, d'être catastrophique, du moins pour ce qui concerne le taux d'attrition qui peut leur être directement lié. Le coût direct, sur la base des échecs et des délais dans la progression, demeure sans doute également relativement modéré, bien que, généralement, il ne soit pas clairement indiqué. On peut évoquer cependant le coût indirect des cinétoses en école qui consiste en l'élimination d'individus qui, s'ils avaient surmonté leur handicap, auraient sans doute pu être d'excellents pilotes, comme en témoignent les résultats obtenus par Stott et Bagshaw (23). On peut ainsi considérer qu'à terme ces éliminations, même peu nombreuses, pourraient conduire à un relatif appauvrissement de la qualité des pilotes qui, par ailleurs, doivent également répondre à beaucoup d'autres critères physiques et intellectuels.

Ceci amène à considérer le problème dans la perspective des possibilités de recrutement. Dans un pays comme la France, le prestige attaché à la carrière de pilote militaire, en particulier celle de pilote de chasse, est encore très grand. En conséquence le nombre de candidats souhaitant accéder à cette carrière est très élevé et tend même à s'accroître assez régulièrement. Tant que cette situation durera, le problème des cinétoses peut être considéré comme relativement mineur. En revanche lorsqu'une pénurie de pilotes est rencontrée, résultant, par exemple, d'une attrition élevée de pilotes qualifiés liée au différentiel de rémunération avec les carrières civiles, alors il devient sans doute plus intéressant de tenter de réduire les éliminations en école de pilotage.

Pour être complet, il faut par ailleurs envisager le contexte particulier du temps de guerre. Dans un conflit majeur on peut supposer la survenue de pertes élevées, vraisemblablement difficiles à combler rapidement. En effet, la complexité des avions de combat modernes implique un entraînement relativement long et, à supposer que le matériel puisse être remplacé rapidement, on peut douter que le mécanisme de la formation en école puisse

jouer efficacement et que le mal de l'air y ait un rôle significatif.

4.1.2. Vulnérabilité de mise à terre

La mise à terre de troupes aéroportées constitue également un contexte favorable au déclenchement des cinétoses. Les conditions du transport précédant le largage sont bien sûr la source essentielle de problèmes rencontrés. Pour échapper à la détection des radars et bénéficier de l'effet de surprise nécessaire à la réussite de l'opération, les appareils de transports tactiques sont amenés à évoluer à très basse altitude, pendant des durées relativement longues (1 à 2 heures). Dans ces conditions le taux de sujets souffrant de cinétose peut atteindre 80%, comme en témoignent les résultats expérimentaux obtenus par Gaiban et coll (7).

Il est, en général, relativement difficile de chiffrer l'impact exact du mal de l'air subi durant le vol sur l'efficacité des combattants au sol. Compte-tenu des observations du temps de guerre et des résultats expérimentaux sur la performance et les cinétoses, on pourrait penser que l'effet direct demeure relativement modéré. Il convient cependant de pousser la réflexion plus loin. En effet, les opérations de transport de troupes que l'on peut attendre du contexte hautement technologique du combat moderne en Europe sont relativement peu propices aux très grands mouvements se soldant par des "vomissements de masse" historiques.

En revanche le déplacement des menaces à l'extérieur du théâtre européen pose le problème de l'intervention ponctuelle sur un point chaud de troupes d'élite amenées à engager le combat très rapidement après leur mise à terre. Il ne s'agit plus là de l'effet isolé du mal des transports après un vol tactique de quelques heures, mais bien de celui du stress combiné (fatigue d'un vol prolongé, perturbations circadiennes, changement de climat) où les cinétoses peuvent contribuer à augmenter la vulnérabilité de mise à terre.

4.1.3. Eléments nouveaux

Par rapport aux données classiques sur l'incidence des cinétoses dans les opérations aériennes et leur signification éventuelle, plusieurs éléments nouveaux sont à considérer.

En premier lieu, aussi bien les machines que les systèmes d'armes et les tâches accomplies par les équipages sont devenus extrêmement complexes. D'une manière corollaire, cette complexité est associée à un coût très élevé des aéronefs de combat et de la formation des équipages. La formation d'un pilote opérationnel constitue un investissement très important, tout comme l'appareil qu'il doit mettre en oeuvre.

Outre ces considérations, de nouvelles conditions d'emploi sont apparues, hautement susceptibles de provoquer l'apparition du syndrome de cinétose , même chez des pilotes adaptés aux évolutions aériennes des avions de combat modernes. Ce sont en particulier les effets du suivi de terrain automatique et les problèmes liés à l'utilisation des dispositifs d'aide à la vision nocturne. Or, il apparaît que dans la population de pilotes de chasse (16), au moins 25 % des pilotes normalement adaptés ont une susceptibilité de base qui peut les rendre sensibles à des conditions de vol inhabituelles.

Le suivi de terrain automatique comporte deux types de caractéristiques propices au déclenchement du syndrome de cinétose. Tout d'abord, le pilote n'est plus directement en contrôle des évolutions de l'avion et se trouve donc dans une situation de passager, d'autre part, les évolutions de l'appareil, constituées typiquement de successions d'accélération Gz positives et négatives et de virages, sont par elles-mêmes hautement susceptibles de provoquer l'apparition du syndrome. Ces deux conditions étant réunies, on peut donc penser que l'apparition insidieuse du mal de l'air chez un pilote opérationnel, ayant par ailleurs une tâche complexe à effectuer, constitue une réelle menace pour la qualité et la sécurité de la mission, du moins jusqu'à ce qu'une habituation à ces conditions de vol ait pris place. En fait, en dehors de rapports de pilotes ayant effectué les essais en vol de ce type de système et des données anecdotiques, ce problème ne semble pas réellement avoir été documenté d'une manière sérieuse.

En revanche, il existe quelques éléments concernant les dispositifs d'aide à la vision nocturne. Ainsi, Cornum et coll. (3) ont rapporté l'existence de problèmes de cinétose dans l'entraînement et l'utilisation opérationnelle des systèmes d'aide à la vision nocturne de l'hélicoptère d'attaque AH-64 "Apache". Les mécanismes en cause semblent être l'existence de problèmes de parallaxe, mais on peut également supposer que la restriction du champ de vision entraînée par l'utilisation des systèmes peut également jouer un rôle dans l'apparition des symptômes. La signification opérationnelle de ces troubles, en dehors de leur incidence sur l'entraînement, est encore mal connue. On peut cependant supposer que l'existence du syndrome dans des conditions opérationnelles peut affecter sérieusement aussi bien l'efficacité que la sécurité d'une mission d'attaque nocturne, déjà difficile en elle-même.

Il convient enfin d'évoquer un dernier point relativement nouveau qui est l'utilisation intensive des simulateurs en particulier des simulateurs de combat aérien. De nombreuses études ont montré que l'incidence du mal des simulateur était relativement élevée (2, 10, 15, 17). Pour Kennedy(15), ce problème est susceptible d'entraîner une certaine répugnance à utiliser ce type de simulateurs. Il ne semble toutefois pas que les effets secondaires des simulateurs de combat puissent être effectivement impliqués dans des problèmes d'efficacité ou de sécurité. Il s'agit cependant d'un problème suffisament significatif sur le plan opérationnel pour que le commandement s'en inquiète, tant pour ce qui concerne l'efficacité de l'entraînement que son organisation.

4.2. Opérations spatiales

Le retentissement opérationnel du mal de l'espace sur les opérations spatiales constitue un point d'intérêt important, tant pour les implications de "rentabilité", si l'on peut employer ce terme, que pour la sécurité. Cet intérêt a été

reconnu assez tôt dans le développement des missions spatiales habitées et a directement ou indirectement motivé de nombreuses études.

Les données connues à ce jour font état d'une incidence voisine de 50 %, aussi bien au cours des vols spatiaux américains que soviétiques (25, 6). Il existe des doutes quant à la sévérité respective des syndromes présentés par les astronautes américains et les cosmonautes soviétiques. Pour ces derniers, l'entraînement vestibulaire intensif auquel ils sont soumis avant le vol pourrait contribuer à réduire la sévérité du malaise provoqué par l'adaptation aux conditions de microgravité. En fait, si l'on peut avoir une assez bonne idée de l'incidence globale du mal de l'espace dans les équipages, la sévérité des symptômes endurés est un point beaucoup plus délicat à évaluer. Pour les opérations de la navette spatiale américaine, une estimation courante donne l'existence d'un malaise sévère et prolongé (donc relativement incapacitant) chez un astronaute tous les trois vols.

La signification opérationnelle du mal de l'espace, si l'on s'en tient aux incidents qui ont été relevés au cours de vols spatiaux, n'est pas extrêmement évidente à évaluer. Homick (13) a récemment exposé l'état de l'art existant dans ce domaine et les implications entraînées pour la conduite des opérations de la navette américaine. Plus qu'une incapacitation totale, le manque d'initiative des sujets atteints peut représenter un problème. Les observations effectuées par Thornton (24) montrent que les sujets atteints accomplissent généralement correctement les tâches qui leur sont assignées, mais ont tendance à se limiter strictement à l'indispensable. En fait, il s'installe naturellement une sorte de compensation, au sein de l'équipage, de l'activité des plus atteints, qui aboutit à l'accomplissement de l'ensemble des objectifs et des tâches prévues (reschke).

Sur le plan pratique, on relève un certain nombre de cas où le mal de l'espace a interféré directement avec des activités opérationnelles programmées. Ainsi, lors du programme Apollo, plusieurs incidents mineurs ont été notés dont le plus significatif, lors du vol Apollo 9, a entraîné un report d'une journée dans le programme. On note également que l'équipage de Skylab 3 a été amené à réduire significativement ses activités pendant les 36 premières heures du Vol. Pour ce qui concerne les opérations du Système de Transport Spatial (STS), divers incidents ont également été rapportés, allégement du programme de STS-3, report d'une sortie extra-véhiculaire lors du vol STS-5 (13). Du côté Soviétique, les données sont moins claires, mais Reason (22) rapporte qu'en 1967 une mission soviétique pourrait avoir été annulée en raison d'un problème de mal de l'espace.

Un élément d'importance dans le problème de la signification opérationnelle du mal de l'espace est la durée des opérations envisagées. Il faut bien être conscient que les éventuelles difficultés rencontrées surviennent dans les premières heures du vol Spatial (26) et que dans la majorité des cas l'habituation neurosensorielle à la microgravité est acquise dans les premières 72 heures du vol. Ainsi, en dehors de cas particuliers, comme les incidents de transfert de l'équipage d'un vaisseau vers une station orbitale, le mal de l'espace pose relativement peu de problèmes dans les séjours de longue durée. En revanche, les vols courts comportant un programme très chargé sont particulièrement susceptibles d'être affectés sur le plan opérationnel. Il faut cependant reconnaître que, sur beaucoup de plans, les premières heures passées en impesanteur sont généralement les plus critiques. En cas de circonstances véritablement exceptionnelles mettant en jeu la sécurité des opérations dans les 36 premières heures de séjour, on peut toujours légitimement se poser la question de l'impact que pourrait alors avoir le mal de l'espace.

4.3. Survie en mer

Il est notoire que les conditions de survie en mer sont particulièrement propices au développement des cinétoses comme le souligne Reason, citant Liano (1955), avec des chiffres de 60 % d'incidence dans les équipages abattus en mer. Sans fournir de données statistiques précises, il avance que, dans cette situation, le mal de mer pourrait bien avoir contribué à la mort en hâtant le processus de déshydratation.

L'éventualité d'observation d'éjections en mer lors d'opérations combinées aéromaritimes ou aéronavales est loin d'être négligeable. Dans ces conditions, l'état de la mer va constituer un point important dans les chances de survie des personnels. La recherche sera d'autant plus difficile et selon toute probabilité longue que les conditions de mer seront mauvaises. Les engins de sauvetages individuels sont généralement statiques et se comportent somme de véritables bouchons sur la mer, ce qui favorise considérablement l'apparition du mal de mer. Les conséquences sont en général triples (9): retentissement sur les besoins hydro-minéraux, atteinte du psychisme, aggravation des états pathologiques intercurrents.

L'aboutissement du mal de mer est l'apparition de vomissements incoercibles qui augmentent de façon considérable les pertes hydro-minérales. Le problème de l'équilibre hydro-minéral dans la survie en mer est dans toutes les conditions extrêmement critique du fait du rationnement de l'eau. La déshydratation avec hémo-concentration est aggravée, dans le cas des vomissement par les pertes ioniques qui peuvent aboutir à l'installation d'une alcalose métabolique.

Sur le plan psychique, il est bien connu que les cinétoses sévères entraînent une véritable prostration avec une perte plus ou moins importante de l'état de conservation. Il est reconnu que la volonté de survivre constitue un élément important dans les situations de naufrage maritime et l'apparition du syndrome de cinétose, en dehors de ses répercussions physiologiques, est toujours extrêmement défavorable. D'autre part, le repérage d'une embarcation monoplace dans un environnement hostile constitue une tâche difficile et la coopération du naufragé constitue souvent un élément décisif. L'apathie engendrée par le mal de mer peut donc se révéler critique dans un moment décisif pour la réussite de la mission de sauvetage.

Enfin, il ne faut pas oublier qu'un pilote peut être blessé au cours d'un abandon de bord et que son état ne peut alors qu'être aggravé par l'apparition du mal de mer et de ses effets débilitants.

5. CONCLUSIONS

L'ensemble des données de la littérature et des perspectives découlant des éléments nouveaux liés à l'évolution des matériels et des tâches des équipages de l'aéronautique militaire permettent de situer les conséquences opérationnelles des cinétoses dans le contexte actuel des opérations aériennes. et spatiales.

L'incidence des cinétoses en école de pilotage semble relativement stable et son impact sur la progression des élèves est relativement bien maîtrisé. Le réel problème est celui de l'effort qu'il faut consentir pour améliorer le taux d'attrition lié au mal de l'air. La nature de cet effort est à l'heure actuelle étroitement liée aux possibilités de recrutement et à la philosophie générale adoptée.

La survenue de manifestations de cinétose chez des pilotes opérationnels mettant en oeuvre des systèmes d'armes et des missions complexes constitue sans aucun doute une situation préoccupante. Ce point mérite très certainement une surveillance particulière, le suivi de terrain automatique et l'utilisation de visionique montés sur le casque du pilote et de dispositifs d'aide à la vision nocturne comportant des éléments particulièrement propices au déclenchement des cinétoses.

L'incidence opérationnelle du mal de l'espace constitue toujours un point d'intérêt soutenu. Si les faits montrent que dans beaucoup de cas les conséquences neurosensorielles de l'adaptation à la microgravité demeurent modérées, les risques potentiels dans des situations critiques survenant dans les premières heures du vol persistent La durée des vols constitue un élément d'importance pour le retentissement sur les opérations.

Enfin, les conséquences des cinétoses sur la survie en mer sont bien connues et représentent toujours une menace importante pour le devenir des équipages contraints à l'abandon de bord lors de survol maritime.

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Factors Influencing Susceptibility: Individual Differences and Human Factors

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SUMMARY

From a conceptual viewpoint, individual differences in susceptibility to motion sickness are determined by differences in: initial reactivity (receptivity), ability to adapt to the motion, ability to retain the adaptation during abstinence periods, and ability to reinstate adaptive responses upon re-exposure to the motion. From a theoretical viewpoint, the adaptive adjustment involves alteration of the timing, magnitude, and direction of sensorimotor reactions so as to increase the efficiency of postural control in the motion environment. However, the threshold linking the sensorimotor adaptive process to the processes that set-off the signs and symptoms of motion sickness may also be an individual characteristic. A conceptual model is presented to organize the discussion of individual differences in motion sickness susceptibility. Factors that have been reported to influence incidence of motion sickness such as age, mental activity, anxiety and fear, perceptual style, physical fitness, active control of the inducing motion, concomitant visual stimulation, quality of the initial exposure to the motion environment and conditioned motion sickness are discussed.

INTRODUCTION

Motion sickness is a significant problem in every major form of modern transportation, including space travel (Homick, 1979, Homick et al., 1984). In passengers, its effects range from mild discomfort to severe disturbance, and in crew, effects range from lowered enthusiasm for work to incapacitation for any useful activity. Extreme prolonged sickness is a threat to survival. In flight training, air sickness is a significant cost due to increased training time and attrition of some otherwise well-qualified individuals. Operational significance of motion sickness is reviewed by Leger (1991) in this volume.

Motion sickness is a normal response to an abnormal motion environment, and it is likely that anyone with a functional vestibular system can be made sick by any of several forms of provocative motion. However some people succumb to provocative motions much more readily than others, and different environmental and personal conditions can increase incidence of sickness engendered by a given motion stimulus. This chapter deals with individual differences in motion sickness susceptibility and with factors other than the motion itself that alter the sickness incidence. The chapter by Griffin (1991) deals with characteristics of motion that influence sickness incidence.

The Scope of Individual Differences

The magnitude of individual differences in reactions to provocative motion can be clearly appreciated by exposing each of 20 flight students to a sequence of head tilts during whole-body rotation at a speed of 15 rpm. It is probable that one subject will describe the experience as "absolutely dreadful," to use the scale of Lawther and Griffin (1986), while another will find the experience interesting and not disturbing as indicated by requests for repetition of the experience with stronger stimuli. Among a group of individuals who have chosen a career in aviation, similar in age and background, a few abhor and are visibly incapacitated by a motion stimulus that a few enjoy. The immediate reaction to such a stimulus foretells subsequent sickness if the exposure is continued, although prediction from initial reactions is far from perfect. Nevertheless, immediate reactions are significantly correlated with subsequent problems in aviation (Lansberg, 1960, Ambler and Guedry, 1966).

Immediate reactions to provocative stimuli fail to reveal the scope of important dimensions of differences in reactions to motion. Some individuals recover very slowly from a given level of sickness while others recover more quickly. A very important category is chronic sickness where

despite prolonged and repeated exposure, severe sickness continues. This category is most easily identified in seasickness, where ample opportunity to adapt fails to yield improvement. While true members of this class are rare, they should be studied in detail to answer some questions that will arise in this chapter.

Nature-Nurture

From earlier considerations of mechanisms of motion sickness (Guedry, 1991), we infer that motion sickness susceptibility (MSS) would be dependent upon the individual's history of exposure to unusual motions but if individuals are somehow intrinsically different initially, then not only will they have controlled, to some extent, their exposure histories but their current state of adaptation will depend upon intrinsic differences in adaptive capacity and adaptation retention. Thus, immediate MSS of individuals involves the classical nature/nurture problem of understanding human behavior. Because motion sickness is a specific and fairly measurable aspect of behavior and because exposure history is also fairly measurable, advances in the understanding of MSS may also provide an approach to the greater problem of understanding human behavior (Mirabile, 1990).

MOTION SICKNESS: AN ENDURING TRAIT THAT CAN BE CHANGED

Based on the conceptual model presented earlier, mismatch between perceived and expected spatial orientation reactions initiates two processes - one that alters the adaptive state of the individual and another accumulation process that leads to the sickness syndrome. In turn, the adaptive state of the individual depends upon 1) history of motion exposure and 2) how the individual reacted to and has been shaped by the motions experienced. Figure 1 depicts factors influencing individual motion sickness susceptibility to a particular motion of stimulus. Adaptation achieved in a motion environment depends upon the activity of the individual in that environment - what the individual has been required to do or wants to do and tries to do. Adaptation to aviation in individuals whose exposure predominantly involves piloting and learning to pilot the aircraft differs from adaptation developed by individuals whose exposure, though equivalent in hours, is that of a passenger. Pilots sometimes become sick when they are in the back seat. History of exposure is thus not a simple accounting of type and number of hours of exposure. It is complicated by the fact that the individual partially determines the history and the history changes the individual.

In addition to the individual's long term motion adaptation capabilities, other personal factors can alter immediate susceptibility to a particular motion environment. Examples of temporary

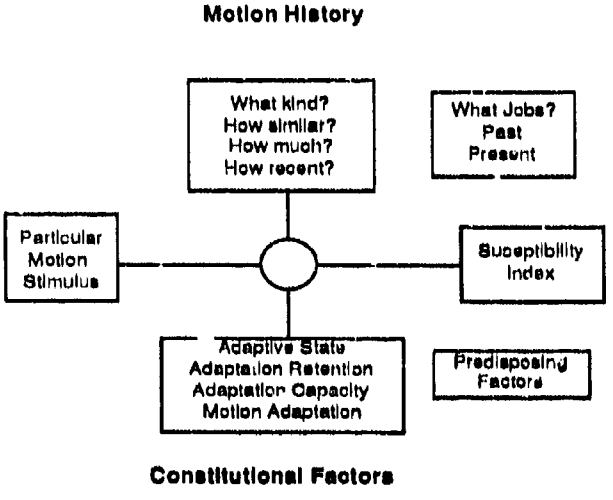

Figure 1. Factors in individual susceptibility to sickness with particular motion stimuli.

predisposing factors are temporary gastrointestinal upset, inner-ear inflammation, alcohol ingestion, headache, and perhaps severe emotional involvement in family matters. More enduring predisposing factors include the psychological characteristics that determined the individual's selection of past motion environments, individual adaptive strategy and effort to perform within the environment, and perhaps individual fear of the environment itself.

Evidence that motion sickness is an enduring trait has led to much speculation about personal characteristics that cause this "weakness." The evidence consists of the fact that individuals who become sick with one kind of provocative motion also tend to become sick with other kinds of provocative stimuli, i.e., susceptibility tends to be constant over different motion conditions (Graybiel and Lackner, 1983, Crampton and Young, 1953, Kennedy and Graybiel, 1962, Hixson et al., 1984, Lentz, 1984). Individuals susceptible to motion sickness early in life tend to be troubled by sickness later in life. Motion sickness is an enduring trait over time and type of motion. Motion sickness history questionnaires are perhaps the best current available predictor of sickness in new motion environments (Kennedy et al., 1990, Hixson et al., 1984). Part of this section deals with mechanisms that might be the basis for idiosyncratic reactions.

The evidence that suggests that motion sickness is an enduring trait also suggests considerable independent variation. The correlation coefficients over different provocative stimuli are not very high particularly when large samples are studied. Motion sickness history correlates significantly with provocative test scores (Lentz, 1984) and also with sickness in operational environments (Hixson et al., 1984), but again there is much room for independent variation. While some of the "weakness" in correlations is probably attributable to reliability of measurement of the predictor and the criterion (Kennedy et al., 1990, Reschke, 1990) which limits the prediction correlation, almost certainly some of the weakness is attributable to changes in some individuals, and not to elastic measurement. In our studies (Prewitt 1975, Hixson et al., 1984) some individuals were highly susceptible to one kind of stimulus but relatively unaffected by another, even though the provoking stimuli were equal in duration and equivalent in provocation incidence. The fact that "desensitization procedures" are effective in returning airsick aviators to flight status shows that adaptation can serve to change the individual. The flight instructor who experiences flight simulator sickness, while his beginning flight student does not, also illustrates changes that occur through experience with particular motion environments. Part of this section will deal with factors that would serve to change motion sickness susceptibility in individuals from one time to another.

A CONCEPTUAL MODEL AND FACTORS THAT ALTER SUSCEPTIBILITY

Figure 2 will serve as a guide for reviewing mechanisms of the reaction to motion that contribute to idiosyncratic reactions as well as to changes in reactions of individuals over time and over different stimuli.

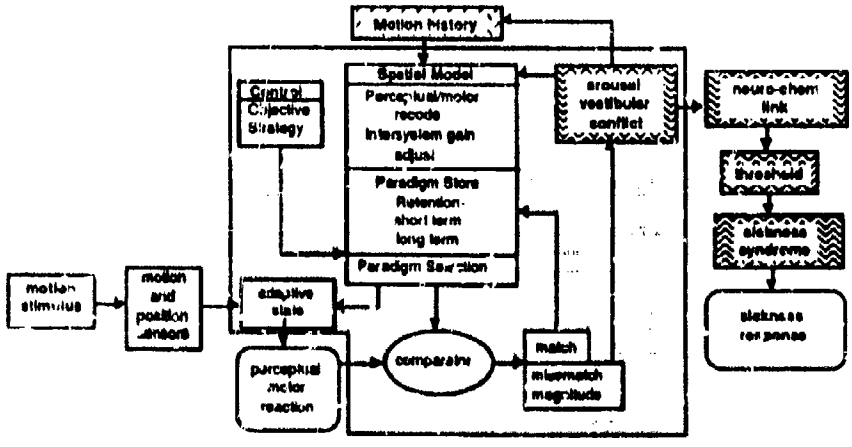

Figure 2. Conceptual Model of Mechanisms of Motion Sickness

The Motion Stimulus

On the left side of Figure 2, is the motion stimulus. The fact that motion sickness tends to be an enduring trait over different forms of provocative stimuli seems to lead some investigators to the idea that, as far as motion sickness is concerned, all provocative stimuli are qualitatively alike, i.e, they differ only in regard to how much sickness they generate. This idea, if wrong, leads to inappropriate interpretation of experimental results. Motion stimuli that are equally provocative can be quite different in some aspects of the total psychological and physiological reactions they generate. Differences in particular aspects of the total reaction could determine whether or not measures of some "predisposing" factor such as trait anxiety achieves significant correlation with measures of motion sickness generated by a particular stimulus. Experiencing the obviously safe off-vertical rotation at 12rpm in a laboratory differs considerably from the initial experience of aerobatic maneuvers in high performance aircraft. Differences in psychological impact of motion are more subtle than this example would imply, i.e., differences are not necessarily ascertainable from a 'common sense' evaluation. For example, off-vertical rotation in the dark at about 15rpm is not at all unpleasant initially. Subjects find interesting the fact that the path of body motion they perceive is quite different from the true body motion. For those made sick by this motion, the pleasant reflection on an interesting experience gives way to stomach awareness which can progress fairly quickly into nausea and vomiting. Before the nausea begins, however, these subjects would be quite willing to repeat the experience.

In contrast is the effect of head tilt during whole-body rotation at 15rpm. Within the first second of experiencing this cross-coupled stimulus, a number of subjects are aware that they do not like it and are even immediately fearful of the experience. Both stimuli involve canal/otolith missmatch, both induce sickness in many subjects but the immediate perceptions of the two stimuli have quite different emotional overtones - even to those made sick by both stimuli. It is curious that only a few individuals find the cross-coupled stimulus sufficiently interesting to want more of it whereas most subjects find the off-vertical stimulus interesting and want to observe it, until sickness symptoms begin. Differences in the total reactions to different motion stimuli may explain some of the inconsistencies in this challenging area of research.

Motion Sensors

Because absence of vestibular function confers immunity to motion sickness (reviewed in Reason and Brand, 1975), measures of vestibular function have been explored for their potential relationship to MSS. Thus far, vestibular function, as tested, has not shown a clear relationship to MSS, other than the immunity that complete loss of function confers. With very few exceptions, measures of vestibular function in these studies have been limited in scope and low in reliability. Kennedy et al., (1990) provide a convenient tabulation of relevant studies. Bles et al., (1984) found that rates of decay of canal-mediated responses to impulse stimuli, the often-investigated 'cupulogram', (Reason and Brand, Chapter 7 1975, Dowd, 1973, Dobie, 1974, Mann and Canella, 1956, Reason, 1967), failed to discriminate seasick from controls, but labyrinthine imbalance appeared more frequently than expected in caloric tests of the seasick group as did unusual visual influence on body sway (see 'field-dependence' below).

Results of Bles et al. (1984) suggest that adaptive capacity may be depleted by the recovery from partial loss of function, leaving some individuals, whose capacity may have been marginal initially, chronically motion sick, when exposed to a provocative environment. Following sudden loss of vestibular function, motion sickness is generated by ordinary voluntary body movement for several days and weeks, until adaptation to the loss (or partial loss) occurs. Physicians would like to have tests that forecast individual adaptive capacity when the treatments they are considering threaten the loss of vestibular function. Some individuals never adapt to the loss successfully. Inputs from the visual and somatosensory partners in the control of body motion induce sickness while relative gains among these partners are adjusted to compensate for the reduced vestibular component. Adjustment continues until automaticity in the control of body motion relative to the earth is achieved.

Failure to find clear and strong relationship to MSS is to be expected considering the number of causal factors and the problems faced by investigators in measuring vestibular function quickly in large numbers of subjects. Some scatter plots that comprise cupulograms more closely resemble circles than ellipses and finding the cupulogram slope is more imaginary than measurable in many responses. Measurement of vestibular function and its interactions with other sensory motor systems is improving and technical advances will enable assessments that are far more adequate than were feasible in the past. Exploration of this area, particularly regarding interaction measures and their changes with repetition, is far from complete but it is still a formidable challenge.

Perceptual-Motor Adaptation

Reason, in the course of a well-organized study of motion sickness (Reason et al., 1969-1978), proposed that a large part of individual variation in susceptibility is attributable to individual receptivity, adaptability, and retention of adaptation. Receptivity, which refers to idiosyncratic processing of sensory information by the central nervous system, was inferred from studies (Reason, 1967a) showing relationships between motion sickness history and the slopes representing the relationship between response magnitude and increments in intensity of different sensory inputs. The idea is that some individuals characteristically damp down stimulation whereas others characteristically amplify it, irrespective of the stimulus condition. Receptivity, which is a broader concept of how the individual interacts with his environment, should be considered with perceptual style, discussed later, but it is included here because of its relation to perceptual-motor adaptation. If in our conceptual model we assume that those who have high receptivity also are strong reactors to sensory missmatch, then the model suggests that those with high receptivity would be more aroused to commence the adaptive process and also more liable to rapid titration toward the sickness endpoint.

Reason also presents evidence that adaptation capability is not necessarily closely related to receptivity (Reason and Graybiel, 1972). For example individuals who exhibit extreme reactions to cross-coupled stimulation may be fast adapters, fast enough to reduce missmatch before the neurochemical link achieves threshold level. While our studies show some relationship between immediate reactions to provocative stimuli and subsequent airsickness susceptibility, many individuals with extreme reactions adapt quite successfully to aviation (Guedry and Ambler, 1972, Hixson et al., 1984). Reason also found individual variation in retention of adaptation. Thus receptivity, adaptability and retention appear to be separate factors. Again referring to the conceptual model in Figure 2, if missmatch magnitude diminishes and if adaptation retention is sufficient then likelihood of sickness with successive exposures to ship or aircraft motion is diminished. In Figure 2, the large central area represents hypothesized components of perceptual-motor adjustment whereby missmatch magnitude is reduced which in turn lessens the chance of sickness.

Autonomic and Neurochemical Adaptive Change

In recent years several procedures have been successful (Bagshaw and Stott, 1985, Jones et al., 1985, Stott, 1990) in treating airsick aviators. Most procedures employ repetitive exposure to cross-coupled stimulation incrementally increased in magnitude in daily training sessions until the aviator tolerates stimuli considerably stronger than were initially tolerable. During this conditioning process, changes in perceptual-motor response to the provocative stimuli occur but there is reason to question that this is the primary source of protection for the aviator. A recent summary (Stott, 1990) shows that a range of procedures have been successful with airsick referrals in different branches of the military services and in different nations. In recent years, the most common adaptation stimulus has been incremental cross-coupled stimulation although in the past others have been used (Gibson and Manning, 1943). Adaptation to vertical linear oscillation appears less readily achieved than adaptation to cross-coupled stimuli and tolerance gained to either stimulus form may transfer minimally, the one to the other (Stott, 1990, p. 378). From casual observations in our laboratory we suspect that adaptation to off-vertical rotation is also difficult. This is an important area for in-depth study. How can adaptation to incremental cross-coupled stimulation transfer to the flight environment if adaptation is minimal to low frequency vertical linear oscillation or off-vertical rotation? The effectiveness may depend upon improved control of the autonomic aspect of the reaction in the flight environment, which is actually less provocative than the sustained unrelenting stimulation that occurs with off-vertical rotation or with steady-state vertical linear oscillation. The flight environment, even in aerobatic training, offers many fairly sustained recovery periods between provocative maneuvers which are anticipated before they occur.

A procedure called autogenic feedback training (AFT) concentrates on bringing the autonomic component of the response to motion under voluntary control. Cowings (1990) proposes that AFT does not involve perceptual-motor adaptive mechanisms but rather increases the threshold to the neurochemical link. In other words if the autonomic aspect of the total reaction is suppressed then the arousal due to missmatch would be reduced thereby reducing neurochemical build-up and also motion sickness. We might also expect that if AFT reduces missmatch arousal, then perceptual motor adjustment would also be obviated, if the task and environment permit effective function without perceptual-motor change. Because the adaptation procedures used in treatment have not been optimal for perceptual-motor change, this may be an appropriate view of the effects of some of these procedures, including the AFT of Cowings'. On the other hand, it is clear that environments that

demand perceptual-motor change for effective function (Melvill-Jones, 1985, Guedry, 1965, Guedry, 1964) yield dramatic perceptual-motor changes that parallel reduction of sickness and are probably instrumental in preventing sickness. Conditioning with optimal conditions for perceptual-motor adjustment may yield beneficial transfer effects by improving 'learning-to-learn'. Perceptual-motor adaptation appears quite stimulus specific when tested soon after the adaptation procedure but after several days, more general adaptation becomes apparent (Guedry, 1965). Intensive incremental exposure to cross-coupled stimulus seems to yield more general adaptation (Graybiel and Knepton, 1972). It is likely that both perceptual-motor learning and the threshold to the sickness syndrome can be altered with conditioning procedures. Both may be somewhat idiosyncratic in the first place creating individual differences in susceptibility that can be altered by conditioning procedures. Aside from the excellent comparative series carried out Reason, wherein adaptation and adaptation-retention were observed with systematic variation in visual-vestibular conditions (Reason and Brand, 1975), and the studies mentioned by Stott (1990, p.378), we have insufficient data-base for further specifying or amending the conceptual model represented by Figure 2.

Both mechanisms, adaptive perceptual-motor change and adaptive autonomic control, involve something akin to learning. However, Graybiel and Lackner (1980), who found no evidence for correlation between signs of sickness and physiological indicators of autonomic activity, challenge the value of AFT, a challenge discussed in some detail by Cowings (1990, p.354). Since Graybiel (1969) proposed a "facultative link" between the perceptual-motor adaptive process and the onset of sickness, we seem to have a concensus for a linking threshold in a conceptual model. Whether the threshold can be modulated through conditioning, the exact nature of the threshold, and where the threshold should be located in the chain of events is somewhat debatable (Oman, 1990).

That the threshold linking perceptual-motor adjustment to the sickness syndrome is subject to conditioning is suggested by evidence for conditioned susceptibility to motion sickness (see Fox, 1990). Money provided a convenient summary of evidence for conditioned susceptibility:

> "There are reports of 'sea sickness occurring at the sight of a ship [or] mounting the gangway' (180). Such reports are frequently heard as anecdotes about persons who have been sick previously at sea (82, 253), and such sickness can be considered an ordinary conditioned response in which the primary and associated stimuli together do give the same response as the associated stimulus alone. Everyday reports of this kind of sickness usually do not mention vomiting, and it is reported similarly that 'with chronic administration of emetics to dogs and cats, there [was] observed no instance of frank vomiting as a conditioned response; on the other hand, conditioned salivation occurred in a number of cases.' The authors refer to other studies in which conditioned vomiting was achieved (41). Again, although many repetitive swing tests of dogs have been reported (27, 215, 230, 294), only one animal is reported to have vomited as a conditioned response (230). Ordinary conditioned vomiting of this kind in man is also rare, but it has occurred (253)."
> (Money, 1970, p.16; reference numbers are those of Money).

Psychological Aspects of Motion Sickness Susceptibility

The idea that motion sickness is a byproduct of a protective mechanism against neurotoxins (Money, 1991) does not seem consistent with consideration of motion sickness susceptibility as a product of disturbed psychologic function, but the question of psychological correlates, causal or otherwise, has been a persistent one in this field. The presence of psychological correlates suggests that MSS is, at least in part, an enduring psychological trait. The autonomic reaction to motion stress is individualistic (Cowings, 1990, p. 356) in that ... "no two individuals produce precisely the same stress profiles"... as indicated by differing patterns of physiological indicators of autonomic activity. High scores on neuroticism, anxiety, feminism and introversion scales correlate with high motion sickness history. Considering results of a number of psychological tests, Collins and Lentz (1977, p.593) concluded that nonsusceptibles "appear better prepared to cope in a nonemotional manner with stressful situations, whereas susceptible individuals may be more likely to manifest emotional responses in the same situation." These authors were careful to point out that their findings were based upon extremes, susceptible and nonsusceptible, in a very large sample. Put in aviator training perspective, scales designed to measure these personality traits yield statistically significant but low correlation to motion sickness as measured by questionnaires; and motion sickness history questionnaires yield statistically significant but low correlation to measures of air sickness in early stages of flight training; and measures based upon an entire early stage of flight training have a correlation of about 0.6 with the next stage of flight training (Hixson et al, 1984). This means that a number of individuals with high scores on

neuroticism, anxiety, feminism and introversion will have little trouble with airsickness in flight training and if a representative sample of all flight students, not just extremes, is evaluated, then correlation between any of these measures and MSS will be very low. The number of factors entering into MSS does not permit any one measure to be highly correlated with measures of motion sickness susceptibility.

Fear and Motion Sickness

Physical characteristics of motion stimuli and factors other than fear and anxiety are the principle determiners of motion sickness (Reason and Brand, 1975, p.192) but some of the symptoms that accompany fear and anxiety also are associated with motion sickness. In some motion environments, like that of the student pilot, anxiety and fear may heighten arousal to the point that subconscious perceptual-motor adaptive learning as well as the learning of flight tasks is impaired and in our model increased arousal may hasten sickness end-points. But fear is not a primary factor in motion sickness, many motions that are obviously not dangerous are extremely provocative. The views of Wendt (1948) remain valuable today:

> "When people speak of psychological factors in motion sickness, they usually refer to one or more of seven different classes of factors, more or less distinct from one another. Let me state these with special reference to airsickness.
>
> 1. Expectation and suggestion. What the traveler has heard about airsickness, the attitude he sees others adopt, the observed effects of rough air on others, and his expectations from his own past experience affect liability to sickness.
>
> 2. The specific conditioning effects of past experience. Sickness in autos, boats, amusement park devices, or airplanes tends to condition nausea to whatever stimuli were present at the time. These include sights, sounds, odors and, most important, the stimuli from motion itself.
>
> 3. The specific habituation effects of past experiences. Experience ordinarily lessens susceptibility by eliminating the unexpected, leading to a more correct estimate of the chances of sickness, and by setting up some barrier against elicitation of nausea such as occurs whenever stimuli are repeated.
>
> 4. The effects of concurrent activity. Airplane pilots and auto drivers infrequently get sick; navigators and passengers more often do. No helpful explanation of this has been proposed but it appears to be well-authenticated that the nature of one's activity can have a considerable effect on proneness to sickness.
>
> 5. The effects of concurrent emotional state. Apprehension, fear, anxiety and grief are often present when passengers become airsick. Opposing emotional states of confidence, satisfaction and well-being are regarded as preventing sickness.
>
> 6. Airsickness may be a motivated symptom which frees a student by wash-out from a situation which he wishes to escape.
>
> 7. Airsickness may be a weakness associated with certain personality types, e.g., anxiety neurotics, who are more susceptible to the effects of the psychological factors involved in flying.
>
> In the brief space available it is not possible to review all of the evidence for the effectiveness of each of these seven classes of psychological factors.
>
> The effects of expectation or suggestion, conditioning and of inexperience are authenticated by laboratory experiment, by the general testimony of those who become airsick, and by statistics of sickness rates gathered from flyers. It is clear that these are important contributory factors."

In 1948 Wendt was attempting to correct the idea that airsickness is primarily due to fear but he clearly included fear and anxiety as potentiating factors. Wendt contrasted findings of relationships between personality traits and motion sickness, where both were measured by questionnaires with relationships between personality questionnaire measures and sickness as measured in flight training. The following partially summarizes his findings:

> "The addition of all the personality items predictive at the .25 probability level or better to the 1 per cent scoring key served to raise the correlation between questionnaire scores and the airsickness scores only from +.43 to +.45. From these data we conclude that airsickness was to only a slight degree associated with such personality traits as were measured by our questionnaire." (Wendt, 1948, p.28).

Wendt clearly includes several psychological factors as potentiators of airsickness. In this connection the simple preventive measure of an introductory lecture, explaining airsickness to flight

students just prior to commencement of flight training, seems to provide a substantial decline in the incidence of subsequent airsickness (Roth and Rupert, 1991).

Perceptual Style

Much of the interest in perceptual style and its relation to motion sickness stems from the work of Witkin (1949-1950) whose studies of spatial orientation seemed to reveal two classes of people, those whose perception of the verticality of a line seemed to be controlled by surrounding visual framework and those whose perceptions were more controlled by body cues so that judgements could be made independently of the surrounding visual framework. The former were referred to as field-depedent (meaning visual-field-dependent) and the latter were referred to as field-independent. Observations were carried out in a variety of conditions wherein a rod, pivoted at its center, was manipulated to apparent verticality while the rod itself was centered in a tilted square frame or in a tilted room. Subjects were either seated upright or tilted relative to gravity or relative to the resultant force while enclosed in a room which could be tilted relative to gravity or to the resultant force (generated by a centrifuge). The pronounced individual differences seemed to be enduring characteristics as indicated by high test-retest reliability, for both men and women, on the order of 0.87, with a year intervening between tests.

Witkin found marked sex differences, women being exceptionally field-dependent, and men tending to be field-independent. This parallels differences in motion sickness susceptibility of men and women. He then correlated Rod and Frame Test (RFT) scores with a variety of non-orientation perception tests. Significant correlations led to the conclusion that particular modes of perception "represents a deep-seated characteristic of the individual." Proceeding on, Witkin (1950) examined relationships between the RFT and a broad range of personality tests, concluding that there is an intimate relationship between perceptual style and the basic personality structure of the individual. Potentially relevant to motion sickness susceptibility was the conclusion that those who depend upon visual experience in their perception of the upright tend to be characterized by passivity and anxiety, lack of self-awareness and inability to organize coping responses (along with other seemingly undesirable traits) whereas field-independent (FI) people, "who rely mainly on postural experiences", tend toward self-assuredness, activity, self-awareness and body-confidence.

These remarkable conclusions have led to investigations of the relations between perceptual style and motion sickness susceptibility, a number of which, as tabulated by Kennedy et. al. (1990), yield significant correlation with motion sickness scores but the findings are not consistent. Reevaluation by Frank and Casali (1986) suggests relationship between field-dependence and motion sickness susceptibility. The spatial model in Figure 2 may be subject to some "deep seated characteristics of the individual."

The following differences between studies probably generate apparent inconsistency in results between studies of relationship between field dependence (FD) and MSS: 1) Studies that involve only subjects with minor differences in FD. 2) Studies that involve only selected extremes in FI/FD. 3) Studies that involve provocative stimuli that are minimally provocative. 4) Studies that use stimuli that are immediately highly provocative. 5) Studies that differ considerably in the qualitative aspects of the provocative stimuli. To ignore the qualitative aspects of provocative stimuli seems unwise when searching for personality correlates. The least attractive possibility to those pursuing this area is that the idea should be discarded.

Continued exploration in this area may be fruitful particularly if interdisciplinary teams are involved that include members with substantial backgrounds in each of several key physics of motion stimulus areas viz., motion stimuli, motion sickness, vestibular function, perception and personality. Individual differences in the dynamics of the response to some motion stimuli are very impressive. During deceleration from a 3 G_z centrifuge run, the otolith system can be stimulated so that it signals essentially upright posture relative to the earth while the semicurcular canals signal high-velocity forward-tumble (Guedry and Oman, 1990). With subjects in complete darkness, a typical perception is that the body pitches forward to approximately a 90 degree nose-down position where it remains despite a persisting paradoxical and confusing forward tumble angular velocity that is superimposed. It is as though the canal-mediated angular velocity signal can induce only so much distortion of the otolith-mediated angular position perception. However this typical perception is not the only perception because a few subjects report head-over-heals tumbling during this same stimulus, as though their otolith (and proprioceptor) position information were completely overcome by the canal information. Howard (1990), who has excellent "Pitch-vection" research devices has indicated similar individual differences - most subjects seated upright relative to gravity, can be displaced in perceived pitch position relative to

gravity only so far, eg., 45 degrees, by visual pitch vection, but a few experience pitch tumble through several revolutions. Exploration of relations of these individual differences in relation to adaptation to the aviation environment and particularly the aerospace environment may prove interesting.

Sex Differences

A consistent finding is that women are more susceptible than men to motion sickness (Money, 1970, Reason and Brand, 1975, Lentz and Collins, 1977), recently confirmed by Lawther and Griffin (1987, p.986) for ship motion (sea-going ferries). Incidence in women appears to be heightened near to menstruation and during pregnancy, suggesting a hormonal factor (Reason and Brand, 1975) in motion sickness.

Considering the recent increased interest among women in physical fitness conditioning and in considering aviation as a career, it would be interesting to determine sickness incidence among women according to physical fitness level. Several recent studies have suggested that exceptional aerobic fitness in men increases susceptibility to motion sickness, possibly due to hormonal changes (Banta et al., 1987, Whinnery and Purnell, 1987, Cheung, et al., 1990). If extreme fitness in women has hormonal effects what will be the effects of exceptional fitness in women on motion sickness susceptibility?

Motion History

Every aspect of the vestibular-mediated physiological reaction to motion seems to be modifiable by previous experience and it has even been suggested that the physical reaction to accelerative forces of the vestibular transducer may be modified by changes in stiffness of the cilia in the transducers (cupulae and otolithic maculae) through vestibular efferent control, a mechanism that is reasonably established for auditory outer hair cells but doubtful for vestibular hair cells. The modifiability of responses by experience tends to cloud detection of enduring idiosyncratic responses because responses of individuals can be changed and the changes can be long lasting (Reason and Diaz, 1972). As indicated earlier, the motion environments that constitute an individual's motion history are also, to some degree, determined by the individual's personality. The environments entered, and the activities in the environment are partially determined by the personality of the individual. Thus exposure history and personality are intertwined in the current state of the individual.

A number of questionnaires have been developed in efforts to obtain quantitative as well as qualitative information for motion history and motion sickness history; see reviews by Reason and Brand, (1975), Kennedy et al., (1990), Collins and Lentz, (1977). Many questionnaires were developed in pursuit of the idea that motion history reveals perceptual-motor and autonomic-response adaptation opportunity and sickness history reveals idiosyncratic sickness susceptibility. Another idea (Prewitt, 1975) is that the individual's motion exposure history, particularly early in life, has been critical in development of many enduring characteristics including personality characteristics.

> In early infancy, motion is predominantly passive, and its quantity and quality depend on the exigencies of life. The possibility that early differences in exposure to motion might have developmental implications has been a matter of substantial interest recently (Peto, 1970; Ornitz, 1970; Prescott, 1971; Guedry, 1972), although the idea ... is not new (Petersen and Rainey, 1910). There are several basic ideas which can be considered in this connection: (1) Deprivation of passive motion early in life may prevent normal neurological development of motion control systems. The effects of sensory deprivation on neurological systems such as the visual system (Kolata, 1975; Greenough, 1972) could be expected to apply to this system as well, considering its close interrelation with both the visual system and motor systems. (2) The quality of early handling might condition idiosyncratic arousal reactions to motion which in turn could influence neurobiological and behavioral development. (3) Differences in development of skilled control of motion could influence many aspects of human behavior.
>
> ... It has been indicated that passive, gentle oscillation of premature infants improves general motor maturation, visual fixation and pursuit, and in general, overall health (Neal, 1968). A series of studies (Korner and Thoman, 1972; Pederson and ter Vrught, 1973; Pomerleau-Malcuit and Clifton, 1973) offer general support for these findings and suggest that regimens of vestibular stimulation in early life may have more developmental significance than special regimens of auditory or cutaneous stimulation. The possibility that reading disabilities and some psychiatric disorders may involve anomalies in

vestibulocerebellar development has also been indicated (Frank and Levinson, 1973; Holzman et al., Hallpike et al., 1951) but not without some controversy (Stockwell et al., 1976). The stereotyped rocking behavior of psychiatric cases and of monkeys in sensory deprivation experiments and the instability and "swimming feelings" reported by mentally disturbed individuals have been cited as evidence supporting the general idea that deficiencies in equilibration systems, either genetically or environmentally determined, may somehow be involved in the etiology of behavioral disturbances. While a range of evidence can be adduced in support of these theoretical positions, more specific hypotheses and neurobiological experiments relevant to these are needed." (Guedry and Correia 1978).

The topic of motion sickness susceptibility in relation to behavioral deviation and psychiatric categories is being studied by Mirabile (1990) whose views reflect those of Witkin. Witkin found tendency for shifts in field-dependence with age, and from evaluation of perceptual styles of young adults in relation to a battery of personality test, he concluded that those who are intermediate between extremes of Field-dependence and Field-independence tend to be better adjusted than those who are at either extreme (Witkin 1950). Thus, good personality adjustment is not associated with either extreme, according to Witkin, but this seems at odds with some of the recent findings of Mirabile, (1990) concerning motion sickness in psychiatric populations.

SUMMARY OF FACTORS IN MSS

Several factors, proposed by several authors, are summarized in Figure 3, along with indication of how they might interact with components of the conceptual model. History of motion, from nature and nurture perspective, is shown influencing idiosyncratic receptivity, perceptual style, autonomic control and hormonal state. Receptivity, the idea (Reason, 1967a) of idiosyncratic tendencies that augment or diminish all sensory inflow, is shown influencing the gain of reactivity state which is also adjusted for gain and response pattern through perceptual-motor adaptation. Perceptual style influences the spatial model which now includes missmatch assessment (perceived tumble relative to the earth may not violate the motion control imperative of some individuals). Control of autonomic reactions has been preconditioned and can be further altered through adaptation procedures such as AFT. The hormonal state is also subject to nature/nurture history, for example aerobic conditioning (discussed below). Hormonal state modifies the neurochemical link. Arousal from vestibular missmatch (intravestibular, vestibular/visual, vestibular/proprioceptor) remains a focal point for both the pathway to perceptual-motor adjustment and the link to the sickness syndrome. Autonomic control influences autonomic arousal from vestibular missmatch which together with hormonal state influences the neurochemical link.

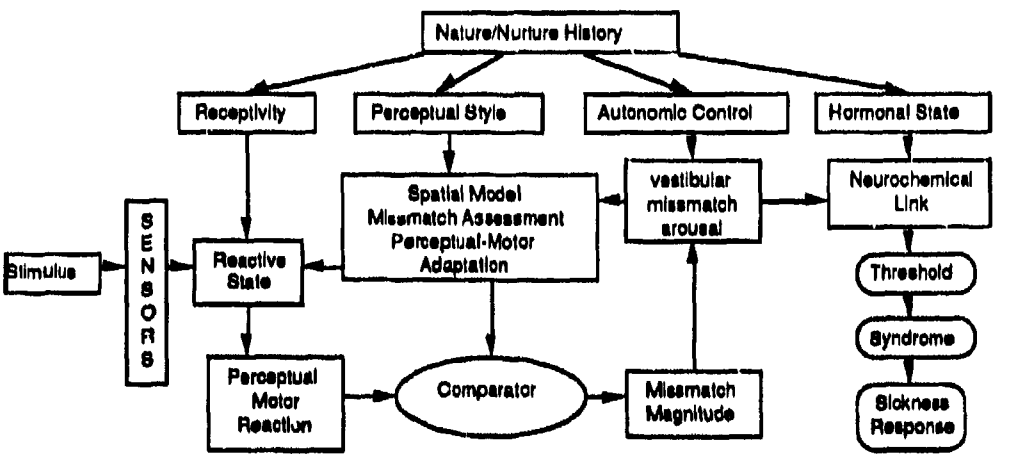

Figure 3. A summary of proposed factors with indiction of possible interactions.

Figure 3 provides a convenient overview of suspected factors in MSS, some more understood than others, and serves to illustrate the complexity of predicting MSS. Much more specificity is needed for this conceptualization to become a true model, testable by experimental observation. For example function of the hormonal component (Kohl, 1990) must fit with current information on anti-motion sickness medication (Wood, 1990). As indicated at the beginning of the earlier chapter (Guedry, 1991), the mechanisms of motion sickness challenge the entire scope of neuroscience disciplines and all levels of neurosciences are challenged by and must be used to challenge any conceptual model developed.

FACTORS THAT ALTER POPULATION INCIDENCE

Other than adaptation and idiosyncratic factors, a number of factors and conditions influence how much sickness occurs in a provocative motion environment and whether or not individuals in that environment will experience motion sickness.

If the job of the individual permits, selecting a location within the motion platform may serve to reduce or avoid sickness. The magnitude of linear accelerations experienced varies with location within a ship or large aircraft. When the entire ship or aircraft rises or falls, location makes no difference but tangential and centripetal linear acceleration components increase in magnitude with distance away from the center of any angular motion such as pitch or roll.

Supine body position is effective in altering sickness incidence (Manning and Stewart, 1942, 1949, Reason and Brand, 1975, p.94). Even the very disturbing effects of deceleration from a Hyper-G centrifuge run are reduced when subjects are supine (Leger, 1984) for reasons not completely understood. When in the supine position it is probably helpful to locate the horizontal semi circular canals in the plane of maximal angular velocity of the platform (usually the roll plane of a ship), as the vertical canals seem more productive of sickness (Benson and Guedry, 1971).

Fixing the head and body relative to the motion platform is helpful in reducing sickness (Johnson et al., 1951, 1953). While this point has been challenged, there is no question about its effectiveness in some motion environments, such as rotating rooms, or continually circling aircraft or during Earth-orbit. Head-rest fixation is helpful in avoiding inadvertent head movements that occur; even heavy breathing induces inadvertant head motion. Frequent head movement relative to any moving platform increases the probability of discrepant otolithic (Hyper-G, Hypo-G) effects and cross-coupled angular velocity semicircular canal effects, all of which are nauseogenic.

Location on ship can also reduce nauseogenic odors. Anyone who has been on a ship driven by diesel engines during a following sea will recall the efforts of experienced crew to find locations away from the diesel fumes that tend to envelope the ship when prevailing winds are from the stern. Money (1970) provides a convenient summary on effects of odors. Reference numbers in the following are those of Money (1970):

> "Tobacco smoke, unpleasant odors, and even food odors have been considered to be promoters of motion sickness (12, 39, 50, 54), and since motion sickness occurs more readily in the presence of nauseating drugs (22, 34, 240, 249), it seems reasonable to expect that it would occur more readily in the presence of a nauseating odor. Odors associated with previous bouts of motion sickness sometimes promote motion sickness very effectively (253). It has been reported that the odor of pyridine promotes motion sickness and, surprisingly, that the odor of toluol suppresses motion sickness (302)."

Ambient temperature has been found not to be a contributing factor in the genesis of motion sickness in a number of formal studies. Typically, in these studies, air exchange was carefully controlled to avoid effects other than temperature (Wood and Graybiel, 1970). My experience, in studying vestibular function over the years, is that when ambient temperature in the chamber or capsule containing the subject is uncomfortably high, incidence of sickness increases in vestibular experiments not intended to study motion sickness. Possibly odors associated with increased temperature are the predisposing factor. The following quotation from Money (1970) is consistent with this interpretation:

> "The time of day and the interval since eating a meal have been found to have *no* influence on average susceptibility (6, 27, 35, 192), except in one study (91) in which the incidence of severe sickness increased with time after the last meal to a maximum at 3 hr and decreased. Ambient air temperature has been found repeatedly not to influence susceptibility (75, 135, 152, 155, 192, 309), although persons suffering from motion sickness frequently express a desire for cool fresh air, and many susceptible persons

consider excessive heat a contributing factor (50). It is possible that the odors that accompany high temperatures in some situations promote motion sickness."

Visual conditions influence sickness induced by particular motion environments. A view of the horizon from the deck of a ship can influence interaction within the brainstem that lessens intralabyrinthine conflict that would otherwise require resolution by higher centers (Guedry, 1978). Vestibular reflexes that stabilize the eyes relative to the earth only potentiate sickness when the individual is enclosed within a ship or aircraft. Closing the eyes can reduce this potentiating effect.

The layout of visual displays and the visual search task within visual displays also influence sickness incidence. Displays that demand frequent and large head-movements would potentiate sickness. Several studies show that tasks that require visual search within a cluttered display potentiate sickness (Guedry, et al., 1982) even when the head is fixed relative to the motion platform and the display. In motion-based simulators it is possible that peripheral visual motion improperly phased to vestibular input will potentiate sickness when the task demands focused attention on cockpit displays (Guedry, et al., 1979).

The task of the individual in the motion environment not only affects the rate and form of adaptation to the environment but it also influences sickness susceptibility in the environment. Mentally dwelling on motion sickness symptoms or envisioning motion of the body in space seems to potentiate sickness, whereas, mental involvement in an interesting task has preventive effects, if the task does not involve a nauseogenic visual display (Reason and Brand, 1975, pp. 71-73).

A number of personal factors influence susceptibility to motion sickness. Age which unfortunately is beyond our control is a factor in motion sickness susceptibility. It has been reported that motion sickness is rare before the age of two years, that it increases to a maximum by about the age of 12, declines between 12 and 21 and declines still further before the age 40 years (Reason and Brand, 1975 p.87). Beyond the age of 50, motion sickness was reported to be rare in civil aircraft (Lederer and Kidera, 1954) but Lawther and Griffin (1986) reported that 22% of those suffering from sickness on Channel Island ferry crossings were over the age of 59 years. Age does not confer immunity to highly provocative motions, but declining incidence with advancing age may be related to declining vestibular afferent information with advancing age. Lawther and Griffin (1987) use different values for constants in their equation for predicting sickness incidence, depending upon the age as well as sex of the subjects that make up the group.

Other predisposing factors that have been indicated are fatigue, alcohol (and of course a variety of drugs) and emotional state. Dowd (1975) found evidence that sleep deprivation altered the vestibulo-ocular reflex to the provocative cross-coupled stimulus and theorized that fatigue reduced the motion-sickness suppression that pilots had achieved through flight experience thereby rendering them more susceptible to the cross-coupled stimulus. Clearly more information is needed in this potentially important, but difficult to study, area. Consider the problems encountered in finding experienced pilots who will agree to congregate for the study the effects of their sleep loss on their tolerance of purposely provocative motion - an important consideration for those interested in conducting research in this topic.

MYSTERIOUS VISITATIONS AND A GENERAL PRUDENTIAL RULE

Anyone who has worked in motion research for a number of years becomes personally aware of occasional heightened susceptibility to motion exposure in laborstory devices. Reasons for these variations are not clear. It is natural to suspect that immediate motion sickness susceptibility would be influenced by conditions and activities that induce some of the many of the signs and effects that are part of the motion sickness syndrome. These signs and symptoms as described by Benson (1988), are listed at the end of the chapter on Mechanisms of Motion Sickness. Until specific studies have been conducted to show that conditions that induce headache, etc, are not effective in potentiating motion sickness susceptibility, a general prudential rule is to avoid conditions that produce symptoms like those that are part of the motion sickness symdrome. Because of the difficulty in conducting studies of motion sickness with human subjects, prudential rules will have to suffice in a number of areas for some time.

In closing this chapter, I wish to call the reader's attention to the very substantial work that was carried on in this field in Canada, England, the United States, and Australia in the period between 1940 and 1965. Much of this work is referenced in Money (1970), and Reason and Brand (1975). Intensive and extensive study in those years forms much of our knowledge base for today.

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SPACE SICKNESS

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ABSTRACT

Motion sickness in spaceflight occurred only rarely in the earliest space flights in small capsules, but in the larger Space Shuttle the incidence is fully 70%. Apparently, in larger spacecraft the requirement to make head movements and body movements in weightlessness increases the likelihood of space sickness. Typically, after its appearance in the first day of a spaceflight, space sickness is made worse by head movements and by disorientation, and it is ameliorated by remaining motionless. The sufferer gets better slowly and usually is entirely well after three days. Anti-motion sickness drugs have been useful in dealing with space sickness and biofeedback techniques might be helpful, but attempts to predict susceptibility (except by assessing susceptibility on previous spaceflights) have not been successful.

INCIDENCE OF SPACE SICKNESS

Motion sickness did not occur in the first American spaceflights, probably because the spacecraft were very small. Only when larger spacecraft were used, and when the astronauts' jobs required them to move about in weightlessness, did space motion sickness appear in the American program. (It is said, however, that on the second Soviet spaceflight, in the small Vostok spacecraft, violent motion sickness was encountered). The incidence of space motion sickness is shown in Table 1, taken from Homick and Vanderploeg (7).

Table 1.

*INCIDENCE OF SPACE MOTION SICKNESS IN U.S. AND U.S.S.R. SPACE MISSIONS (a)

United States (as of October 1984)

Program	Number of Crewmen (b)	Incidence of Motion Sickness
Mercury	6	0
Gemini	20	0
Apollo	33	11
Skylab	9	5
ASTP	3	0
Shuttle	48	25

Soviet Union (as of 1981)

Program	Number of Crewmen (b)	Incidence of Motion Sickness
Vostok	6	1
Voskhod	5	3
Soyus	38	21
ASTP	2	2
Salyut 5	6	2
Salyut 6	27	12

The incidence of space motion sickness in the Shuttle as of 1988, considering flights by 125 astronauts, 91 individuals some of who flew more than once (23) in the first 24 Shuttle flights, is 71% (8).

SPACE SICKNESS IS MOTION SICKNESS

Motion sickness in space *is*, apparently, motion sickness, although in space there is no sweating, flushing is more common than pallor, the vomiting has been described as "brief" and "usually sudden ... without nausea or other prodrome", and after 3 days or so when the sickness disappears there is a peculiar nonsusceptibility to even the most nauseogenic (Coriolis) accelerations (9,24). It is possible that the reported nonsusceptibility to Coriolis accelerations resulted from the lack of a reference "down"

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*(a) Reports of one or more symptoms of space motion sickness are included. No attempt was made to categorize by severity of symptoms.
(b) Includes some crewmen who flew more than once. Table taken from Homick and Vanderploeg (7).

in spaceflight, or from the small number of experimental subjects studied, but the Soviets also mention, as a "peculiarity", that once the habituation to space motion sickness is complete, there is then a "clearly defined higher resistance to cumulative vestibular loads" (1). In Soviet cosmonauts, unlike American astronauts, nausea is listed as a "typical" complaint (1,12). In the motion-sick cosmonauts, pallor and cold sweating are "typical" signs according to one report (12), but in another report pallor and cold sweating sweating were omitted from the list of "typical" signs but were reported to be present "in a number of cases" (1). In both the American and Soviet experience, vomiting is usually followed by an improvement in the condition(at least for a while).

MOTION SICKNESS IS A POISON RESPONSE

In motion sickness the body behaves as if it had been poisoned (6,13). Motion sickness is a poison response provoked by motion. More specifically, motion sickness is a poison response provoked by motion acting (directly or indirectly) on the vestibular system (14). There is now evidence that the vestibular system is involved not only in the poison response of motion sickness, but also in the poison response to certain poisons. Experimental animals were found to be strikingly defective in their response to poisons after surgical removal of the vestibular apparatus (15). This involvement of the vestibular system in poison response was hypothesized by Claremont (4) and by Treisman (25). Of course, those humans or animals who are lacking vestibular function are also completely nonsusceptible to all forms of motion sickness that have been tried on them. A recent study of labyrinthine-defective humans found them to be entirely nonsusceptible even in response to a purely visual motion sickness stimulus (3). Motion sickness is a poison response provoked by motion acting on the vestibular system.

THE PECULIAR NONSUSCEPTIBILITY AND LONG-TERM SPACEFLIGHT

It is important to note what follows logically from the observed peculiar nonsusceptibility to the sickness in response to Coriolis accelerations in spaceflight. To the extent that available evidence supports conclusions, this nonsusceptibility suggests (hints) that a rotating spacecraft could be used for long-term spaceflight without serious vestibular/motion sickness complications. On the basis of the (sparse) evidence available, it might be anticipated that rotations starting after a week or so of spaceflight would cause no motion sickness problem. Rotation could of course stop for short periods during a long flight, and for a week or so before landing to allow readaptation to a nonrotating environment. At least it can be said that, as of now, it would be premature to rule out the use of rotating spacecraft because of anticipated vestibular/motion sickness problems. It might be that no such problems exist. Before launching a rotating spacecraft on a long flight, however, further inflight experimentation could be required to see whether the nonsusceptibility or ability to adapt could hold in spite of the variable-strength "down" (the linear acceleration) produced by the rotation. Earlier experimentation in slow rotating rooms (including walking on the walls) suggests that any problems encountered would be readily overcome by habituation.

THE SEVERITY OF SPACE SICKNESS

There is little detail available concerning the severity of space motion sickness. One analysis (Davis et al, unpublished data cited in ref 23) of 57 Shuttle astronauts who experienced motion sickness in spaceflight, describes 46% of the sick astronauts (26 astronauts) as mildly sick, 35% (20 astronauts) as moderately sick, and 19% (11 astronauts) as severely sick. "In this rating scale, even those characterized as mild could have at least one episode of retching and vomiting. In the severe category, the crew person had to express several symptoms of a persistent nature, have more than two episodes of retching and vomiting, and remain ill beyond 72 hours into flight."(23) The above numbers are reasonably accurate, but it should be remembered that, in general, an astronaut does not want to be known as someone who was sick in space and it is part of the culture of astronauts to be positive and healthy and capable. If asked about the degree of sickness being experienced, an astronaut's culture would incline him to believe and to say that he was OK, and this inclination is possibly responsible in part for reports of vomiting in spaceflight without noticeable prodromal symptoms. There is even one report of an episode (5) in which an astronaut vomited and then got the agreement of his fellow crewmen that they would all keep the episode secret. This cultural factor has perhaps acted to reduce the reported incidence and severity of motion sickness in space.

THE INFLUENCE OF HEAD MOVEMENTS

Some astronauts have expressed the opinion that the incidence of space motion sickness would be almost (although not quite) 100% if all the astronauts had to do considerable moving about in the spacecraft during the first 3 days of all spaceflights. On some shuttle flights there are some crewmen whose jobs allow them to remain strapped into a seat for almost all of the first three days. Another influence on the observed incidence of space motion sickness is the past practice of premedication: until recently, some Shuttle astronauts took antimotion sickness drugs (usually scopolamine 0.4mg and dexedrine 5.0mg) before launch. (Present NASA policy, May 1991, is to refrain from premedication and to inject promethazine 50 mg intramuscularly if motion sickness in flight is troublesome. This policy was adopted partly for the reason that antimotion sickness drugs taken prophylatically can impede the natural adaptation to the new motion environment.) As a guess, perhaps 10% or so of

unmedicated astronauts who were obliged to move about and do a lot of head movements during the first three days of a spaceflight would be utterly unaffected by space motion sickness.

DURATION OF SPACE SICKNESS

When motion sickness occurs in spaceflight it gradually gets better but typically it persists for two or three days. The astronaut can get some relief by taking antimotion sickness drugs, and more relief can be gained by remaining motionless (17). After a period of being motionless the astronaut starts to feel better, but when he starts to move about again the nausea gets worse again, and if he persists in moving, vomiting can result. After three or four days, when habituation is complete, the astronaut is completely free of symptoms and can move about as much as he wants and even do gymnastics without provoking any symptoms. In one Soviet cosmonaut, however, motion sickness in space lasted two weeks (although vomiting occurred only during the first week); this cosmonaut also experienced motion sickness, including vomiting, for three days after returning to Earth (Matsnev E.I., personal communication). In fact, of the first 27 Salyut-6 cosmonauts, 9 suffered significant sickness after returning to Earth, and 8 of these 9 had suffered sickness in space at the beginning of their flights (12). The sickness associated with the return to Earth is analogous to the 'mal de debarquement' experienced by some people upon getting off a ship. Its occurrence in American astronauts has perhaps been prevented by the relative brevity of their spaceflights, and in the longer Skylab flights it could have been masked by seasickness, since the Skylab astronauts were recovered at sea. Even so, two cases of motion sickness after return to Earth have been reported in the American program (23), and it is possible that motion sickness after a long flight could appear with disastrous results upon stepping onto Mars, or upon stepping onto Earth after a long flight (perhaps after a Space Station flight).

PREDICTION OF SUSCEPTIBILITY

Available tests on Earth have not allowed prediction of susceptibility to space sickness (18,23) although, since 70% or so of people who fly in the Shuttle do experience space sickness, a prediction that all future flyers will be sick in space would be correct in about 70% of cases. There is no significant difference in the incidence of sickness between men and women, and the likelihood of being sick in space does not correlate significantly, positively or negatively, with susceptibility to motion sickness stresses on Earth (nor, probably does it correlate with the amount of the right stuff possessed by the astronaut).

MOTIONS THAT CAUSE MOTION SICKNESS

Motions of the body that cause sickness are, at least usually, motions that exceed the normal frequency and/or magnitude ranges of the vestibular receptors, so that some of the vestibular receptors are reporting false information to the brain in motion sickness situations (22). Vision and some proprioceptors continue to report correct information. In such motion sickness situations the brain says, in effect, 'My vestibular system is giving me false information; therefore my vestibular system has been poisoned by something I ate, and I should vomit.' Motion sickness is not a sickness; it is the normal healthy response to poisoning, triggered by a stimulus that masquerades as poisoning.

In spaceflight there are many sensory inputs that could be interpreted as false information in the vestibular system. For example, throughout weightless flight the vestibular receptors probably tell the brain that the body is falling, whereas vision, and the duration of the "fall", contradict that vestibular input. This falling versus not falling is possibly the most important provocation of space motion sickness. Also, the vestibular receptors tell the brain that there is no down (no gravity), although astronauts usually do perceive a (visually determined) down. The conflict between "down" and "no down" could also be important in space motion sickness, especially in view of the possibility (11) that people who maintain the logical visual indication as the perceived down (instead of taking the "feet are down" perception are the ones more susceptible to space motion sickness. The vestibular receptors also tell the brain that head tilts relative to a perceived down have taken place (according to the semicircular canals) but have not taken place (according to the otoliths), as Benson has pointed out (2). It is seldom considered, but static head tilts relative to the down direction are normally (on Earth) reported by neck proprioceptors, and the absence of gravity's pull on the head in certain tilted head postures in spaceflight could well be an important contributor to space sickness. It has been shown on Earth that motion acting on proprioceptors can cause motion sickness (10). Similarly, the absence of gravity on an extended arm, in an astronaut who perceives himself upright, is consistent with an angular acceleration of the arm and hand in the "down" direction, and since the same angular acceleration of the head and body would have to be taking place together with the arm (because no rotation of the shoulder is taking place), denial of such an angular acceleration by the semicircular canals could create further conflict. This would be especially so if the hand were holding an object known to be "heavy".

AVOIDING PROVOCATIVE MOVEMENTS

Perhaps astronauts should be told that, to avoid motion sickness, they should not only avoid pitching and rolling head movements but they should also avoid, as much as

possible, postures in which the spinal axis is offset from the direction of the perceived down, and they should avoid static tilt positions of the head relative to the perceived down, and they should avoid extended limbs at right angles to the spinal axis, and when moving (translating, floating) about the spacecraft they should maintain the spinal axis parallel to the direction of the perceived down and not soar "horizontally" like Superman. When the body's spinal axis is perceived to be "horizontal" (at right angles to the perceived down), then of course yawing head movements should also be avoided.

THE ADAPTATION

It is thought that there are probably three main aspects of the central adaptation to the unusual vestibular environment in spaceflight: first, a general tendency to ignore or suppress inputs from the vestibular apparatus, especially otolithic inputs; second, a tendency to interpret all changes in otolithic inputs as changes of linear acceleration rather than changes of tilt angle relative to gravity (called variously "tilt-translation reinterpretation", or "sensory reinterpretation" or "stimulus rearrangement", refs 19,29,30); third a substitution of other sensory inputs, especially visual inputs (26,31), for the previously used vestibular ones. Another possibility is that the cupulae of the exquisitely sensitive semicircular canals do not normally have exactly the same density as endolymph (16), a complication usually overcome with the help of information from the otoliths, and that habituation to weightless cupulae also takes place. It is also possible that different cupulae have different densities so that asymmetry of cupulae produce the kinds of problems that have been ascribed (27) to asymmetry of otoliths. The space motion sickness is thought to stop when the central adaptation has proceeded sufficiently. Again, the proprioceptive contributions to the motion sickness and to the adaptation have been largely ignored but could be important. It is known that proprioceptive function is altered in weightlessness (20,21,26,28,29,30) and that proprioception on Earth can be important in motion sickness (10).

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SIMULATOR SICKNESS

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ABSTRACT

Sickness in a flight simulator can compromise training, and it can also give rise to effects that persist afterwards and create hazards for the trainee. Generally, simulator sickness involves more visual disturbances, more dizziness, and more after-effects than in other kinds of motion sickness, and less gastrointestinal disturbance (although a few instances of frank vomiting have been reported, both in the simulator and after leaving it). Simulator sickness can interfere with, and discourage participation in, simulator training. Its aftereffects could cause accidents, and to avoid these accidents the trainees are often grounded for a while after flying the simulators. Different incidences of simulator sickness, most between 10 and 60%, have been found in different simulators and depend partly on the criteria for the "sickness" and on how the simulator is used. Procedures for minimizing the problem have been developed.

INTRODUCTION

The use of simulators for flight training reduces the costs and the risks of training in aircraft. In modern simulators that include a wide angle visual display, however, there are problems with simulator sickness. With technological progress, it seems that as the simulation gets better the simulator sickness problems get worse (4,7,15). These problems have been the subject of symposia and the subject of some excellent reviews and overview papers (1,3,11,12,15,16,20) and they are being addressed with some success. The present paper will attempt to summarize the field briefly and indicate where further information can be found.

INCIDENCE OF SIMULATOR SICKNESS

The incidence of simulator sickness is usually higher among pilots with little experience on the simulator, it is usually (15,21,11) but not always (19) higher still among pilots with extensive experience on the real aircraft and little experience on the simulator. The incidence also varies with the individual simulator, with the kind of mission being flown, and with the criterion for recognising the presence of simulator sickness. In a survey of more than 2500 "flights" in ten different simulators, "simulator sickness incidence varied from 10 to 60%" (15). Other surveys have also produced incidences in this range (5,6,9,10,11), including studies of air combat simulation (5,21), but another survey of (mostly) air combat simulators found an incidence of 67% (18), and the air combat simulator at Williams Air Force Base at one time had an incidence of 87% (R.S. Kellogg, personal communication). Overall, the fraction of pilots who experience nausea in simulators is roughly 10%, and the fraction with "eyestrain" is 25%.

It is not known whether there are comparable problems during and after flying the real aircraft (12).

SIMULATION SICKNESS IS ONLY PARTLY MOTION SICKNESS

The gastrointestinal symptoms encountered in simulators are, in all probability, symptoms of motion sickness (1,15) These include stomach awareness and nausea, and they are probably produced by a conflict in the pattern of sensory information concerning orientation and motion (1,15). In addition to the motion sickness, simulator sickness includes other, separate, visual and vestibular phenomena.

Primitive simulators without visual displays, or with visual displays that are small or night-only, produce almost no motion sickness. Only when large dusk and day time visual displays became available did motion sickness become a noticeable problem in simulators. The large visual displays produce sensations of self motion in the pilot (13,14); in all likelihood these large displays also stimulate the visual system in such a way that the visual system provokes the vestibular system (17) to produce both the sensations of self motion and (in some pilots) motion sickness (pg 328 in ref 15). "Stimulation of the vestibular centers by visual inputs" (pg 17 in ref 22) can produce motion sickness, presumably because the stimulation creates in the vestibular centers a pattern of activity that includes aspects of conflict or mismatch.

Modern simulators include also a motion base (2,24) and the mechanical motions of the motion base can also be suspected of contributing to the motion sickness, especially if the motions are of the low frequence variety that are known to provoke motion sickness in other environments (15).

Kennedy's classification of the symptoms of simulator sickness include visual, vestibular, and vagal (=gastrointestinal) symptoms. Only the vagal (gastrointestinal) symptoms are properly considered motion sickness. In addition to the motion sickness symptoms there are separate phenomena in simulator sickness: the visual symptoms of eyestrain, difficulty focusing, and sensations of visual distortions, and the vestibular symptoms of dizziness, postural instability, and false sensations of bodily orientation. The visual and vestibular symptoms can occur for many hours after leaving the simulator, and they are possible sources of accidents both on the ground and in flight.

In addition to a pattern of conflicting sensory inputs, flight simulators inflict on the pilots visual inputs that are imperfect in apparent depth and that vary with the location of the pilot's eyes as he moves his head. There is also, usually, a very high workload in flight simulators, sometimes for several hours, and the crew is often being tested. Simulator rides are often check rides in which many emergencies are simulated, and if the crews do not perform well they can be subjected to rerides, failure, temporary disqualification, or even permanent disqualification if unsatisfactory performance persists. Simulator rides can be long, stressful, unpleasant, and fatiguing, whether or not motion sickess or any other part of simulator sickness occurs.

The headaches associated with flying flight simulators could be caused by the motion sickness, by imperfections in the visual display, or by the demands of the task. Similarly the drowsiness could be part of the motion sickness or simply the result of prolonged hard work.

MOTION SICKNESS IS A POISON RESPONSE

Motion sickness is a poison response provoked by motion. It is a poison response provoked by motion acting (directly or indirectly) on the vestibular system. The motion sickness that is part of simulator sickness is a poison response provoked, for the most part, by motion of the visual field acting indirectly (through the visual system) on the vestibular system.

DEALING WITH SIMULATOR SICKNESS

Simulator sickness can compromise training (4,15). Some trainees sensitive to simulator sickness will avoid the simulator as much as possible. Some will adapt to the simulator by adopting habits that would be counterproductive or hazardous in the real aircraft; for example, simulator sickness might be reduced by minimizing head movements, but minimizing head movements in the real aircraft would degrade the lookout. The aftereffects (4) of simulator sickness (postural disequilibrium, false sensations of orientation, and visual flashback) can appear several hours after leaving the simulator, and could cause accidents on the ground or in flight. Because of the aftereffects, same day flights in real aircraft are restricted in some organizations, with a loss of operational availability of pilots by as much as 5% (15). Also, simulator sickness is miserable; this kind of misery can be inflicted deliberately in order to induce desired kinds of adaptation (8,25,26), but managers with responsibility for pilots should take steps to eliminate it.

To deal with simulator sickness, current recommendations are (15,23):

1. Avoid creating simulator sickness by suggestion. It can be contagious if one pilot sees another sick. Pilots, should, however, be warned about the aftereffects. No one should go directly home to repair his roof after a simulator flight.
2. Do not fly the simulator when you are hung over or have an upset stomach.
3. Adaptation is the strongest fix for simulator sickness. Try to fly the simulator every day until adaptation is complete (but do not fly it past moderate nausea; stop for the day if you become nauseated). In some simulators adaptation occurs more readily if hops are every 2 to 5 days.
4. Do not fly the simulator the same day as a flight in the real aircraft.
5. Get seated and organized before turning on the visual.
6. Keep to a minimum the number and magnitude of turns while taxiing or while flying near the ground.
7. Keep to a minimum the number of steep turns, abrupt changes in speed or pitch, or turbulance in flight.
8. Keep to a minimum the number and magnitude of head movements, especially large nodding movements during turbulence or during changes of aircraft heading or airspeed.
9. Use mostly instrument flight in layer cloud and not too much clear hood flying, and use night flying rather than day or dusk flying.
10. Use the freeze mode and resetting mode as little as possible.
11. Ensure that all projected channels of visual imagery are correctly aligned.
12. Turn off the visual and turn on the simulator lights before exiting.
13. Whatever is seen or perceived to be the cause of the sickness should be minimized.
14. Be aware that the initial flights are more nauseogenic than later ones, and that some particular missions are much more nauseogenic than others.
15. When adaptation is complete, most (but not all) of these rules can be discarded.

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SEA SICKNESS

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SUMMARY

An historical illustration of the prevalence of motion sickness at sea is followed by a review of experimental studies in which both ship motion and sickness have been quantified. The motions responsible for sea sickness are identified and alternative methods of predicting sea sickness from measures of ship motion are defined. The influence of causal factors other than motion are also considered.

1. INTRODUCTION

Sea sickness may have been the first form of motion sickness experienced by man and animals - it has probably inflicted more persons than any other form of travel sickness. Nevertheless, while all who go to sea have something to say about motion sea sickness, scientific study of the subject has been meagre. There have been remarkably few systematic attempts to determine the motions responsible for sea sickness and little serious scientific effort to minimise sea sickness.

1.1 Types of motion

Various terms are used to describe the motions of ships and people, with several different terms being used for the same motions. Vertical motion of a ship may be called scending (or heaving) or identified by the conventions for vertical motion of seated or standing persons: z-axis motion. The axes used to describe human motion are shown in Figure 1. When a person is sitting or standing facing the front of a vessel the x-axis of the body corresponds to the fore-and-aft axis of the vessel. Similarly, roll motion of the body corresponds to roll motion of the vessel. A person who is free to turn may stand sideways so that the x-axis of the body is parallel to the lateral axis of the vessel and roll of the boat will cause the body to move in pitch. Lying down will render the z-axis of the human body horizontal. These variations may affect human sensitivity to vessel motion. Most studies of ship motion and sea sickness have ignored these variations and described vessel motions using axes of the boat rather than axes of the exposed people. At most, the orientation of the body (upright or recumbent) is indicated.

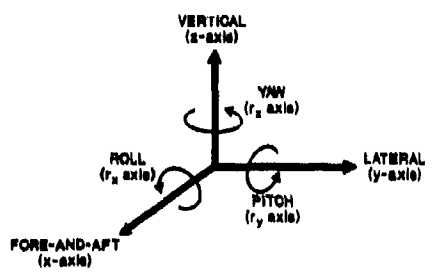

Figure 1 Axes of motion.

Three alternative co-ordinate systems might be used to describe the motions to which people are exposed on ships: a co-ordinate system centred on the person (i.e. a biodynamic co-ordinate system), a co-ordinate system centred on the ship, or a co-ordinate system centred on the Earth. With people moving around on ships, biodynamic co-ordinate systems are inconvenient. Earth-referenced co-ordinate systems are technically difficult because they require that the axes of measurement remain vertical and horizontal as the vessel rolls and pitches. Although some measurements have been made with earth-referenced axes most measurements related to motion sickness have used ship-referenced axes. In consequence, measurements in the 'vertical' and 'horizontal' directions are only truly vertical and horizontal when there is no roll and no pitch. Measurements of 'horizontal' motion (i.e. fore-and aft or lateral motion) using ship-referenced axes can be very different from those obtained using earth-referenced axes due to the gravitational components detected by transducers as they tilt in the gravitational field of the earth (see Section 6 below).

1.2 Effects of motion

Vomiting is the most obvious sign of motion sickness but it is not the only sign of sea sickness. Vomiting may be neither the most sensitive nor the most important consequence of sickness. Where the incidence of motion sickness is low, some less dramatic effect may be a more sensitive indicator since it may be present in greater quantity. Additionally, nausea may be a more accurate indicator of performance and mood than vomiting. Many studying motion sickness have therefore chosen to use scales which give some weight to effects other than vomiting. However, various scales have been employed so comparisons of data from different studies is difficult (see scales defined by Kanda *et al*, 1977; Graybiel *et al*, 1968; Lawther and Griffin, 1986). Terms such as 'nausea' and 'sickness' have different meanings in different studies. Some rating scales have been

designed for experimental studies in the laboratory and are less suitable for application to untrained travellers by means of a questionnaire. Consequently, it is currently only possible to compare the results of a wide range of field trials by using vomiting as the dependent variable. Fortunately, studies at sea reported by Kanda *et al* (1977) and Lawther and Griffin (1987) suggest mild and moderate symptoms of sickness may be predicted from the relation between vomiting incidence and ship motion magnitude.

2. HISTORICAL PERSPECTIVE

Most reports of the incidence of sea sickness give little information on the physical cause of the sickness but provide a background view of the extent of the problem. Sea studies of the efficacy of alternative anti-motion sickness drugs have often included control groups taking no drug (or a placebo) and provide a major source of information on the underlying extent of motion sickness at sea. Other information comes from a few experimental studies and from medical records.

During the years 1909 to 1931 (excluding the period 1916 to 1920) 2.3 men per 100,000 were invalided out of the Royal Navy through motion sickness, while 10.4 men per 100,000 were placed on the sick list (Keevil, 1935). These figures were interpreted as showing that most persons develop an "*immunity*" to motion. From a questionnaire of 100 sailors, Keevil found that 40% admitted to having experienced motion sickness - but in no case was the problem sufficient for it to be entered into their medical records. Of the 40% who reported having been seasick, 38% had been sick on destroyers and only 10% on battleships.

Figures for the numbers of passengers and crew seeking medical attention on the United Fruit Company Steamship Service between 1915 and 1923 are given by Desnoes (1926). About 0.5% of approximately 30,000 passengers, and a slightly lower percentage of about 1.,000 crew, suffered sufficiently severe motion sickness to seek medical attention. There were no recorded deaths due to sea sickness.

Holling *et al* (1944) report a study using soldiers on two minesweepers sent to sea whenever there was a prospect of sufficiently rough weather to test the efficacy of alternative anti-motion sickness drugs. With the vessels at sea for between 4 and 6 hours the incidence of sickness in control subjects varied between 22% and 61% on different trips, with most of these vomiting before the end of each trip.

Data obtained from 1385 troops undergoing assault exercises in India, mainly in small landing craft, showed that 12.4% vomited (Hill and Guest, 1945). In other studies with soldiers in landing craft, Tyler (1946) found that about 35% became sick and about 14% were severely sick during three hour periods. The sickness was found to depend on posture (see Section 3.4.4 below) and medication.

Chinn *et al* (1950) conducted a drug trial aboard a 16,000 ton troop ship on a two-way crossing of the Atlantic Ocean during which 20% of a control group vomited on the eastbound journey and 41% of a different control group vomited on the westbound crossing. Chinn *et al* (1952) report a drug study with troops aboard two further vessels crossing the Atlantic. Some of the troops had made the crossing previously; of these, 36.9% on one ship and 38.8% on the other ship said that they had been seasick previously. In the study, among those who were administered a placebo, 38.0% vomited on one vessel and 37.6% vomited on the other vessel. The incidence of vomiting declined with increasing age; when including those who received active drugs there were 33.6% who vomited in the age range 17 to 20 years while 14.2% of those in the range 30 to 39 vomited.

Handford *et al* (1954) found a 34% incidence of vomiting among troops on a military transport ship crossing the Atlantic. A similar study involving 15 crossings resulted in the prevalence of vomiting varying between 1.1% and 43.8% with an overall average of 22.9% among those taking a placebo drug on 10 eastbound trips and varied from 15.5% to 35.7% with an overall average of 19.6% on westbound trips (Anon, 1956). Trumbull *et al* (1960) reported that on military transport ships travelling across the Atlantic the incidence of vomiting in control groups varied between 8.5% and 22.1% on three crossings. A questionnaire survey of 699 men aboard destroyers involved in escort duty in the U.S. Navy indicated that 39% were never sick, 39% were occasionally sick, 10% were often sick and 13% were almost always sick (Bruner, 1955).

Pethybridge *et al* (1978) found that 67% and 73% of the crew of two British Royal Navy ships had been sea sick during their career, and 42% and 56% had been sick in the past 12 months. During sea trials over five days with rough weather (wave heights generally between 4 and 14 metres), 38% and 47% of the crew on the two vessels were sick on at least one occasion. From 1746 responses to a questionnaire of men serving on a variety of British Royal Navy vessels, Pethybridge (1982) estimated that about 70% of naval personnel suffer from some of the varied symptoms of sea sickness. The incidence of sickness varied with vessel size (see Section 4.3 below).

Among 335 participants in an Ocean Youth Club holiday, 57% experienced sea sickness when no active drug was taken; this reduced to 26% among those taking a drug (Hargreaves, 1980). Attias *et al* (1987) report a 3 day drug trial aboard a 300 ton vessel in sea states 2 and 3 during which 53% of those receiving no drug were sick on the first two days and 23% were sick on the third day.

3. SEA STUDIES OF THE CAUSE OF MOTION SICKNESS

3.1 Introduction

This section reviews studies which have obtained measurements of motion and a measure of the sickness caused by the motion. Effects of non-motion variables are also summarised.

Limitations, especially to the equipment used, render some results insufficient to draw general conclusions as to the motions causing sickness. Nevertheless, observations made during these studies may be of some value in identifying other factors which contribute to the complex relation between motion and motion sickness. Where appropriate, the reported incidence of sickness and vessel motions are compared with predictions using the 'motion sickness dose value' (MSDV) calculations defined in Section 4.2 below.

3.2 Summary of experimental studies

3.2.1 Studies with small vessels

Using fast patrol boats for more than 2 hours and encountering rough sea (giving vertical motion at 0.36 to 0.48 Hz with acceleration in the range -2.6 to +3.3 ms^{-2}) Glaser and Hervey (1951) found that approximately half of a group of soldiers vomited. Soldiers within enclosed floats in a swimming pool were made motion sick by artificial waves having a frequency of 0.29 Hz and peak acceleration in the range -1.6 to +4.3 ms^{-2} (Glaser and Hervey, 1952). Approximately 57% of soldiers felt ill and 36% vomited with 1 hour of the motion - although there was evidence of habituation to the motion over six exposures. Some drugs were found to be effective in reducing the incidence of illness and vomiting.

Tokola *et al* (1984) reported that a 24 hour study in life-rafts at sea in 'hard weather conditions' (e.g. 3 m waves) did not produce much vomiting because the volunteers were "*accustomed to heavy sea*". A similar study in a wave tank producing a frequency of 0.37 Hz with acceleration reported to be in the range 3 to 5 ms^{-2} produced vomiting in all 14 men exposed to the condition without any medication (drugs or placebo) within one hour on their first exposure (Glaser and McCance, 1959). Repeating the exposure five times at two day intervals reduced the incidence of vomiting.

Landolt and Monaco (1989) report that in four cases of crew abandoning oil rigs in totally-enclosed motor-propelled survival craft, sea sickness occurred in 75% or more of the occupants. The condition developed quickly and was severe in some cases.

3.2.2 Study by Handford

Handford *et al* (1953) report a survey of 638 men aboard a troop ship crossing the Atlantic Ocean from west to east. A gyroscope was used to measure roll and pitch while vertical motions were measured with accelerometers at four locations in the 186 metre vessel. The average roll frequency was 0.07 Hz while the average pitch frequency was 0.17 Hz. The vertical acceleration varied with location and with time. It is reported that the "*overall average*" vertical acceleration for a 7 hour period on the second day was 0.7 ms^{-2}. (This may be the average of the peak accelerations recorded over 5 minute periods every half an hour.) The average roll was reported as 1.9 degrees and the average pitch as 0.7 degrees.

The crossing was described as exceptionally smooth yet 35.5% of the men succumbed to sickness (i.e. they either vomited or were obviously seasick to an observer). The authors were unable to find any correlation between sea sickness and measures of the vessel motion. The rate of sickness reached a peak between 05.00 and 07.00 hrs, corresponding to reveille. (It is not possible to compute r.m.s. acceleration from an 'average peak acceleration', but if the r.m.s. acceleration is assumed to be 1.4 times lower than the 'average peak value', as for a sinusoid, a seven hour exposure to 0.7 ms^{-2} will yield a motion sickness dose value of 79 $ms^{-1.5}$ and a prediction of 26% of persons vomiting).

3.2.3 Study by Nieuwenhuijsen

Nieuwenhuijsen (1958) reported the results of a questionnaire survey of motion sickness among 423 passengers aboard a 149 metre vessel travelling across the Atlantic Ocean. (The accuracy of apparatus used to measure motion is unknown.) The vertical motion is reported to have had a dominant frequency of 0.14 Hz with a magnitude of about ±1 ms^{-2} when the sea was in a "*normal*" condition during the first 24 hours at sea. The motion increased over a period of 8 hours to more than ±4 ms^{-2}, when sailing in the tail of a hurricane, and then subsided over the next 36 hours. The number of persons motion sick increased from about 4% to 22% as the magnitude of the motion increased but did not reduce until almost 36 hours after the reduction of the motion (see Figure 2).

Assuming a vertical motion of 1 ms^{-2} r.m.s. (i.e. a peak acceleration of 4 ms^{-2} with a crest factor of 4) the motion sickness dose value procedure would suggest that 20% of unadapted persons would vomit within one hour. This calculation is highly dependent on the crest factor but a value of 4 has been obtained in some studies (see Griffin, 1990). The motion sickness dose value procedure is intended for short duration exposures and does not incorporate a recovery (or habituation) characteristic. Consequently, it implies that some passengers will become sick even if the magnitude of the motion reduces: this seems appropriate to the data reported by Nieuwenhuijsen. The data in Figure 2 suggest that the rate of recovery from motion sickness is slow and that in the conditions encountered by this vessel, the rate of recovery was matched by the rate of new cases for about 36 hours after the height of the storm. The author says that the average passenger needs two or three days to become "*adapted*".

Nieuwenhuijsen reported that females were more susceptible than males (in the ratio 2:3), that there was a non-linear decline in susceptibility with increasing age, and that

those taking anti-motion sickness drugs were more likely to be sick than those who did not take such drugs. The latter finding was attributed to both the higher susceptibility to sickness among those who took drugs and their tendency to wait until they felt ill before taking the tablets. The taking of anti-motion sickness drugs was twice as frequent in the females (73%) as in the males (37%).

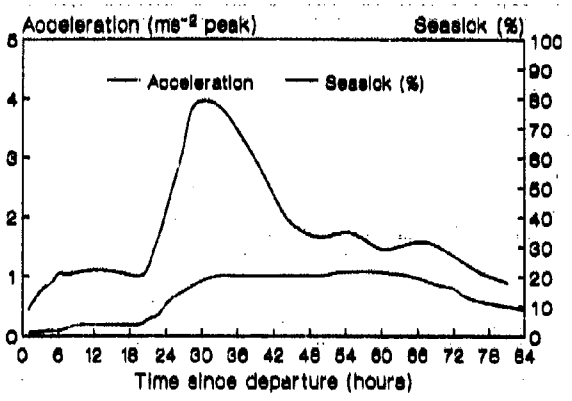

Figure 2 Variation in vertical motion and percentage of seasick passengers during 84 h voyage (adapted from Nieuwenhuijsen, 1958).

3.2.4 Japanese studies

During a 4 month voyage in the Pacific Ocean aboard a 97 metre sail training ship, 35 cadets (aged 18 and 19 years) with no recent sea experience were asked to report their motion sickness experience (Kanda and Yamagami, 1962). The vessel had dominant roll, pitch and vertical motions between 0.14 and 0.17 Hz. Using data from a later study (see below), Goto and Kanda (1977) reanalysed these results to quantify the decrease in vomiting with days at sea. They suggest that motion sickness incidence should be calculated from the product of two variables: a 'human response function' and an 'exposure effect function'. The human response function is determined from the acceleration raised to a suitable power (see below) while the exposure effect function is derived from the change in motion sickness on successive cruising days. They showed that serious motion sickness, and lesser symptoms, decreased as days at sea increased, with a straight line inverse relationship between days at sea and the logarithm of sickness incidence: the incidence of motion sickness symptoms fell to about a tenth over the first 10 days at sea.

A study of motion sickness among first year cadets with no previous experience of the sea during one month aboard a 115 metre training ship is reported by Kanda *et al* (1977). The cadets were monitored at half hour intervals during watches and during rest with the reported data coming from 2-day cruises during a one month period which also involved time at anchor and in port. Vertical motion was measured in the navigation bridge and is reported as r.m.s. acceleration with a frequency in the range 0.17 to 0.21 Hz. Observation over a period of 8 hours after leaving port, with a 4 hour watch commencing 2 hours after leaving port, showed that the incidence of motion sickness increased greatly during a period commencing half an hour before going on watch. Those with 'severe symptoms of motion sickness' increased from less than 5% before this period to more than 60% while on watch; those with 'any symptoms of motion sickness' increased from about 20% to about 80%. The incidence of motion sickness fell immediately after the end of the watch. In the example shown, the magnitude of vertical motion increased for a period shortly before the watch but subsequently remained almost constant at about 0.6 ms^{-2} r.m.s. The authors explain the fall in motion sickness incidence following the watch by the crew lying down or going to sleep. Goto (1981) links the increase before the start of the watch to the cadets waking up to prepare for tasks.

The study by Kanda *et al* (1977) also yielded graphs showing how the percentage of the cadets with sickness increased with exposure time during seven different periods at sea. Differences between voyages are explained by either differences in the magnitude of motion or the prior experience obtained during previous voyages. It is shown that the incidence of sickness continued to decline with up to 12 days at sea. Graphs are also provided of the maximum number of persons to suffer from motion sickness during a voyage as a function of the vertical motion present at the time when this incidence of motion sickness occurred (after about 2 to 3 hours). It is shown that for slight, moderate and severe motion sickness the incidence of sickness increased with the logarithm of the magnitude of the vertical acceleration, a (ms^{-2} r.m.s.):

$$\text{Percent with slight or worse symptoms} = 106 \log_{10} a + 104$$

$$\text{Percent with moderate or worse symptoms} = 108 \log_{10} a + 75$$

$$\text{Percent with serious symptoms} = 114 \log_{10} a + 56$$

These relations are illustrated in Figure 3 using both logarithmic and linear scales of acceleration. The similar gradients for the regressions between sickness and the logarithm of acceleration for the three degrees of sickness suggests that the use of only severe symptoms (i.e. vomiting) in other studies may allow the estimation of the relation between motion and lesser degrees of illness. However, also shown in Figure 3 are the values obtained for vomiting incidence produced by vertical oscillation in the laboratory at 0.166 Hz by O'Hanlon and McCauley (1974) and vomiting incidence predicted using the motion sickness dose value procedure (MSDV) for a two hour period as specified in British Standard 6841 (1987). The authors suggest the greater sensitivity to motion in their study at sea

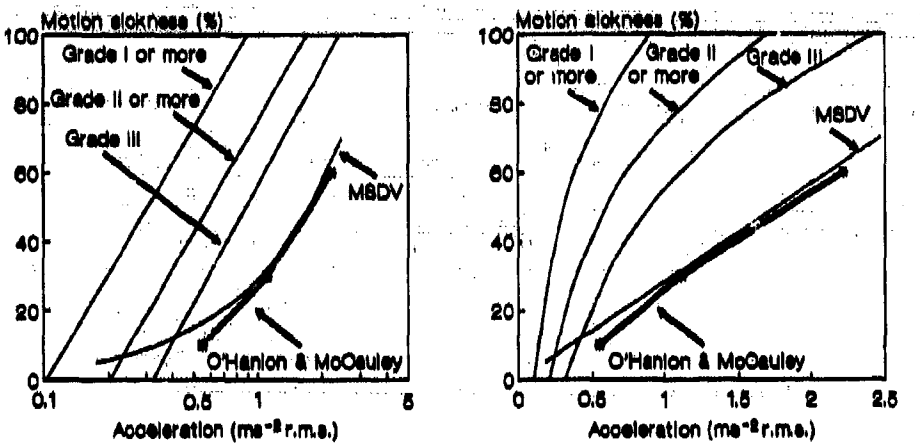

Figure 3 Variation in motion sickness with magnitude of vertical motion. [Data from Kanda et al, 1977; O'Hanlon and McCauley data (see Section 4.3) and MSDV predictions (see Section 4.2) for 2 h exposures to 0.166 Hz].

was due to the absence of roll and pitch motion in laboratory experiments. Other explanations are possible, including a different interpretation of the descriptors of motion sickness severity: the definition of serious symptoms in these sea trials is given as "*extremely unpleasant sensations, vomiting, loss of desire to do anything*", whereas the data from the laboratory experiments and the data used in the motion sickness dose value prediction are based solely on the incidence of vomiting. (The degrees of motion sickness used by Kanda *et al*, 1977 and shown in Figure 3 are: Grade I (slight) = "*somewhat unpleasant sensations, but capable of carrying on normal, unchanged life ...*"; Grade II (moderate) = "*considerable unpleasant sensations, occasional nausea, hard to do work or carry on other activities aboard ship*"; Grade III (serious) = "*extremely unpleasant sensations, vomiting, loss of desire to do anything*").

Further analysis of three one-month voyages consisting of two days at sea interspersed with two or three days at anchor around the coast of Japan is reported by Goto and Kanda (1977). Vertical motions measured aboard the 115 metre motorised ship were all in the range 0.4 to 0.7 ms^{-2} r.m.s. (dominant frequency 0.14 to 0.2 Hz). Groups of 40 to 60 merchant cadets with little sea experience participated in the study and were questioned about their symptoms of motion sickness at half hour intervals throughout watches. An analysis of data from the two or three hours of the first day of each voyage showed that the incidence of vomiting increased very rapidly with increases in the root-mean-square vertical motion: approximately 10% vomited with 0.45 ms^{-2} r.m.s. while about 50% vomited with 0.55 ms^{-2} r.m.s. The greater rate of increase in these field studies than in laboratory studies with vertical motion was attributed to the presence of motion in other axes (e.g. roll) and the infectious influence of sailors seeing others vomit. It was inferred from the data that, between 0.4 and 0.75 ms^{-2} r.m.s., the motion sickness incidence increased in proportion to the r.m.s. acceleration raised to the power of 4.5. Above 0.75 ms^{-2} r.m.s. it was assumed that there was 100% sickness incidence while below 0.4 ms^{-2} r.m.s. it was assumed that sickness incidence increased in proportion to r.m.s. acceleration raised to the power of 1.6. Compared with the earlier study it was observed that acclimatization took place more slowly when sea experience consisted of a series of short voyages as opposed to a continuous voyage. The authors concluded that the incidence of motion sickness reaches a peak within 2 or 3 hours and that there is both a fatigue effect and an acclimatization effect. They suggest that motion sickness does not, therefore, follow a simple time-dependency in which exposures are equally severe if the exposure times multiplied by the squares of their r.m.s. accelerations are similar. (Unlike some other studies, the measure of sickness used in this series of studies was the number of persons suffering at half hour intervals and not the cumulative number of persons to have suffered since the commencement of the voyage.)

Goto (1981) suggests a refinement to the above prediction method such that the power to which the r.m.s. acceleration is raised varies with the severity of sickness in addition to the magnitude of the motion. Formulae are also provided to predict the rise and fall in numbers of persons suffering from sickness as the voyage proceeds over a few hours and over several days. He suggests that habituation to motion is less on days when the magnitude of motion is low.

3.2.5 United States Coast Guard Studies

Wiker *et al* (1979a, 1979b, 1980) report a comparison of sea sickness among 18 Coast Guardsmen aboard three very different craft steaming side-by-side in sea state 3 over an octagonal course for 4 hours twice a day for three days. The craft were a 29 metre patrol

boat, a 115 metre cutter and a 27 metre small waterplane area twin hull (SWATH). The vertical motion on the patrol boat fell in the range 0.15 to 0.75 ms^{-2} r.m.s. (over the frequency range 0.2 to 0.45 Hz); the vertical motion on the cutter was in the range 0.08 to 0.27 ms^{-2} r.m.s. (0.12 to 0.37 Hz); vertical motion on the SWATH was in the range 0.1 to 0.37 ms^{-2} r.m.s. (0.12 to 0.4 Hz). Of 84 episodes of vomiting, 83 occurred in the patrol boat and one aboard the SWATH - a finding consistent with the higher magnitude of vertical motion in the patrol boat. Vomiting and lesser symptoms of motion sickness were greatest when steaming with a component of head seas. Multiple regression analysis of data from the patrol boat showed that motion sickness severity was greatest when motion frequencies were low and magnitudes were high; decreasing the magnitude of the motion or increasing the frequency of the motion resulted in a decrease in the severity of motion sickness. The authors suggest that the data support the view that motion sickness is primarily caused by vertical translational motion and not by roll or pitch motion.

A further study was conducted to compare responses of 11 Coast Guardsmen to the motions aboard the 29 metre patrol boat with the 27 metre SWATH during one day in dock and two days at sea (Wiker and Pepper, 1981). Again, there was more severe motion and more severe motion sickness aboard the patrol boat. Motion sickness severity increased during the eight hours at sea each day. Although the motions were similar on the patrol boat on both days at sea, the motion sickness was appreciably greater on the first day.

3.2.6 David W Taylor Naval Ship Research and Development Center Study

A four day sea-keeping trial on a 42.6 metre Coast Guard Cutter yielded data on ship motion and crew sickness while sailing around an octagon course seven times (Applebee *et al*, 1980). Each leg of the course lasted 30 minutes, commencing with head seas and subsequently turning 45 degrees to port until the octagon was completed. Vessel motions were measured in earth-referenced axes at the centre of gravity and in ship-referenced axes within the pilot house and the mess. The vertical motions and percentage of crew reporting sickness were greatest with head seas and least with quartering and following seas.

Significant sickness occurred among the small crew even in sea state 4. The percentage reporting to be mentally or physically impaired by either "*seasick only*" or "*seasick and excessive ship motions*" varied between 0 and 65%. Figure 4 shows the variation of this measure of sickness with heading for the six runs around the octagon for which it is available. Figure 4 also shows the equivalent variation in the motion normal to the deck surface in the crew mess. On some runs there is evidence of the severity of motion sickness symptoms lagging behind the severity of the motion but, generally, the motion sickness varies in a similar manner to the magnitude of vertical motion.

Figure 5 shows the relation between motion and motion sickness over the octagon course for the six runs. It is apparent that some habituation occurred during the trial so that the extent of symptoms decreased as the runs increased independently of changes in the magnitude of the vertical motion. The percentage reporting sea sickness is consistently greater than the percentage who would be expected to vomit based on 30 minute exposures to the reported motions during each leg of the octagon and the motion sickness dose value (see Section 4.2).

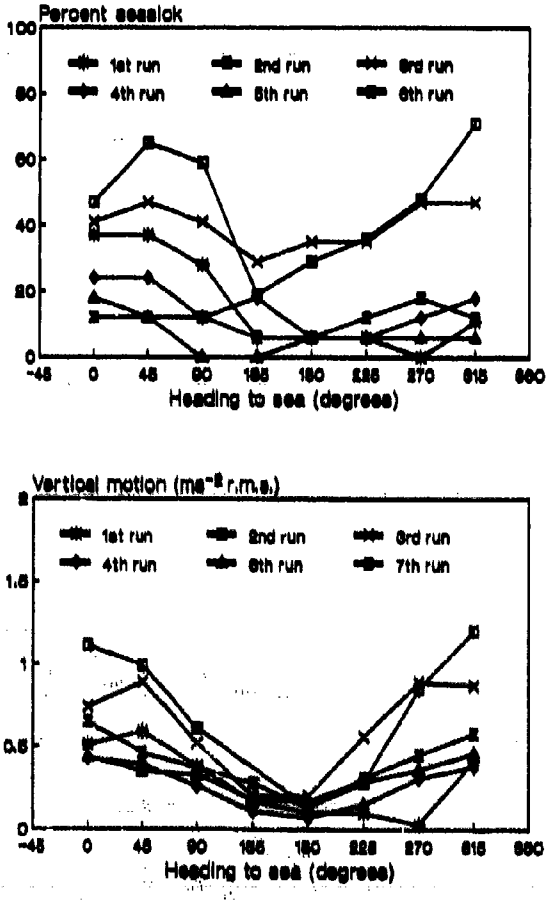

Figure 4 Variation in magnitude of vertical motion and motion sickness during six runs around an octagonal course in a 42.6 m Coast Guard Cutter. (From Applebee, 1980; 0 deg corresponds to head seas).

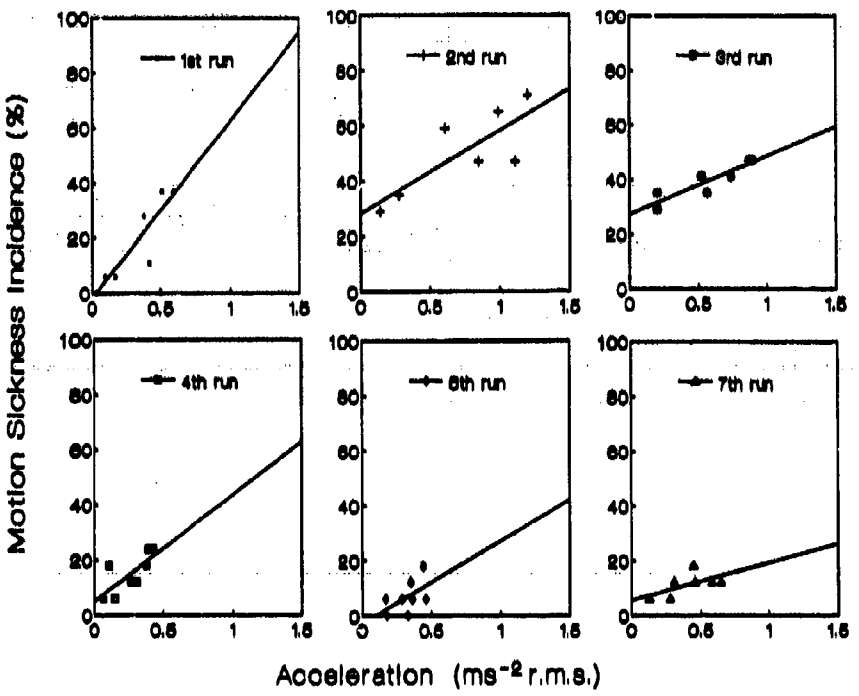

Figure 5 Relation between magnitude of vertical motion and motion sickness during six runs around an octagonal course in a 42.6 m Coast Guard Cutter (data from Applebee, 1980).

This may be partially due to the cumulative effects of motion experienced prior to each leg (there was appreciable transit time to the location for the trials and a 30 minute period of wave measurement before the first run). Additionally, the reported sea sickness responses do not necessarily imply vomiting as predicted by the motion sickness dose value.

Applebee and Baitis (1984) report sea trials aboard an 82 metre Coast Guard cutter during which both motion and the effects of motion were monitored. The magnitude of the motion and the extent of sea sickness varied with heading relative to the sea and the location of crew in the vessel.

3.2.7 Studies by the Institute of Sound and Vibration Research, I.S.V.R.

Studies of motion sickness among fare-paying passengers on ferries around the British coasts have been reported by Lawther and Griffin (1986, 1987, 1988a,b). The full series of investigations, which were conducted by the Institute of Sound and Vibration Research at the University of Southampton in England, yielded data from 20,029 passengers on 114 voyages aboard six ships, two hovercraft and a hydrofoil. Passenger characteristics and responses to motion were determined by means of questionnaires administered near the end of each voyage. Information obtained about passengers from the questionnaire included their regularity of travel, whether they had felt ill, when they had first felt ill, whether they had vomited, whether they had taken anti-seasickness tablets, how much alcohol they had consumed, where they sat on the vessel, their age and their gender. The illness rating was obtained from a four point scale: 'I felt all right', 'I felt slightly unwell', 'I felt quite ill', 'I felt absolutely dreadful'. In the subsequent analysis these responses were scored from 0 to 3 and the average determined over the groups surveyed. The vessel motions were measured in six axes (x-, y-, z-, roll, pitch and yaw) such that the instantaneous motion throughout every voyage at any location on any vessel could be determined. The motions were based on ship-referenced axes (not earth-referenced) since it was considered that these motions better represent the forces experienced by passengers.

Lawther and Griffin (1986) report results from 17 voyages on one vessel where the sea conditions varied from calm to very rough, the vertical motion varied from less than 0.1 ms^{-2} r.m.s. to almost 1.0 ms^{-2} r.m.s. and the incidence of vomiting varied from 0% to almost 40%. Figure 6 illustrates a typical 100 s period of ship motion on one voyage. The variation in motion with position in this vessel is discussed in Section 3.3 below. It was shown that there was a high correlation between the z-axis motion of the vessel and both vomiting incidence and illness rating. Although a significant percentage of passengers who

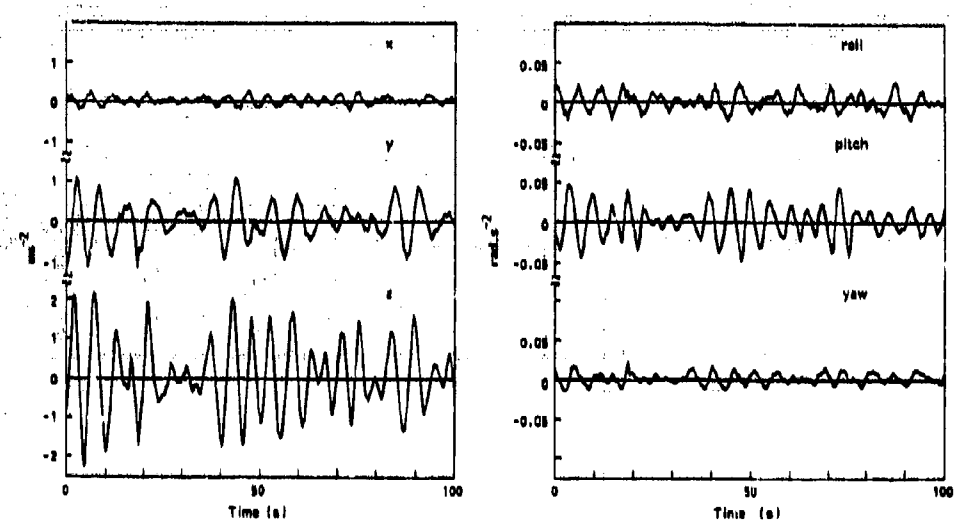

Figure 6 Acceleration time histories for a 100 second period of translational and rotational motion on a 4000 tonne, 99 metre passenger ferry as reported by Lawther and Griffin, 1986. [Accelerations relative to a ship-referenced co-ordinate system]

felt unwell did not vomit, there was a high correlation between vomiting incidence and illness ratings across voyages. It was found that the mean illness ratings increased with time at sea and, of course, the number to have vomited also increased as the voyages progressed. Figure 7 shows how the relation between vomiting incidence and vertical motion and the relation between illness rating and vertical motion changed as the voyages progressed. It was shown that the effect of exposure time could be incorporated within a measure of motion dose given by the integral of the acceleration raised to a power of either 2 or 4 with respect to time. A power of 4 (equivalent to the vibration dose value, see British Standard 6841, 1987; Griffin, 1990) provided the highest correlation coefficients (0.86 for vomiting and 0.88 for illness rating) but the advantage of this measure over the more easily calculated power of 2 (equivalent to r.m.s. acceleration) was not great (correlation coefficients of 0.83 for vomiting and 0.86 for illness rating).

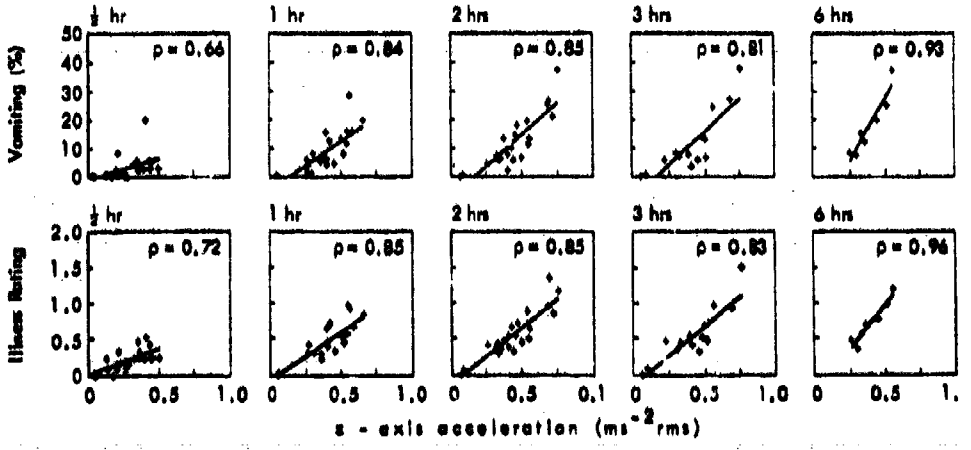

Figure 7 Percentage vomiting incidence and illness rating as a function of the magnitude of vertical motion for periods of ¼, 1, 2, 3 and 6 hours as reported by Lawther and Griffin, 1986. [Data obtained on a 4000 tonne, 99 metre passenger ferry; each point represents one voyage; Spearman's rank correlation coefficients also shown]

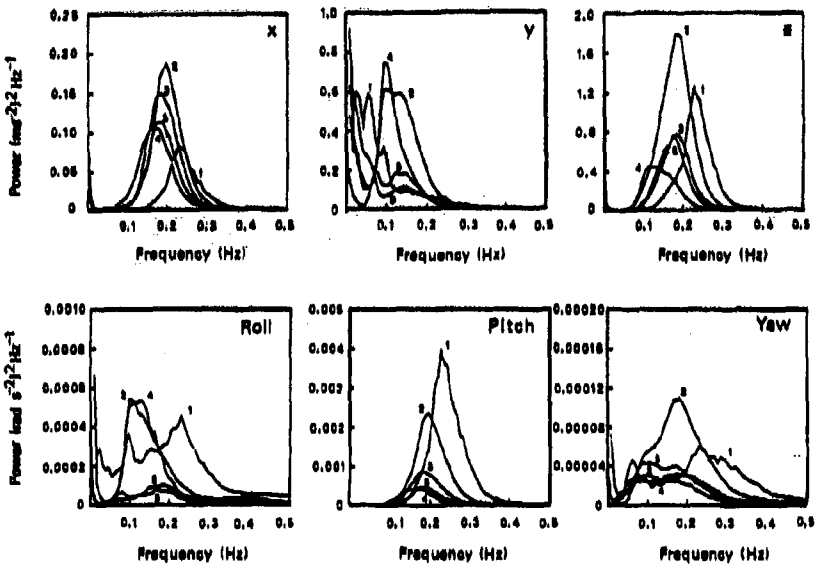

Figure 8 Averaged acceleration power spectral density functions for various voyages on five passenger vessels as reported by Lawther and Griffin, 1988. [Vessel 1: 67 metres; vessel 2: 99 metres; vessel 3: 109 metres; vessel 4: 121 metres; vessel 5: 130 metres. Accelerations apply to the centre of each vessel and are relative to ship-referenced co-ordinate systems. Frequency resolution 0.01 Hz.]

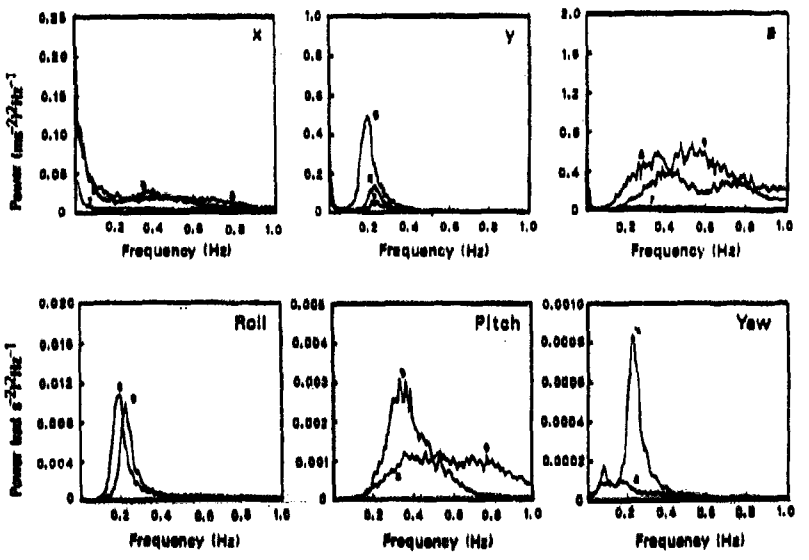

Figure 9 Averaged acceleration power spectral density functions for various voyages on hovercraft and a hydrofoil as reported by Lawther and Griffin, 1988. [Vessel 7: hydrofoil (translational axes only); vessel 8: 56 metre hovercraft; vessel 9: 37 metre hovercraft. Accelerations apply to the centre of each vessel and are relative to craft-referenced co-ordinate systems. Frequency resolution, 0.01 Hz.]

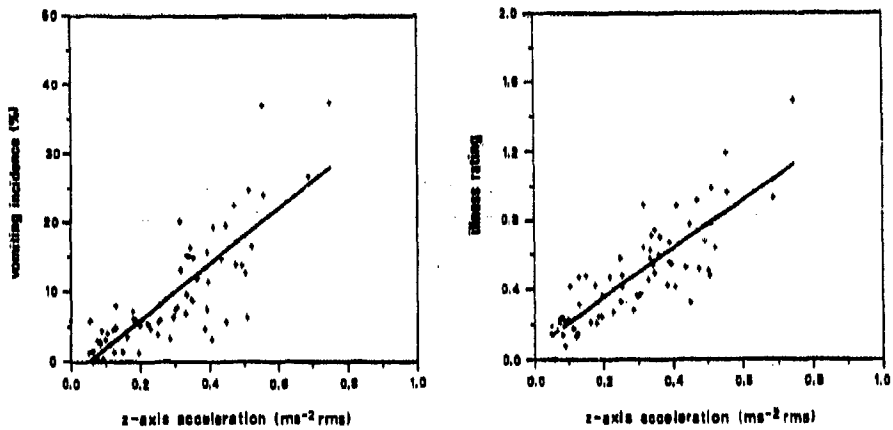

Figure 10 Vomiting incidence and illness rating as a function of the magnitude of vertical motion during 3 hour voyages on four different ships as reported by Lawther and Griffin, 1988a. [Each point represents one voyage]

A similar analysis using data from all vessels is reported by Lawther and Griffin (1988a). Figure 8 illustrates the average spectra for each axis of motion obtained from all voyages in five of the ships which varied from 67 to 130 metres in length and from 1255 to 7003 tons. Figure 9 shows similar averaged spectra for the two hovercraft and the hydrofoil - the hydrofoil data were only obtained in calm seas. Figure 10 shows the high correlation between r.m.s. vertical acceleration averaged over 3 hours and both the vomiting incidences and illness ratings on ships over the same period. The data suggest that, over a three hour period, a vertical motion of 0.6 ms^{-2} r.m.s. will result in about 20% of persons vomiting. The average illness rating for this exposure is about 1.0 - equivalent to everyone feeling 'slightly unwell', or 50% feeling 'quite ill' and 50% feeling 'all right', etc. Data from the hovercraft were shown to fit the same relation between the magnitude of vertical motion and sickness after the motion had been frequency weighted (using weighting W_f, see below) to reduce the importance of high frequency motion. Using this frequency weighting and the previously defined measures of motion dose it was again found that there was a slightly better correlation using a power of 4 (i.e. VDV) rather than a power of 2 (i.e. r.m.s.). However, the difference was again small and the authors conclude that both vomiting incidence and illness rating were linearly related to the r.m.s. vertical motion. Figure 11 shows these relations for all vessels on all voyages.

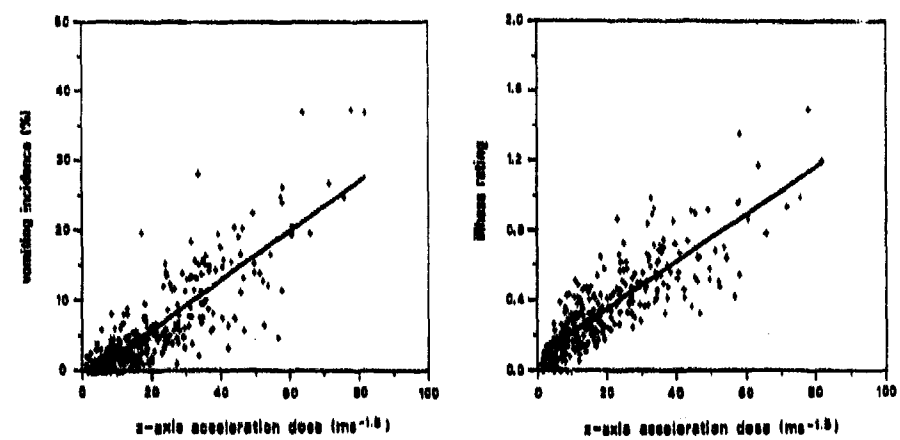

Figure 11 Vomiting incidence and illness rating as a function of the magnitude of vertical motion expressed as a dose: top figures, dose = $(\int a^2(t)dt)^4$; bottom figures, dose = $(\int a^2(t)dt)^4$. [Data from Lawther and Griffin (1988A) obtained with 6 ships, 2 hovercraft and 1 hydrofoil; hovercraft data frequency weighted. Each point represents one voyage.]

From the questionnaire data obtained in the above studies, Lawther and Griffin (1988a) report that, overall, 7.0% of passengers vomited at some time on their journey, 21.3% felt 'slightly unwell', 4.3% felt 'quite ill' and 4.1% felt 'absolutely dreadful'. Vomiting incidence and illness ratings were greatest in females (a male to female ratio of 3 to 5) and there was a slight decrease in sensitivity with increasing age (mostly due to increased sensitivity among those aged less than 15 years). Vomiting incidence and illness ratings were greater in passengers with least experience of sea travel. Twice as many passengers vomited in the group taking anti-motion sickness drugs as in the group not taking drugs - an effect attributed to a greater use of drugs among passengers who are more susceptible to motion sickness. Consumption of alcohol during the voyages was associated with a lower incidence of vomiting and illness - possibly because those susceptible to sickness are less likely to drink when exposed to motion.

3.3 Effect of position in vessel

Chinn *et al* (1953) observed that during drug trials on five crossings of the Atlantic in transport ships, the sea sickness was least frequent among those quartered amidships and increased among those quartered to the fore and those quartered to the aft. Handford *et al* (1954) reported a similar finding. From the results of 15 crossings of the Atlantic Ocean it was found that there was sickness in 17.9% of troops quartered midships, 23.7% among those quartered fore and 32.6% among those quartered aft (Anon,1956).

In their surveys of sea sickness on passenger ferries, Lawther and Griffin (1986, 1987, 1988a,b) measured the vessel motions so as to be able to calculate the different motions experienced by different groups of passengers. Figure 12 illustrates how the acceleration power spectra of motions on one vessel varied with longitudinal, lateral and vertical position within the vessel. Since vertical motion was shown to be the dominant cause of sea sickness, the variation from bow to stern is of greatest importance.

In ships, the least vertical motion (and lowest incidence of motion sickness) may be expected amidships - unless this location is worse for some other reason such as a more restricted visual field. On hovercraft there is appreciably more motion at the front and more sickness may be expected at this location (Lawther and Griffin, 1988b).

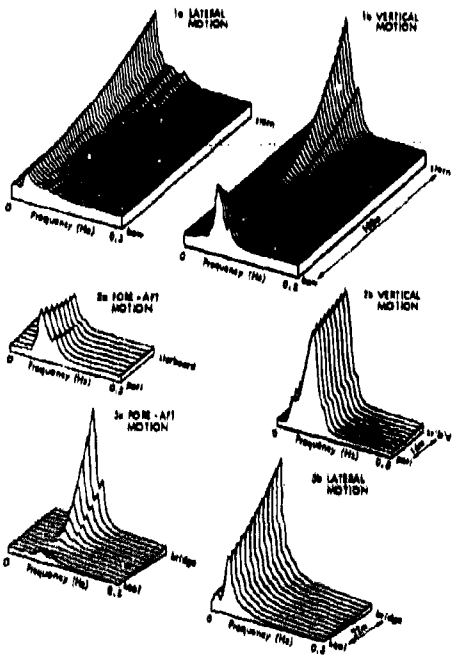

Figure 12 Variation in the acceleration power spectra of translational motion as a function of position in a ship (from Griffin, 1990).

3.4 Effect of subject characteristics

3.4.1 Gender

Two studies at sea have found females to be more susceptible to motion sickness than males, and similar findings have been reported in laboratory experiments (see Griffin, 1991). Nieuwenhuijsen (1958) reported that females were more susceptible than males (in the ratio 3:2). Using data from one ship, Lawther and Griffin (1986) found a ratio of 5:3. Lawther and Griffin (1988b) showed that the effect occurred in all age groups over 15 years and that it was not due to the increased use of anti-motion sickness drugs among females. Indeed, the effect was greater among passengers not taking drugs (a female to male ratio of 1.87 to 1) than among drug takers (1.15 to 1). In contrast, Levy and Rapaport (1985) report no difference between the males and females participating in a drug trial aboard large sailing yachts.

3.3.2 Age

Chinn *et al* (1952) observed that on a troop ship the incidence of vomiting declined with increasing age: when including those who received active drugs there were 33.6% who vomited in the age range 17 to 20 years while 14.2% of those in the range 30 to 39 vomited. Chinn *et al* (1953) again observed that the incidence of sickness on transport ships decreased with increasing age between 17 and 39 years. Handford *et al* (1954) found a similar trend with 33% vomiting incidence among troops less than 20 years of age and 13.3% vomiting among those over 40 years of age. Using data from troops on 15 crossings of the Atlantic Ocean, a clear age effect has been reported with 28.1% sick among those aged 17 to 19 years, 22.3% among those aged 20 to 24 years, 16.1% among those aged 25 to 29 years and 9.7% among those aged over 30 years (Anon, 1956). Nieuwenhuijsen (1958) reported a non-linear decline in susceptibility with increasing age.

Lawther and Griffin (1988b) found that both vomiting incidence and illness ratings among passengers below the age of 15 years were about double those of older passengers. Above the age of 15 years there was a slight decline in illness ratings with increased age but no change in vomiting incidence.

3.4.3 Vestibular function

Minor (1896) reported that he had observed deaf mutes to be immune to sea sickness and concluded that the problem was caused by "*irritation of the semicircular canals*". He mentioned some experimental results (involving rapid turning around) in support of the conclusion that a solution of cocaine dropped into the ears was a most valuable remedy for sea sickness. Among laboratory studies, Sjöberg (1931) reported that three deaf women failed to become sick when raised and lowered by a crane in a manner which caused others to become sick.

Kennedy *et al* (1968) compared sickness in twenty control subjects and 10 labyrinthine-defective subjects during a 28 hour voyage in a small vessel in very severe seas. While the labyrinthine defective subjects mainly experienced only drowsiness, 15 of the 20 control subjects vomited and the other five felt rather ill. The authors conclude that the presence of vestibular function is necessary for sea sickness.

3.4.4 Posture

Nollaston (1809) offered a theory of sea sickness based on the motion causing movement of the blood and intestines. He referred to a friend who achieved relief from sea sickness by lying on the deck with his head towards the stem of the vessel. It was suggested that with pitch motion of the vessel when lying in this posture he was in a position equivalent to descending backwards in a swing and, unlike descending forward in a swing, there was no tendency for the motion to force the blood towards the head. Irwin (1881) refers to "*the well-known fact that sea-sickness is least felt in the recumbent posture, with the head low and the feet towards the stern*" and offers an explanation based on the anatomy of the vestibular system and the intent of nature to provide for body equilibrium in a vertical, but not a horizontal, orientation. Brooks (1939) advocates a prone posture with the head raised very little, if at all, facing away from the sea.

Studies with more than 2100 soldiers on landing craft found that there was 25% to 42% prevalence of sickness when the men crouched for up to 3 hours but only 5 to 19% sickness when they were allowed to stand, apart from the last ten minutes before reaching the beach (Tyler, 1946). The increased sickness when crouched might be due to the different orientation of the head or the reduction in external vision.

Several studies have reported that motion sickness increases when crew leave their sleeping quarters (e.g. Handford *et al* 1953; Kanda *et al*, 1977; Goto, 1981). Studies of the effects of head position and body orientation on sickness induced by the drug apomorphine, revealed that vomiting was far less when subjects were supine (Isaacs, 1957). The benefit was shown to be due to the supine posture of the body and not from the changed orientation of the head.

3.4.5 Visual field

Looking at the horizon is commonly said to reduce the incidence of sea sickness, while working below deck with no external visual field will increase sickness. Studies at sea have not clearly separated the influence of the change in the visual field from the changes in the motion and the changes in body posture that occur with different locations. For example, Tyler (1946) found that a crouching posture gave rise to increased motion sickness on landing craft but was unsure of the extent to which the effect was due to the posture or the inability of the soldiers to see over the gunwales.

The widely accepted benefit of a view of the horizon is largely based on the personal experience of many sailors. Controlled studies have not been conducted to determine the precise requirements for the visual field to be beneficial or how this benefit could be simulated without an external view. The percentage of persons who benefit from 'viewing the horizon' is also unknown.

3.4.6 Activities

Some old accounts of sea sickness advocate the benefits of taking active exercise so as to lessen the signs and symptoms of motion sickness (e.g. Savory, 1901; Brooks, 1939). Among troops crossing the Atlantic Ocean the type of duty did not appear to affect the incidence of sickness (Anon, 1956).

3.4.7 Susceptibility to different types of motion

Kennedy and Graybiel (1962) exposed 21 subjects to motion in a slow rotation room, aerobatics in an aircraft and to heavy and calm seas in a 19 metre boat. They report a correlation between findings in the slow rotation room and sickness in heavy seas. Kennedy *et al* (1968) also report a significant correlation between the speed with which control subjects succumbed to sickness at sea and their susceptibility to sickness in a slow rotation room.

3.4.8 Placebo effect

It is often suggested that the administration of an alleged remedy may be sufficient to reduce the incidence or severity of motion sickness. Tyler (1946) found that in studies with 563 soldiers in landing craft those given a placebo drug suffered from sickness to a similar degree to those not provided with any medication. In contrast, during a study using waves generated in a tank, Glaser and McCance (1959) found that there was significantly less vomiting among men taking a placebo than among men taking no medication.

When passengers choose whether to take anti-motion sickness drugs there is greater sickness among those who take the drugs than among those who do not (Nieuwenhuijsen, 1958; Lawther and Griffin, 1988b). Passengers presumably take the drugs because they believe that they may reduce or eliminate sickness. The high incidence of sickness among drug takers suggests that neither the active component of such drugs nor the placebo effect of consuming the drugs are sufficient to counteract the increased susceptibility to motion sickness among those who believe it is both necessary and beneficial to take anti-motion sickness drugs.

3.4.9 Habituation

Various studies have shown that the incidence of sickness declines after a period of exposure to motion and two or three days is often quoted as the period required for significant habituation (see Glaser and Hervey, 1952; Bruner, 1955; Glaser and McCance, 1959; McCauley *et al*, 1976; Kanda *et al*, 1977; Goto and Kanda, 1977; Attias *et al*, 1987). However, a previous exposure to the motions of ships does not guarantee immunity to sea sickness. For example, a famous English Admiral, Horatio Nelson (1758 - 1805) was continuing to complain of sea sickness after a career in which he had spent more than 6300 days at sea.

3.4.10 Other effects

A trend towards greater sea sickness among heavier men was found in studies of troops crossing the Atlantic (Anon, 1956).

Lawther and Griffin (1988) found that passengers who had rarely or never travelled to sea before had a greater incidence of vomiting and greater illness ratings than those who travelled more frequently.

4. STANDARDS FOR ASSESSING SHIP MOTION WITH RESPECT TO SEA SICKNESS

4.1 International Standard 2631 Part 3 (1985)

This standard suggests the magnitudes of vertical motion in the frequency range 0.1 to 0.63 Hz which will cause vomiting in seated or standing young fit males. The magnitudes are specified for exposures of 30 minutes, 2 hours and, tentatively, eight hours. The magnitudes and durations are in an inverse-square relationship so that doubling the magnitude of the motion is equivalent to a four-fold increase in the exposure duration. The dependence of motion sickness on motion frequency given in this standard is such that sensitivity to acceleration is greatest between 0.1 and 0.315 Hz and then falls so that the acceleration magnitudes required to produce sickness at 0.63 Hz are 3.15 times greater than those required to produce sickness at 0.315 Hz and below (see Figure 14 below). The contents of this standard were formerly known as ISO 2631 Addendum 2 (1982).

4.2 British Standard 6841 (1987)

This standard defines a frequency weighting (W_f) to be used for assessing vertical acceleration over the frequency range 0.1 to 0.5 Hz. The weighting is formulated mathematically so that it can be incorporated within analogue or digital filters - but a simplified, straight line approximation is also defined. This has maximum sensitivity (i.e. unity gain) in the range 0.125 to 0.25 Hz where sensitivity is dependent on acceleration. Between 0.25 and 0.5 Hz, sensitivity falls at 12 dB/octave so that response is dependent on the displacement associated with the motion. The shape of the weighting was mainly influenced by laboratory studies, its derivation has been detailed by Lawther and Griffin (1987) and is compared with some laboratory data in Figure 13. Other information is provided in Griffin (1991).

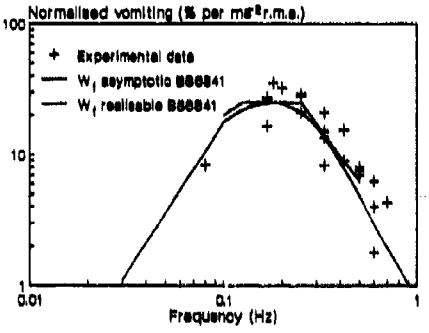

Figure 13 'Normalised vomiting incidence' for 2 h exposures & weighting W_f from British Standard 6841 (1987). [Data from McCauley et al 1976; normalised vomiting incidence = (% vomiting)/(r.m.s. acceleration, ms^{-2})]

British Standard 6841 also defines a means of predicting the incidence of sickness due to varying durations of z-axis vertical motion. A 'motion sickness dose value', $MSDV_z$, is defined:

$$\text{motion sickness dose value, } MSDV_z = a_{rms}t^{1/2}$$

where a_{rms} is the root-mean-square acceleration (in ms^{-2} after frequency weighting using weighting W_f) determined by linear integration over the period t (seconds).

The standard says that the percentage of unadapted adults who will vomit is given by:

$$\text{percentage who may vomit} = {}^1/_3\ MSDV_z$$

Doubling the magnitude of the motion, or a four-fold increase in exposure duration, will therefore double the predicted incidence of vomiting. The standard states that the above relations are based on exposures lasting from about 20 minutes to six hours with a

prevalence of vomiting up to 70%. Figure 14 shows the root-mean-square acceleration required for 10%, 20% and 40% of persons to vomit due to 2 hour exposures to motions in the frequency range 0.1 to 0.5 Hz. The peak displacements corresponding to these values are shown in Figure 15.

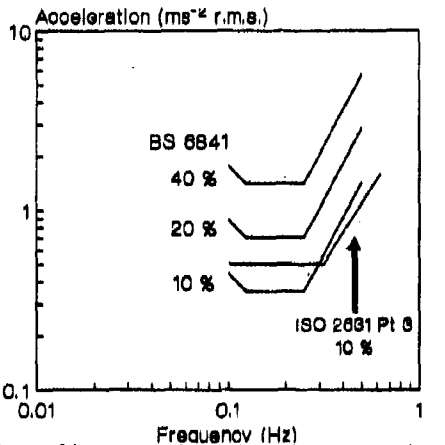

Figure 14 Vertical acceleration expected to cause 10%, 20% and 40% vomiting during 2 h exposures from British Standard 6841 (1987). (10% vomiting incidence in 2 h according to ISO 2631 Pt 3 also shown. Figure from Griffin, 1990).

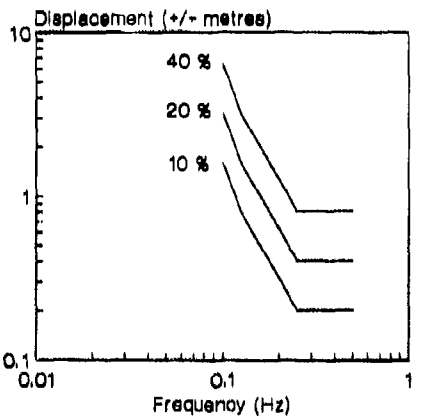

Figure 15 Peak displacements of vertical sinusoidal oscillation required to produce 10%, 20% and 40% vomiting incidence during 2 hour exposures according to British Standard 6841 (1987). [Figure from Griffin (1990)]

4.3 Other methods

The data from various studies, and common experience, suggests that problems from motion sickness are greatest on smallest vessels. Pethybridge (1982) reported the results of a survey of motion sickness among crews of various British Royal Navy vessels and determined mathematical relationships between the incidence of sickness and vessel size. For example, with displacements of 200 tons, 1000 tons, 5000 tons, 10000 tons and 20000 tons the predicted incidence of motion sickness was 67%, 62%, 50%, 41% and 29% respectively. In this study, motion sickness incidence included any of the varied symptoms of motion sickness and was not restricted to vomiting.

Only two series of studies at sea have resulted in suggested methods of assessing ship motion with respect to motion sickness. The findings from the studies by Lawther and Griffin are embodied in the motion sickness dose value procedure defined in British Standard 6841 (1987). The studies of Goto *et al* led to a prediction method which is somewhat more complex but has some similarities. Unlike other procedures, the method allows for the percentage of sick persons to fall during a voyage and for motion sickness to decrease on subsequent voyages (see Section 3.2.4 above).

From the results of laboratory studies of motion sickness caused by vertical oscillation, a series of formulae for predicting motion sickness incidence (MSI) were proposed by O'Hanlon and McCauley (1974), McCauley and Kennedy (1976) and McCauley *et al* (1976). The method applies to motion in the frequency range 0.08 to 0.63 Hz with maximum sensitivity to acceleration at about 0.16 Hz. The assumption that MSI will vary with acceleration and with time in ogival form (i.e. a cumulative normal distribution) resulted in mathematical operations somewhat more complex than those required to calculate vomiting incidence using the motion sickness dose value. Motion sickness incidence (MSI), expressed as a percentage, is assumed to be the product of a term representing the influence of motion magnitude and frequency, P_A, and a term expressing the effect of motion duration, P_t:

$$MSI = 100\ P_A\ P_t$$

The term P_A is calculated from a term, z_A, quantifying the effect of magnitude and frequency and a term describing the form of a cumulative normal distribution (in practice this may be obtained from statistical tables). The effect of magnitude and frequency was determined from a curve describing the acceleration required to produce vomiting at various frequencies in 50% of persons during two hour exposures. The mathematical expression may be written as:

$$z_A = 2.13 \log_{10} a - 9.28 \log_{10} f - 5.81 (\log_{10} f)^2 - 1.85$$

Where a is the r.m.s. acceleration in g; f is the frequency in Hz. The term P_t is calculated similarly to give a value for z_t:

$$z_T = 2\log_{10}t + 1.13\,z_A - 2.90$$

where t is the exposure time in minutes.

Values of P_A and P_T are obtained by consulting a table of the normal deviate z at the values of z_A and z_T respectively. For example, with a 60 minute exposure to an acceleration of 2.1 ms^{-2} r.m.s. (i.e. 0.21 g r.m.s.) vertical sinusoidal motion at 0.25 Hz, $z_A = 0.19$ and $z_T = 0.87$ so $P_A = 0.57$ and $P_T = 0.81$ giving a predicted motion sickness incidence of 46%.

Lawther and Griffin (1987) compared the extent to which predictions derived from the above procedure for calculating motion sickness incidence (MSI) differed from the procedure for calculating vomiting incidence from the motion sickness dose value ($MSDV_v$). It was shown that the frequency weightings were similar and that the dependence of vomiting on acceleration magnitude and duration were similar for magnitudes up to about 2.5 ms^{-2} r.m.s. and durations up to about 6 hours. It appeared that the differences between the methods were not sufficient for the available data to identify either method as being appreciably more accurate than the other. The MSI procedure has an advantage of not being capable of predicting a vomiting incidence greater than 100%, although in practice this does not appear to be a significant problem with the motion sickness dose value procedure. The motion sickness dose value procedure may fit the available data slightly better at low incidences of sickness and allows the prediction of illness rating as well as vomiting incidence. Additionally, the motion sickness dose value is easier to calculate and provides a convenient standardised method of quantifying low frequency motion.

5. PREVENTION OF MOTION SICKNESS

A wide variety of drugs, potions and behaviours have been advocated for lessening the problems of sea sickness. Many recommendations appearing in the early scientific literature appear to be based on personal experience or anecdote and are not supported by controlled studies. Today, many individuals going to sea still adopt procedures that have not been proven effective by conventional scientific methods. Statistically significant benefits to groups of travellers from the consumption (or avoidance) of certain foods and drinks or the use of various commercial devices have yet to be proven.

Some behaviourial changes may help minimise the likelihood of sea sickness. Those that can be recommended with most confidence involve minimising exposure to low frequency vertical motion, adopting a position where there is a distant external view, minimising motion of the head and eyes, or adopting a recumbent posture. In general, learning the true nature of the complex vessel motion is likely to be beneficial; this may minimise conflict between information from different sensory systems and harmonise the various reflex responses to motion.

Many studies of the effectiveness of drugs in minimising motion sickness have been conducted using laboratory apparatus. Most laboratory studies have been conducted using Coriolis stimulation (see Griffin, 1991). The sensory mechanisms involved in the production of motion sickness with this type of motion may not be the same as those involved in sea sickness. The studies reported here are restricted to investigations conducted at sea.

Holling *et al* (1944) report an investigation of various drugs on minesweepers and trawlers. Drugs containing atropine, hyoscyamine, and hyoscine provided some protection with the greatest protection coming from hyoscine (i.e. hyoscine hydrobromide, also called scopolamine hydrobromide): 57% were protected with a 0.6 mg dose and 73% were protected with 1.2 mg. Hill and Guest (1945) found hyoscine was the most effective drug with about 80% of soldiers in landing craft protected by a dose reported to be 10 mg. Tyler (1946) also found that hyoscine (alone or combined with hyoscyamine, atropine, or barbiturates) afforded protection in landing craft.

From studies aboard troop ships crossing the Atlantic Ocean, Chinn *et al* (1950) concluded that hyoscine, diphenhydramine hydrochloride, dimenhydrinate, trihexyphemidyl and chlorcyclizine were all effective in reducing sea sickness. Chinn *et al* (1952, 1953) found useful protection with a variety of other active drugs on many further voyages. Handford *et al* (1954) reported a study conducted on a voyage across the Atlantic Ocean which included a comparison of hyoscine, benadryl and postafene. While 50 mg of benadryl and 50 mg of postafene were effective, a dose of 1 mg of hyoscine provided no significant protection from sea sickness. A later study (Anon, 1956) found some protection with hyoscine but the greatest benefit was obtained with either 50 mg of meclizine, 50 mg of cyclizine, or 25 mg of promethazine. On three trans-Atlantic voyages, Trumbull *et al* (1960) again found significant benefits from 50 mg doses of both cyclizine and meclizine; 2.5 mg of phenglutarmide and 7.5 mg of dihnarazine also afforded some protection.

Glaser and Hervey (1951) report that 1 mg of hyoscine afforded protection to 96%, 25 mg of phenergan protected 61%, and 25 mg of benadryl protected 44% of men on small boats from vomiting. Using artificial waves in a swimming pool, Glaser and Hervey (1952) found that 1 mg of hyoscine (and 0.6 mg hyoscine with 15 mg promethazine) were more effective than 35 mg promethazine hydrochloride. A 1 mg dose of hyoscine given only five to ten minutes before the exposure to motion was effective, but less effective than when administered 75 minutes before the exposure. Glaser and McCance (1959) compared four drugs with soldiers within tented rubber floats in a wave-tank. Again, 1 mg of hyoscine provided

the greatest protection from vomiting (81%). A 50 mg dose of cyclizine hydrochloride protected 58% whereas 25 mg of meclozine hydrochloride and 8 mg of perphenazine were no better than a placebo.

Among participants in an ocean youth sailing club, Hargreaves (1980) reported that 'seasickness' was experienced by 57% of a group provided with a placebo and by 26% of those provided with 15 mg of cinnarazine. Treatment consisted of two doses one or two hours before the start of cruises and one dose every six to eight hours at sea. A similar procedure found that subject reports of drug effectiveness were similar for 15 mg of cinnarazine and 0.3 mg of hyoscine (Hargreaves, 1982). A study on life-rafts at sea for 24 hours found that both 0.3 mg of hyoscine and 0.3 mg of hyoscine combined with 25 mg ephedrine hydrochloride provided useful protection from sea sickness (Tokola *et al*, 1984).

Levy and Rapaport (1985) report benefits of hyoscine when administered transdermally at least eight hours before sea travel. Athlas *et al* (1987) found that protection provided by transdermal hyoscine was 74%, 73% and 39% over three days at sea on a 3000 ton vessel. A study of long term use of transdermal hyoscine at sea has also reported benefits (Shupak *et al*, 1989).

The effectiveness of drugs depends on the conditions and the time of consumption. Most drugs are said to be suitable for administration about half an hour before exposure to motion, but cinnarizine should be taken about 2 hours before exposure. It is possible that while some may have a protective action others may be more effective therapeutically.

Many drugs have unwanted effects which vary between individuals and range from trivial discomfort to serious interference with activities. The most common unwanted effects are dry mouth, drowsiness and headache. The wanted and the unwanted effects of drugs may have non-linear dependencies on dose, vary between individuals and depend on the consumption of food and drink. The time of administration and method of administration (oral, transdermal or injection) may also influence effectiveness. The preferred drug for a single dose may not be appropriate for continued administration over several days.

6. DISCUSSION AND CONCLUSIONS

Laboratory studies have shown that a wide variety of motions can give rise to motion sickness. Sickness can be induced by translational oscillation (in the vertical, lateral or fore-and-aft directions), by rotation about a vertical axis, by continuous rotation about an off-vertical axis, by rotational oscillation, by head movements when rotating about a vertical axis, and by some other conditions - including motion of the visual scene (see Griffin, 1991).

It is generally concluded that the vertical motion on ships is primarily responsible for sea sickness. This conclusion arises from evidence that vertical motion alone is sufficient to cause the sickness while other motions alone are generally insufficient to cause sickness.

From data obtained during a range of voyages, Lawther and Griffin (1988a) found high correlations between the magnitudes of motion in all six axes on vessels: when the sea was rough the motion tended to increase in all axes. Correlations between motion sickness (vomiting prevalence or illness rating) and the magnitude of motion were high for all axes, but the z-axis motion and the pitch motion gave the highest correlations. Using multiple regression analysis, after including the z-axis, the addition of other axes into the regression only marginally improved the overall regression coefficient. Lawther and Griffin (1987) showed a high correspondence between motion sickness data obtained at sea and data obtained in the laboratory by considering only vertical oscillation. Figure 16 compares vomiting incidence as a function of motion sickness dose for the Lawther and Griffin sea studies, the laboratory data obtained by Alexander *et al* (1947) and the laboratory data reported by McCauley *et al* (1976). This figure suggests that laboratory data and sea data fall about the same regression line, with most of the laboratory data giving high vomiting incidences and most sea data giving lower vomiting rates.

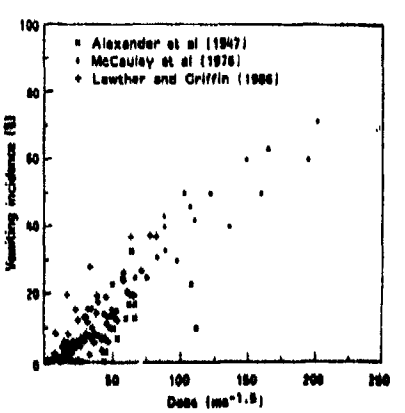

Figure 16 Relation between motion sickness dose value and vomiting incidence for laboratory studies with vertical motion reported by Alexander et al (1947) and McCauley et al (1976) and sea studies reported by Lawther and Griffin (1986).

The vertical motions of vessels are partially caused by pitch motion, so sickness will reduce if the pitch motion is reduced. However, the magnitude of pitch motion is usually very small (sometimes below the threshold of perception of rotation) and there is no evidence that the pitch motion experienced on ships is sufficient to cause sickness

independently of its action in causing vertical motion. The magnitudes of motion, and the visual conditions that appear to be required to cause sickness with oscillation about a horizontal axis, suggest that roll and pitch motion can often be discounted as prime causes of sea sickness (see Griffin, 1991).

Roll motion of ships does not normally greatly contribute to the vertical motion and is unlikely to contribute to sea sickness as much as the vertical motion or the pitch motion. While the provision of roll stabilisers on vessels may have various benefits (e.g. assisting the postural stability of crew and passengers, the stability of cargo, the accuracy of weapon aiming, and reducing the difficulty of aircraft deck landings) they have not been shown to reduce motion sickness.

When using ship-referenced axes, the magnitudes of x-axis and y-axis motion are influenced by the pitch and the roll of the vessel, respectively. An accelerometer orientated in these axes (i.e. parallel to the deck) will sense a gravitational component proportional to the *sin* of the angle of pitch or roll. Data presented by Lawther and Griffin for five passenger vessels show that the magnitudes of x-axis motion were generally less than the magnitudes of y-axis motion which were generally less than the magnitudes of z-axis motion when using ship-referenced axes (see Figure 8). The relative magnitudes varied between vessels and the y-axis component (arising partly from roll) can sometimes be greater than the z-axis component. However, in ships, the dominant frequency of roll (producing y-axis motion) is lower than the dominant frequency of pitch (producing z-axis motion). Consequently, even if the body were similarly sensitive in all three translational axes, it would not follow that y-axis acceleration arising from roll motion would contribute greatly to sickness. Also, the human body may remain upright while the vessel rolls - so lessening the lateral component due to gravity. Dose-effect data for horizontal oscillation are currently insufficient to identify the role of horizontal acceleration arising from rotational acceleration in causing motion sickness - so it is not known whether earth-referenced or ship-referenced axes give a better indication of the severity of horizontal motion on vessels. Some laboratory experiments have been conducted with roll motion about an axis well below the body so as to cause both roll and horizontal translation of the body (e.g. McCauley *et al*, 1976). The results of these experiments suggest that when a horizontal component of acceleration arises as a result of rotation about a horizontal axis it may not be as nauseogenic as a similar acceleration occurring solely in the vertical direction. This would suggest that the measured values of x-axis and y-axis motion using ship-referenced axes are not as nauseogenic as the corresponding measures of z-axis motion.

Complex combinations of vessel motions are sometimes credited with special powers of causing sickness. Corkscrew motions caused by seas on the bow or quarter, for example, involve a combined rolling, pitching and yawing motion. Irwin (1881) commented that "*many persons who experience no inconvenience during the regular swing of a yacht under sail become sick in a rowing boat or a steamer*". He attributed the difference to the "*irregularity and uncertainty*" of the movement. Varying the phase relationships between motions in different axes can give rise to perceptibly different motions which some mariners blame for sickness. However, there appear to have been no studies showing that phase is important and it remains possible that changes to the frequency, the magnitude or the direction of the motion may be primarily responsible for many claimed variations in the provocative nature of some motions. Additionally, since habituation to motion is motion-specific, some of the reports may be due to inadequate habituation to a rarely encountered motion rather than special nauseogenic properties of the motion.

Non-vertical motions might be hypothesised as having a synergistic effect so that sickness is greater than would be expected from the sum of the sickness due to vertical motion alone and the sickness due to the other motions alone. Currently available data suggest that this is not the case and indicate that sea sickness is mainly due to vertical oscillation. Useful predictions of the incidence of vomiting and illness can be made if only the magnitude and frequency of the vertical motion and the duration of exposure are known.

Although vertical motion appears to be the prime cause of sickness at sea it is likely that other factors have an influence even though their effects cannot yet be quantified. A greater understanding of the influence of vision, body posture, head movements and habituation may assist the reduction of sea sickness among passengers and crew.

Sickness is widely accepted as a natural consequence of going to sea. Little effort has been expended in seeking a solution to sea sickness other than by the use of anti-motion sickness drugs. The long history of sea sickness may have led some to the conclusion that scientific study should not be wasted tackling an insoluble problem. However, the extent of the problem, and the hitherto scant scientific attention it has received, suggests that it is a problem meriting more investigation. Scientific study will determine the extent to which the problem is soluble when understanding has been increased.

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EVALUATION ET PREDICTION DE LA SUSCEPTIBILITE AUX CINETOSES

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RESUME: L'évaluation et la prédiction de la susceptibilité individuelle aux cinétoses présentent de multiples difficultés. Celles-ci trouvent leur origine dans les particularités étio-pathogéniques du syndrome telles que la mise en jeu rapide de processus d'habituation, la distribution non-gaussienne de la susceptibilité dans la population, la spécificité des stimulus déclenchants. Ces caractéristiques entraînent la nécessité d'une grande rigueur méthodologique pour toutes les études traitant de ce sujet.

Trois grandes techniques méthodologiques sont habituellement utilisées : les questionnaires, la recherche de corrélations psycho-physiologiques indirectes et les tests provocateurs.

L'utilisation des questionnaires anamnestiques présente des limitations importantes. Les corrélations psycho-physiologiques indirectes sont rares et généralement peu fiables sur le plan de la prédiction individuelle.

Les épreuves provocatrices en laboratoire restent à l'heure actuelle la meilleure méthode d'évaluation de la susceptibilité. Elles posent cependant le problème de la nature des stimulations employées ainsi que celui de la fiabilité des critères d'évaluation de la sévérité. A côté des critères subjectifs habituellement utilisés (échelles d'observation ou d'auto-estimation), des efforts sont déployés pour définir des paramètres physiologiques objectivement mesurables.

Ces tests conduisent généralement à une prédiction correcte de la susceptibilité en situation réelle. La prédiction du mal de l'espace constitue cependant une exception à cette règle et pose des problèmes encore mal résolus aujourd'hui.

1. INTRODUCTION

Depuis la second conflit mondial, la susceptibilité aux cinétoses a suscité de nombreux travaux, donnant lieu à une littérature extrêmement abondante, comportant parfois des résultats relativement contradictoires. Ceci s'applique tout particulièrement aux aspects d'évaluation et de prédiction de la susceptibilité individuelle. Il est en effet bien connu que de très larges variations sont rencontrées dans la susceptibilité vis à vis des stimulations provoquant l'apparition du syndrome. Alors qu'une partie de la population semble pratiquement immune, l'autre est affectée plus ou moins gravement avec un retentissement parfois important sur la motivation et l'efficacité. La détection des individus présentant une sensibilité importante et, plus généralement, l'évaluation de la résistance des personnels aux environnements nauséogéniques présente donc un intérêt particulier en milieu aéronautique et spatial. Il s'agit cependant là d'un domaine complexe et difficile qui requiert une grande prudence dans l'interprétation des résultats obtenus, quelle que soit la technique d'évaluation employée.

Outre l'évaluation de la susceptibilité individuelle, l'estimation "a priori" de la capacité nauséogénique d'une stimulation vis à vis d'une population exposée, c'est à dire la prédiction de l'incidence des cinétoses constitue un autre problème. Plusieurs études ont également été menées dans ce domaine (1), appliquées en particulier aux stimulations rencontrées en navigation maritime. Cet aspect ne sera cependant pas étudié ici et seules l'évaluation et la prédiction de la susceptibilité individuelle seront envisagées.

La revue exhaustive des résultats très divers obtenus jusqu'à présent serait sans aucun doute très longue et fastidieuse. Aussi, l'approche du problème se veut ici avant tout synthétique et didactique, au risque de paraître relativement réductrice par rapport à la masse des connaissances qui ont été accumulées dans ce domaine. Les difficultés d'interprétation des données de la littérature sont pour une part importante liées aux différences dans les conditions expérimentales employées, celles-ci rendant parfois les comparaisons très difficiles. Dans une revue récente (9), Kennedy s'est attaché à résoudre les apparentes contradictions qui pouvaient exister entre diverses études en proposant une approche fondée sur l'analyse statistique.

L'emploi de techniques statistiques représente un élément tout à fait fondamental quant à la validité des méthodes de prédiction et des critères d'évaluation de la susceptibilité vis à vis des cinétoses (29, 9). Elles demeurent cependant pour une grande part du domaine des spécialistes et, du point de vue du physiologiste, une approche plus concrète et pragmatique apparaît donc préférable. Ceci conduit à envisager en premier lieu les différents éléments qui contribuent aux difficultés rencontrées en matière d'évaluation et de prédiction. Les différentes méthodes et techniques employées seront ensuite passées en revue, de façon à effectuer une synthèse globale illustrée de quelques exemples pratiques.

2. LES DONNEES DU PROBLEME

Si quelques grandes lignes peuvent être dégagées quant aux facteurs généraux influant sur la susceptibilité aux cinétoses, l'évaluation de la susceptibilité d'un individu en particulier présente plusieurs difficultés.

En suivant le modèle présenté par Benson (1), quelques points intéressants peuvent être recensés. C'est tout d'abord le problème de la spécificité des stimulations provocatrices, bien connu pour ce qui concerne l'adaptation, mais qui peut également jouer au niveau de la susceptibilité individuelle. C'est aussi la mise en jeu généralement très rapide de processus d'adaptation, si bien que toute tentative d'évaluation de la susceptibilité à un instant donné doit être pondérée en tenant compte de ce facteur. Enfin, il ne faut pas oublier que le syndrome de cinétose s'exprime par la mise en jeu du système nerveux autonome et que les caractéristiques intrinsèques de fonctionnement de ce système vont également exercer une influence sur la susceptibilité. Ce sont finalement les deux versants du syndrome, neurosensoriel et neurovégétatif, qui doivent être considérés pour évaluer au mieux la susceptibilité d'un individu et éventuellement tenter de prédire son comportement dans la situation réelle où il doit évoluer.

Pour Reason (26), ceci se traduit globalement en termes d'adaptabilité, de réceptivité et de capacité de rétention. D'une manière plus générale, on peut considérer que la distribution non-gaussienne de la susceptibilité dans la population correspond sans doute à la combinaison des variables qui peuvent influer aux différents niveaux sur la sensibilité individuelle.

Ces différents points vont donc être maintenant abordés successivement de façon plus détaillée pour tenter de mieux faire comprendre les difficultés liées à l'évaluation et à la prédiction.

2.1 Spécificité des stimulus

On admet classiquement (22, 27) que les sujets très susceptibles aux cinétoses sont sensibles à l'ensemble des stimulations nauséogéniques qui peuvent leur être appliqués, celles-ci entraînant, d'une manière plus ou moins identique, l'apparition des symptômes caractéristiques des cinétoses. Ceci représente la base des techniques d'évaluation utilisant des épreuves provocatrices.

Cette règle n'est pas totalement vérifiée, notamment pour la susceptibilité au mal de l'espace (28). De même, Money et Coll. (23) ont rapporté qu'avec des sujets dont la sensibilité est intermédiaire ou faible, on pouvait observer des dissociations entre différentes stimulations, en particulier entre les cinétoses induites visuellement (prismes inversants) et celles induites par des stimulations vestibulaires (Coriolis, rotations autour d'un axe horizontal,...). Plus indirectement, Leger et Sandor (15) ne relèvent pas de corrélation entre la susceptibilité aux cinétoses évaluée au moyen d'un questionnaire dans une population de pilotes et leur sensibilité au mal des simulateurs.

Il pourrait donc exister des éléments de susceptibilité préférentielle vis à vis d'un ou de plusieurs types de stimulation. On peut faire l'hypothèse que ce type de susceptibilité individuelle pourrait être lié aux caractéristiques de fonctionnement des capteurs (tout particulièrement des transducteurs vestibulaires) ou aux modalités d'intégration multisensorielle des informations d'orientation spatiale. Quoi qu'il en soit, c'est ici le "versant neurosensoriel" du syndrome de cinétose qui serait directement en cause.

Un autre aspect de la susceptibilité individuelle est manifestement lié à l'expérience préalable d'un individu donné au moment où l'on cherche à évaluer sa tolérance. Ainsi, il a pu être observé anecdotiquement que des personnels issus de la marine, habituellement résistants au mal de mer, révélaient une sensibilité relativement plus élevée vis à vis de stimulation du type accélérations de Coriolis qu'une population de pilotes de chasse. L'habituation préalablement acquise pour un type de stimulation (accélérations verticales), ne se transfère pas ou peu dans la nouvelle situation choisie pour évaluer la susceptibilité de la population. Il est donc clair que l'interprétation de résultats d'évaluation, quelle que soit la méthode utilisée, devrait prendre en compte, s'il s'agit d'une sélection, les éventuelles différences liées à l'expérience des candidats.

2.2 Mise en jeu des processus d'habituation

L'habituation aux stimulations provocatrices est un point central du problème des cinétoses. L'apparition de ce syndrome est pour certains un effet secondaire de la mise en jeu de la plasticité nerveuse requise pour cette habituation (9, 25). L'habituation résultant de l'exposition à l'environnement ou aux stimulations de laboratoire survient plus ou moins rapidement selon les individus. Reason distingue à ce niveau des "adapteurs" rapides ou lents (26), certains semblant ne jamais atteindre un niveau satisfaisant (21). La plupart des techniques d'évaluation utilisant des stimulations provocatrices conduisent donc à l'apparition d'une accoutumance à ces stimulations. Ainsi faut-il être bien conscient du fait qu'une épreuve vestibulaire isolée ne donne qu'un cliché "instantané" de la susceptibilité d'un individu vis à vis d'une stimulation donnée.

2.3 Caractéristiques du système nerveux autonome

L'implication du système nerveux autonome dans le syndrome de cinétose est un élément reconnu de longue da ,21, 27) avec une dominante très nette de l'activité parasympathique au cours du développement des symptômes. Ainsi, il est possible de penser que les caractéristiques intrinsèques du système neurovégétatif ou des modifications répondant à des phénomènes intercurrent peuvent affecter la susceptibilité individuelle (9). Les auteurs soviétiques, pour leur part (10), insistent sur la

nécessité d'une très grande stabilité "vestibulo végétative" pour les candidats cosmonautes.

La labilité excessive du système nerveux autonome ou son "excitabilité" semble donc pouvoir constituer un bon candidat comme élément du seuil figurant dans le modèle de Benson. La grande variabilité dans le temps du tonus neurovégétatif chez un individu donné offre ainsi une explication physiologique simple aux variations de la susceptibilité observées pour un même individu (1, 9, 21, 27). Ceci pose bien sûr un problème dans l'évaluation de susceptibilité, principalement avec l'utilisation d'épreuves provocatrices.

2.4 Distribution de la susceptibilité dans la population

Il est bien connu que la distribution de la susceptibilité aux cinétoses dans la population générale ne suit pas une loi normale. Quelle que soit la méthode d'évaluation, on observe généralement des distribution fortement assymétriques, parfois multimodales, mais en aucun cas gaussiennes.

On peut penser que la combinaison des différents éléments constitutifs de la susceptibilité individuelle sont à l'origine de ce type de distribution. Cette approche correspond d'ailleurs assez bien à l'idée que l'on peut en avoir en utilisant les notions de réceptivité et d'adaptabilité préconisées par Reason (26), synthétisées plus récemment par Benson (1).

Quelle qu'en soit l'origine, la distribution erratique de la susceptibilité dans la population constitue une difficulté supplémentaire en matière d'évaluation. Dans ces conditions, il n'est en effet pas possible d'utiliser les techniques relativement simples d'analyse statistique comme l'analyse de variance et les estimateurs aussi commodes que la moyenne et l'écart-type. En règle générale, il faut recourir à des techniques non paramétriques utilisant le classement par rang des variables ou à l'analyse multifactorielle.

3. METHODES D'EVALUATION ET DE PREDICTION

On considère généralement (7, 9, 21, 27) que les méthodes d'évaluation de la susceptibilité individuelle se répartissent en trois groupes principaux: questionnaires anamnestiques, corrélations psychologiques ou physiologiques, épreuves provocatrices. Chaque type de méthode présente des avantages et des inconvénients qu'il convient d'analyser en fonction de l'application que l'on veut en faire.

3.1 Questionnaires anamnestiques

Les questionnaires s'intéressant à l'histoire de mal des transports vécue par un individu afin d'évaluer ou de prédire sa susceptibilité vis à vis d'un environnement ont été largement employés dans de multiples études. Des questionnaires très divers ont été développés à cet effet. Les plus fréquemment utilisés à l'heure actuelle sont sans doute le "Pensacola Motion Sickness Questionnaire" (MSHQ) ou ses dérivés (9) développés aux Etats-Unis et le "Motion sickness Questionnaire" Britannique de Reason (MSQ).

La plupart des auteurs (7,, 9, 22, 27,) s'accordent à considérer que les diverses études expérimentales menées depuis les travaux historiques de Alexander et Coll.(1945) et de Birren (1949) démontrent la validité des questionnaires. D'assez bonnes corrélations ont ainsi pu être obtenues avec des épreuves de laboratoire, mais aussi avec le mal de l'air et le mal de mer, plus récemment, en utilisant un questionnaire spécifique, avec le mal des simulateurs (8). Bien que plus faible mais statistiquement significative, une corrélation a également été rapportée avec le succès en école de pilotage (7).

Plusieurs points importants méritent d'être soulignés à propos des questionnaires.

L'histoire de mal des transports rapportée par un sujet doit toujours être pondérée par rapport à l'expérience des situations provocatrices. Ceci est très bien pris en compte par le MSQ qui effectue une pondération en fonction de l'intensité des symptômes rapportés et de la fréquence d'exposition à la situation qui les a déclenchés. Ce questionnaire comporte également une section qui explore l'histoire de mal des transports avant l'age de 12 ans.

Les questionnaires sont habituellement très faciles à utiliser même avec une population très importante. Leur coût est modéré aussi bien sur le plan financier que sur celui du temps consacré à l'acquisition des données et à leur traitement.

En revanche, ils comportent des inconvénients inhérents à la méthode anamnestique. Ainsi, selon le degré de motivation du candidat et le contexte, on peut facilement aboutir à une sous-estimation (candidat très motivé) ou une surestimation (sujet peu motivé) de la susceptibilité réelle. Guedry (7) note que l'association avec des épreuves de laboratoire conduit habituellement à des résultats plus réalistes que lorsque le questionnaire est utilisé isolément en début de sélection.

Parmi les inconvénients rencontrés, le pouvoir discriminant des questionnaires constitue un problème majeur. Il est en effet reconnu (27) que les questionnaires donnent des résultats satisfaisants pour les susceptibilités extrêmes (très susceptibles ou non-susceptibles) mais sont de peu d'utilité pour classer les sujets ayant une susceptibilité intermédiaire.

3.2 Corrélations psychologiques et physiologiques

D'assez nombreuses variables psychologiques ou physiologiques corrélées avec la susceptibilité au mal des transports ont été décrites (9, 21, 27,). Il s'agit là de variables qui sont censées refléter la "réceptivité" (versant neurosensoriel) ou le "terrain" (versant neurovégétatif).

Par définition, l'évaluation ou la mesure de ces variables ne nécessite pas la mise en oeuvre d'épreuves provocatrices. Ceci les différencie donc des critères physiologiques parfois

utilisés pour juger de la sévérité du malaise provoqué par des stimulations nauséogéniques. Leur intérêt en matière de prédiction pourrait être très grand dans la mesure où, comme les questionnaires, elles présentent une certaine indépendance par rapport aux stimulations provocatrices. Les corrélations relativement faibles incitent cependant à rester très prudent, ce qui est généralement le cas en matière de sélection. Ainsi, pour ce qui concerne la susceptibilité d'un individu donné, elles ne constituent généralement qu'une indication.

3.2.1 Variables psychologiques

Elles sont surtout mentionnées ici pour mémoire car, si elles semblent être parfois significatives dans le cadre d'une population, les corrélations obtenues sont très loin d'être absolues.

Des relations avec la susceptibilité aux cinétoses ont ainsi pu être avancées pour des traits de personnalité, comme l'extraversion-introversion, les tendances névrotiques, la peur et l'anxiété. Des variables comportementales comme la notion de dépendance-indépendance du champ ont également fait l'objet d'un certain nombre d'études, avec des résultats parfois assez contradictoires.

3.2.2 variables physiologiques

Les variables physiologiques ou psycho-physiologiques apparaissent un peu plus intéressantes et ont fait l'objet d'études récentes. Leur intérêt pratique dans la prédiction de la susceptibilité est malgré tout relativement limité et les résultats les plus récents méritent encore d'être validés sur une plus grande échelle. Elles peuvent se diviser en variables neurovégétatives et neurosensorielles.

3.2.2.1 variables neurovégétatives

Une très bonne revue des études anciennes sur les variables physiologiques associées avec la susceptibilité aux cinétoses est présentée dans l'ouvrage de Reason (27). C'est dans la sphère cardio-vasculaire que les résultats les plus nombreux ont été avancés. L'idée qu'une susceptibilité élevée est fréquemment associée à une tendance à la bradycardie et à l'hypotension est relativement répandue (9), bien que largement controversée. De même, Leguay (16) retrouve "avec une grande fréquence, vagotonie et spasmophilie chez les élèves pilotes adressés pour mal de l'air sévère" sans apporter réellement de données statistiques. L'un des principaux problèmes dans ce type d'études est bien sûr l'absence d'un groupe témoin.

Les études plus récentes menées par Kohl (11) sur des critères hormonaux (ACTH, CRF, ...) indiquent que les sujets non-susceptibles présenteraient pour ces variables neuroendocrines des taux de base et des augmentations plus importants sous stimulation que les sujets susceptibles. Toutefois ces valeurs resteraient à l'intérieur des valeurs limites admises en clinique. Sous réserve d'une validation plus approfondie, ce type de critère pourrait se révéler intéressant, bien qu'il ne représente sans doute qu'un aspect du problème de la susceptibilité individuelle.

3.2.2.2 variables neurosensorielles

Les variables neurosensorielles qui ont été proposées comme indices de susceptibilité peuvent être classées en deux catégories, selon qu'elles concernent le fonctionnement du labyrinthe ou qu'elles touchent au codage de l'information sensorielle et à la plasticité du système nerveux.

La première catégorie a suscité de nombreux travaux et certainement autant de controverses. L'exemple le plus classique est le cupulogramme de sensation qui consiste à mesurer la durée de sensations post-rotatoires en réponse à des stimulations impulsionnelles. Alors que certains auteurs comme De Wit et Van Egmond ont trouvé une corrélation significative entre la pente du cupulogramme et la susceptibilité au mal de mer, Doble travaillant sur une population de 1000 élèves pilote n'en trouve aucune pour ce qui concerne le mal de l'air (27). Ces résultats négatifs ont été ultérieurement confirmés par Bles (2) dans une étude sur le mal de mer chronique.

Il existe en fait des relations entre les illusions sensorielles en réponse à des stimulations vestibulaires et les cinétoses qui n'ont jamais été parfaitement clarifiées. En travaillant sur les perceptions illusoires au départ et à l'arrêt d'une centrifugeuse (13), il a pu être mis en évidence que l'amplitude des illusions sensorielles perçues par un groupe de sujets susceptibles était significativement plus importante que pour des non-susceptibles. Le faible nombre de sujets dans chaque groupe ne permet cependant pas de conclure sur ce chapitre.

Pour Reason (27), il apparaît peu évident que des mesures de la sensibilité vestibulaire ou même de l'habituabilité de la fonction puissent fournir des indices fiables de la susceptibilité aux cinétoses.

Des résultats expérimentaux récents obtenus par Diamond et Markham (4, 19) relancent cependant cette problématique en montrant une relation très significative entre la susceptibilité au mal de l'espace et l'asymétrie de la fonction otolithique. Il faut remarquer que les soviétiques utilisent depuis de nombreuses années comme prédicteur de la susceptibilité l'asymétrie de la réponse otolithique lors d'épreuves sur la balançoire parallèle (5, 20).

En complément de ces indices liés directement au fonctionnement vestibulaire, Reason (26, 27) a tenté de développer des mesures indirectes qui, selon lui, réfléteraient la réceptivité des sujets vis à vis des conflits sensoriels. Il a ainsi pu mettre en évidence des corrélations significatives entre la susceptibilité aux cinétoses déterminée par un questionnaire, la persistance de "spiral after effect" (SAE) et la mesure psychophysique de l'intensité d'un son de fréquence 1000 HZ. Il propose ainsi, en associant les mesures de réceptivité, d'adaptabilité à des stimulations provocatrices et de rétention de l'adaptation,

une démarche globale utilisable pour l'évaluation et la prédiction, sinon de la susceptibilité d'un individu vis à vis des cinétoses en général, du moins de la catégorie de risque dans laquelle il se situe.

3.3 Epreuves provocatrices

A ce jour, en dépit de l'intérêt présenté par les méthodes indirectes qui viennent d'être évoquées, les épreuves provocatrices, dont le but est de faire apparaître les symptômes caractéristiques des cinétoses, constituent encore l'essentiel d'une démarche d'évaluation de la susceptibilité.

Les revues de la littérature (21, 27) montrent que de très nombreux moyens ont été mis en oeuvre pour provoquer l'apparition du syndrome de cinétose, aussi bien en laboratoire que sur le terrain. En fait quelques techniques principales, largement utilisées pour l'évaluation de la susceptibilité, peuvent être identifiées et seront décrites ici. Quelles que soient les stimulations employées, plusieurs problèmes se posent à l'expérimentateur.

C'est tout d'abord le problème de l'intensité et de la durée des stimulations, le protocole choisi devant être discriminant et éviter l'effet plancher et l'effet plafond. Ce sont aussi les critères utilisés pour arrêter l'épreuve, pour juger de la sévérité du malaise provoqué et pour quantifier la susceptibilité vis à vis de la stimulation employée.

3.3.1 Techniques et protocoles de stimulation

Lentz (17, 18) a effectué une revue comparative détaillée des épreuves de laboratoire les plus utilisées pour évaluer la susceptibilité. Elles ont pour principe commun de générer des situations où se produisent des conflits sensoriels qui aboutissent beaucoup plus rapidement au syndrome de cinétose que les situations réelles de terrain. En utilisant une dichotomie simple, on peut également classer ces épreuves selon le type de stimulation de mouvement délivré au sujet: principalement accélérations linéaires, accélérations angulaires et interactions visuo-vestibulaire. Les méthodes utilisant les situations de terrain où les stimulations sont habituellement beaucoup plus difficilement contrôlables ne seront pas évoquées ici.

3.3.1.1 Accélérations linéaires

Ces épreuves vont essentiellement stimuler les récepteurs otolithiques.

L'accélérateur vertical a été très largement utilisé en particulier pour la prédiction du mal de mer. Compte tenu du domaine de fréquence relativement bas des stimulations efficaces (0,1 à 0,4 Hz) et du niveau d'accélération requis, ce type de stimulation exige une grande amplitude de mouvement donc un appareillage lourd et encombrant dont peu de laboratoires peuvent disposer.

Une alternative est constituée par l'utilisation d'une balançoire parallèle qui permet de délivrer des stimulations otolithiques adéquates. Ce type d'épreuve figure dans les tests de sélection des pilotes et des cosmonautes soviétiques (10). Plus récemment, Money et Oman (22) ont tenté (avec peu de succès) d'utiliser un accélérateur horizontal (sled).

Finalement la meilleure épreuve de stimulation otolithique consiste sans doute en une rotation à vitesse constante autour d'un axe incliné par rapport à la verticale terrestre. De nombreuses équipes se sont intéressées aux effets de cette stimulation qui sollicite la fonction otolithique par le biais de la réorientation constante du vecteur gravité relativement aux macules utriculaires et sacculaires. Il s'agit là d'une épreuve très provocatrice dont la mise en oeuvre demeure relativement simple.

Plusieurs versions codifiées de ce test existent comme le "Tilted Axis Rotation Test" (TART) utilisé à Pensacola ou le "Off Vertical Rotation test" initialement conçu par Graybiel et Miller et encore utilisé sous une forme modifiée (OVR) par la NASA (28). Dans les deux cas l'axe de rotation est incliné de 25 à 30° au maximum avec des vitesses de rotation qui ne dépassent pas 100 °/s pour le TART et peuvent atteindre 240 °/s pour l'OVR. Notons enfin que les rotations autour d'axes purement horizontaux (PAM) ont également été utilisées avec succès dans des études d'évaluation de la susceptibilité (12, 22).

3.3.1.2 Accélérations angulaires

L'utilisation des accélérations angulaires constitue une méthode de choix pour provoquer rapidement le syndrome de cinétose. Elle ne requiert généralement qu'un simple fauteuil tournant ce qui en fait une technique très économique sur le plan des installations. Le fauteuil tournant a maintenant presque totalement remplacé les autres générateurs d'accélérations angulaires, comme les balançoires largement utilisées dans les travaux historiques, en particulier au Canada.

Plusieurs techniques différentes peuvent être décrites en fonction des protocoles utilisés.

La répétition d'arrêts rapides de la rotation après une phase à vitesse constante est connue de longue date pour ces effets provocateurs. Cette technique est identifiée comme le "sudden-stop test". Elle consiste mettre le sujet en rotation à 300 °/s pendant 30 secondes puis à l'arrêter en 1,5 seconde. Après 30 secondes d'arrêt, la procédure est répétée 20 fois les yeux fermés, puis 20 fois dans un sens les yeux ouvert et éventuellement 20 fois dans l'autre sens jusqu'à ce que le sujet atteigne un critère de malaise défini à l'avance.

Les épreuves utilisant l'effet des accélérations de Coriolis sur le système vestibulaire sont sans doute les plus répandues en matière d'évaluation de la susceptibilité. Fondamentalement, ces épreuves reposent sur l'exécution de mouvements actifs ou passifs de la tête pendant une rotation à vitesse constante. De nombreux protocoles ont ainsi été utilisés.

Le "Brief vestibular disorientation test" (BVDT) est une épreuve extrêmement répandue. Après 30 secondes de rotation à une vitesse constante de 90 °/s le sujet exécute toutes les 30 secondes des mouvements actifs de la tête d'amplitude 45°, en alternant inclinaison à droite et inclinaison à gauche (répété deux fois), puis flexion vers l'avant. Après chaque mouvement le sujet revient en position neutre tête droite. Le temps maximum de rotation est de 5 minutes et 30 secondes et le sujet effectue à l'issue du test une auto-évaluation confirmée par un score établi par un observateur.

Les études menées dans la chambre de rotation lente de Pensacola avec le "dial test" ont été à la source d'un des protocoles les plus utilisés à l'heure actuelle (18), le "Coriolis Sickness Susceptibility Index test" (CSSI). Ce test a été développé en vue d'aboutir à une quantification de la susceptibilité au moyen d'un score numérique unique. L'épreuve consiste en l'exécution de mouvement actifs de la tête à 90 ° alternativement dans les quadrants avant, droite, arrière, gauche, avec retour en position neutre entre deux mouvements. La vitesse de rotation du fauteuil tournant est augmentée par paliers pour atteindre au maximum 180 °/s. L'épreuve est arrêtée lorsque le sujet atteint un niveau de malaise prédéterminé. Le score de susceptibilité est alors calculé en prenant en compte le nombre de mouvements effectués pendant chaque palier, pondéré par un facteur lié à la vitesse de rotation.

De nombreuses variantes de cette épreuve ont été adaptées, en utilisant des protocoles en rampe de vitesse ou autres et en jouant sur le nombre, l'amplitude et la nature des mouvements de tête, selon le but recherché.

Les Soviétiques utilisent pour la sélection des cosmonautes plusieurs protocoles faisant appel à l'effet cumulé des accélérations de Coriolis.

Le plus répandu consiste à faire effectuer des mouvements du buste de 90 °dans le plan sagittal avec une périodicité de 5 secondes et une vitesse de rotation du fauteuil établie d'emblée à 180 °/s. La durée minimum de l'épreuve est de 8 minutes et, si la tolérance du sujet le permet, elle se prolonge jusqu'à 12 minutes ou plus pendant l'entraînement. Il s'agit donc d'une épreuve extrêmement brutale.

Les auteurs soviétiques (10) attachent une grande importance aux réactions neurovégétatives spécifiques du syndrome, mais aussi aux réactions cardio-vasculaires. Un autre protocole utilisé en sélection utilise des mouvements alternatifs de la tête à droite et à gauche pendant une rotation à 180 °/s avec arrêt toutes les minutes. Les paramètres cardio-vasculaires, pression artérielle et rythme cardiaque, sont alors mesurés puis la rotation reprend en sens inverse. Le candidat doit subir cette procédure 10 fois successivement avec un très bref arrêt entre deux période de rotation.

3.3.1.3 Interactions visuo-vestibulaires

Le "Visual-Vestibular Interaction Test" (VVIT) constitue un exemple d'épreuve utilisant les interactions visuo-vestibulaires. Il consiste à soumettre un sujet à une stimulation oscillatoire à 0,02 Hz avec une vitesse crête de 155 °/s en lui demandant simultanément d'effectuer une tâche visuelle d'extraction de données dans une matrice de nombres. Il a été ainsi montré que la complexité de la tâche visuelle intervenait significativement dans la sévérité du malaise observé.

Money et Coll. (22) ont utilisé le port de prismes inversants pour tester la susceptibilité des astronautes du premier vol Spacelab. Toutefois les mouvements de la tête et les déplacements effectués étant libres, l'évaluation a une valeur plus qualitative que quantitative.

D'une manière générale, on peut également assimiler les épreuves utilisant la vection circulaire ou linéaire aux épreuves d'interaction visuo-vestibulaire.

3.3.2 critères de jugement

Les critères utilisés pour juger de la susceptibilité des sujets présentent une grande importance. Il convient en effet d'éviter les problèmes d'effet plancher et d'effet plafond si l'on veut réaliser une classification suffisamment discriminante de la population étudiée. Par exemple une épreuve peu provocatrice associée à des critères de malaise sévère donne lieu à un effet plancher dans la mesure où beaucoup de sujets risquent de ne jamais atteindre le critère. L'inverse, (épreuve très provocatrice et critère faible) donne lieu à un effet plafond. On doit distinguer ici les épreuves effectuées en iso-stimulus comme le BVDT et les épreuves en iso-malaise comme le CSSI. Dans les deux cas le problème du critère de jugement, critère d'arrêt ou critère d'évaluation va se poser. La validité de l'évaluation de la susceptibilité va reposer pour une part importante sur la fiabilité de ces critères.

3.3.2.1 critère d'arrêt

De nombreux critères d'arrêt ont pu être utilisés. L'apparition du vomissement, signe objectif de la sévérité du malaise, est considéré comme un bon critère mais présente des inconvénients évidents, particulièrement dans les études nécessitant la coopération des sujets. La plupart des auteurs utilisent plutôt l'apparition d'une nausée franche et non équivoque.

Pour pallier les difficultés d'estimation, des échelles d'auto-estimation ou d'observation de l'intensité des symptômes ont été développées. Un repère sur cette échelle, généralement numérique, permet alors de fixer commodément le critère d'arrêt de l'épreuve.

3.3.2.2 Echelles d'évaluation de sévérité

La notion de transformation d'un jugement subjectif en variable numérique est à l'origine des diverses techniques d'estimation psychophysique. Il est en effet possible d'estimer de façon introspective l'amplitude des effets

conscients résultant des évènements auxquels nous sommes soumis par l'environnement. Stevens a montré que cette estimation pouvait être numérique et se représenter sous forme d'échelles psychophysiques.

Les échelles psychophysiques ont pour principal avantage d'éviter que l'observateur induise un biais par son influence sur les réponses du sujet. Il ne s'agit toutefois que d'une auto-estimation représentant l'expérience qui résulte à la fois de la stimulation présente et de la mémoire d'expériences analogues plutôt que le niveau d'intensité du stimulus physique auquel le sujet est soumis.

Par principe, l'expérience vécue par le sujet ne peut être comparée à celle d'un autre individu du fait des caractéristiques émotionnelles mais aussi d'une mémoire des expériences passées propres à chacun. Ceci explique la réalisation fréquente d'études en iso-malaise à l'aide d'échelles dont les niveaux sont fixés à priori. On utilise donc rarement de véritables échelles de "bien-être global" puisqu'elles ne peuvent bénéficier de niveaux fixés à priori. Il semble préférable d'employer des échelles chiffrant en priorité l'intensité des symptômes digestifs, et notamment de la nausée qui est le signe cardinal de l'apparition et de la sévérité des cinétoses. Ainsi les premières études réalisées durant la seconde guerre mondiale par Wendt utilisaient déjà une échelle psychophysique basée sur les symptômes digestifs. Les cinétoses étaient classées dans un ordre croissant de sévérité selon trois réponses : inconfort, nausées sans vomissements, vomissements.

Les données de la littérature sur ce sujet montrent que de nombreuses échelles peuvent être construites sur ce principe. Il y a quelques années, Oman (24) a bien souligné l'intérêt de ces échelles dans l'appréciation de la dynamique des symptômes de cinétose. Elles ont en particulier l'avantage de donner une image dynamique de l'évolution de la sévérité du malaise, ce qui peut être utilisé dans des processus de modélisation.

Lorsque l'on utilise de telles échelles comme critère d'arrêt, on convient généralement d'éviter les niveaux les plus élevés car l'intensité des symptômes croît alors en cascade et les vomissements peuvent survenir. L'arrêt est décidé par le sujet, préalablement informé du niveau à atteindre, et non par l'observateur.

A l'opposé, un autre procédé d'évaluation consiste à utiliser les observations effectuées par un tiers sur la sévérité des symptômes. L'exemple le plus élaboré de cette technique est sans doute l'échelle de Graybiel et Millar ou échelle diagnostique de Pensacola, qui associe des informations d'auto-estimation à l'évaluation par l'expérimentateur des manifestations objectives qu'il observe. Ce score fait appel au sens clinique de l'observateur pour la mesure de la pâleur et de la sudation. L'identification et la mesure des autres symptômes (nausées, salivation, endormissement, céphalées, déséquilibre) font appel à l'auto-estimation.

Le score de Graybiel et Millar est constitué par la somme des points attribués par l'échelle aux différents symptômes.

Il faut noter que ce mode de quantification fait appel à des appréciations subjectives de la part du patient et de l'observateur. De plus, les symptômes pris en compte peuvent refléter une réponse végétative caractéristique de certains sujets et ne pas être liés à la sévérité des cinétoses. On risque par exemple de surestimer le malaise présenté (si l'on se réfère à une échelle psychophysique) par les sujets soumis à un entraînement aérobie régulier car ils présentent une sudation plus facilement stimulable qu'une population de référence.

Bien que Reason et GraybieL aient montré une corrélation monotone entre leur échelle d'auto estimation en 12 points et le score de Pensacola, le but essentiel de ce dernier demeure la détermination d'un critère d'arrêt d'expérimentation. En effet, comme le souligne Oman (24), la tendance à ne présenter qu'un chiffre global représentant l'intensité du malaise enlève à cette technique une partie de son intérêt pour suivre l'évolution des symptômes.

Les échelles psychophysiques ont l'avantage d'être facilement et rapidement établies. Elles peuvent être utilisées pour suivre l'évolution du syndrome de cinétose ou pour définir un critère d'arrêt. Cependant elles sont fondées sur des appréciations subjectives et sur la transformation de variables qualitatives en une ou plusieurs variables quantitatives. Elles sont donc relativement imprécises et souvent ne permettent pas de bien différencier des niveaux très proches d'intensité du syndrome de cinétose, particulièrement pour les niveaux élevés qui peuvent s'enchaîner en cascade.

3.3.2.3 Mesures physiologiques objectives

L'évaluation de la sévérité du malaise par une échelle laisse donc subsister une relative imprécision liée aux aspects subjectifs de la technique employée. De nombreux efforts ont été consacrés à la mise au point de mesures physiologiques représentatives de l'intensité des symptômes provoqués par les stimulations de laboratoire. La détermination d'éléments objectifs suffisamment fiables et précis pour pouvoir être utilisés en tant que critère d'arrêt ou comme indice de sévérité serait bien évidemment d'un grand intérêt aussi bien pour les protocoles en iso-malaise qu'en iso-stimulus.

La mesure de variables cardio-vasculaires, comme la fréqence cardiaque et de la pression artérielle, a de tout temps suscité un vif intérêt. En fait, il semble difficile d'utiliser ces variables en raison des grandes variations interindividuelles observées dans les réponses aux stimulations nauséogéniques. Signalons toutefois que les soviétiques préconisaient encore il y a quelques années une ligne "dure" assimilant à une mauvaise tolérance toute variation de la fréquence cardiaque, dans un sens ou dans l'autre, de plus de 10 battements par minute.

La mesure objective de signes neurovégétatifs comme le degré de sudation ou la pâleur présente des difficultés importantes. Dans le même domaine, il faut cependant signaler les résultats encourageants obtenus par Parker (9)

avec la mesure de la conduction cutanée palmaire et ceux plus récents de Golding (6) qui a utilisé avec succès la même méthode au niveau du front. Il faut également signaler les résultats obtenus avec les enregistrement EEG pendant la stimulation (3), qui semblent montrer l'apparition de complexes pointe-onde pendant le développement du malaise.

Le retentissement des cinétoses sur la sphère digestive constitue également un point d'intérêt pour l'obtention de mesures objectives. Deux types de techniques ont été principalement utilisés dans les études récentes.

C'est d'une part la gastro-entérographie, c'est à dire la mesure des variations du champ électrique de l'estomac et des viscères sous l'influence de stimulations provocatrices. En dépit des difficultés d'enregistrement et de traitement du signal qui peuvent être rencontrées, ce type de mesure semble pouvoir aboutir à des résultats intéressants (30). Cette technique montre en particulier l'existence d'une atonie antrale qui semble bien corrélée avec la sensation de nausée.

la deuxième technique fait appel à l'imagerie échographique. A l'aide de cette technique il est possible de mettre en évidence le retard de vidange gastrique résultant du syndrome de cinétose (31). Une étude récente (32) a montré que le retard de vidange gastrique d'un repas semi-liquide ingéré avant une épreuve provocatrice était corrélé linéairement (R=0,85) à l'intensité du malaise provoqué par la stimulation (évaluée au moyen d'échelles). Il existe cependant une assez forte variabilité interindividuelle et l'étude ne s'est pas intéressée aux éventuelles variations intra-individuelles (test-retest). La mesure nécessite une bonne pratique de l'échographie digestive et la répétition à intervalles réguliers des prises d'images, ce qui suppose une immobilisation relativement prolongée du sujet. Si sa fiabilité était démontrée, ce type de mesure pourrait être particulièrement intéressant pour les épreuves en iso-stimulus où la détermination de la sévérité du malaise est toujours délicate.

Dans la même étude, Valmalle et Coll. se sont également intéressés à l'évolution de variables hormonales en fonction de la sévérité du malaise. Il s'agit là plutôt d'une étude pilote, portant sur 6 sujets et les résultats obtenus doivent être considérés avec prudence. Une relation linéaire (R=0,8) été observée entre le taux d'AVP plasmatique et la sévérité du malaise à l'issue des épreuves provocatrices. De même, une assez bonne corrélation est obtenue entre la sévérité du malaise et le taux d'épinéphrine, mais le taux de norépinéphrine ne semble pas avoir été modifié d'une manière significative.

3.3.2.4 scores de susceptibilité

Il convient généralement de ne pas confondre les échelles aboutissant à l'estimation du malaise avec les indices de tolérance qui reflètent la "quantité de stress" subie par un sujet pour arriver à un niveau de malaise déterminé. Toutefois les scores de malaise sont parfois utilisés comme indices de tolérance, en particulier dans les études en iso-stimulus (BVDT).

Dans les études en iso-malaise, les méthodes les plus simples consistant à utiliser directement la durée de la stimulation ou le nombre de stimulations provocatrices subies par le sujet. La notion de durée peut également entrer avec le score de malaise dans la composition d'un indice unique utilisé par exemple pour ranger des données en vue d'une exploitation statistique (12).

L'indice le plus utilisé est très certainement le C.S.S.I. (Coriolis Sickness Susceptibility Index) mis au point par Miller et Graybiel. Cet indice a été établi pour les études utilisant les accélérations de Coriolis selon le protocole évoqué plus haut (CSSI test). Pour une vitesse donnée, le nombre de mouvements de tête nécessaires à l'atteinte du niveau de sévérité fixé est un témoin de la susceptibilité individuelle aux cinétoses. Plus le nombre de mouvements de tête réalisé est important, moins le sujet est sensible et inversement. Lorsque la vitesse de rotation augmente, le stimulus devient plus important et le nombre de mouvements de tête nécessaire décroît . Miller et Graybiel ont calculé, lors d'études en isomalaise et à des vitesses de rotation différentes, l'effet "stressant" d'un unique mouvement de tête. Cette relation est de type logarithmique.

La susceptibilité aux cinétoses induites par les accélérations de Coriolis (score CSSI) peut donc être calculée simplement :

$$C.S.S.I. = \Sigma E_i N_i$$

E : effet d'un mouvement de tête

N : nombre de mouvements à une vitesse de rotation donnée.

La fiabilité de cet indice dépend donc essentiellement de la fiabilité de la détermination du critère d'arrêt qui est en principe le "moderate malaise II" de l'échelle d'observation de Graybiel et Miller.

4. SYNTHESE

Les méthodes d'évaluation de la susceptibilité aux cinétoses sont donc multiples. En règle générale, ce type de situation signifie essentiellement qu'aucune d'entre elles ne constitue une panacée. Ceci est également vrai pour les critères ou mesures utilisés par ces différentes méthodes.

En s'appuyant sur des considérations statistiques, Kennedy (9) souligne très clairement que la fiabilité de la prédiction est directement conditionnée par la fiabilité de la technique elle-même et celle des mesures qu'elle utilise. Ceci constitue bien sûr un point essentiel à prendre en considération lorsque l'on tente d'évaluer la susceptibilité d'un individu donné vis à vis d'une situation opérationnelle. En appliquant ce type de raisonnement statistique aux différentes techniques d'évaluation, il aboutit à la

conclusion que les variables physiologiques et les traits de personnalité ont une valeur prédictive faible alors que des résultats plus satisfaisants sont obtenus au moyen des questionnaires et des épreuves provocatrices. Les meilleures prédictions sont cependant obtenues en utilisant des mesures en situation opérationnelle.

Sans entrer dans des considérations statistiques avancées et avec le souci de rester concret, le principal enseignement que l'on peut tirer des études sur l'évaluation de la susceptibilité est qu'il faut adapter soigneusement les techniques et les mesures au but recherché. Le problème est en fait d'utiliser au mieux des techniques que nous savons être imparfaites. Deux points essentiels doivent être soulignés: la nécessité d'une grande rigueur méthodologique d'une part et la prudence dans l'interprétation des résultats d'autre part.

Les méthodes d'évaluation utilisant les questionnaires ont un intérêt certain lorsque l'on cherche à évaluer la susceptibilité d'un grand nombre d'individus. Elles permettent de trier relativement aisément les individus extrêmes, très susceptibles ou très peu susceptibles. Malheureusement, pour les susceptibilités intermédiaires, le pouvoir prédictif devient beaucoup plus incertain.

Le problème qui peut se poser concrètement est donc celui de la calibration des résultats en fonction du but à atteindre. Utilisés dans un processus de sélection, ils aident principalement à éliminer les sujets très susceptibles. La question qui se pose alors est de savoir où placer la limite entre les individus très susceptibles et les intermédiaires. L'expérience montre qu'il s'agit là d'une tâche délicate.

L'illustration de ce propos peut faire appel aux données acquises lors d'un processus de sélection de candidats cosmonautes pour les vols Franco-Soviétiques (14). La population initiale de candidats retenus après un premier tri sur dossiers se composait de pilotes militaires et civils, d'ingénieurs et de scientifiques. L'histogramme des scores de susceptibilité obtenus à l'aide d'une variante du questionnaire MSQ donne un profil relativement habituel pour ce type de population (figure 1). A ce stade, les quelques sujets présentant des scores très élevés, supérieurs à 50, ont été éliminés.

Parmi les autres, après passage dans différents filtres, 80 ont subi une épreuve provocatrice sur fauteuil tournant, utilisant les accélérations de Coriolis selon un protocole à deux niveaux de vitesse de rotation (90 et 180 °/s) avec un mouvement actif de flexion du buste toutes les 5 secondes. La durée maximale du test était de 8 minutes.

A l'issue de cette épreuve, les candidats ont été classés en trois catégories (réactivité faible, modérée, élevée) selon l'intensité des symptômes observés en suivant l'échelle de Graybiel. Lorsque l'on fait l'histogramme des scores MSQ obtenus selon ces trois catégories on constate différents points. Dans le groupe à faible réactivité (figure 2a), plus de 50 % des candidats ont obtenus un score MSQ compris entre 0 et 2, aucun n'a de score supérieur à 20. Dans le groupe intermédiaire, le pourcentage des scores entre 0 et 2 diminue et est proche de 30 % (figure 2b). Les individus classés dans ces deux groupes ont tous terminé l'épreuve sans atteindre le critère d'arrêt qui était la nausée sévère ou le vomissement. Dans le groupe à réactivité forte, on retrouve encore presque 15 % de scores très bas (figure 2c), ce qui n'est pas étonnant étant donné la motivation de candidats, mais on constate aussi que les quelques candidats admis aux épreuves avec des scores élevés se trouvent dans ce groupe. Ces quelques schémas montrent assez bien les limites des questionnaires dans les processus de sélection.

En revanche, dans l'évaluation de la susceptibilité vis à vis de certaines situations de terrain, comme le simulateur de vol, les questionnaires constituent une méthode très intéressante, comme en témoignent les résultats obtenus par Kennedy (8).

Les épreuves provocatrices en laboratoire ne sont pas dépourvues d'inconvénients dans les sélections. Il faut distinguer ici deux cas, selon que l'objectif recherché est de prédire la tolérance dans un environnement proche de la situation de laboratoire, ou qu'une prédiction plus générale est recherchée.

Dans le premier cas, le problème est relativement simple et la difficulté majeure consiste à ajuster le niveau de difficulté des épreuves et à essayer de tenir compte des capacités d'adaptation des candidats. C'est le cas dans les protocoles Franco-Soviétiques où l'on s'efforce de prédire la capacité des candidats à endurer l'entraînement vestibulaire extrêmement poussé préconisé par les Soviétiques avant les vols spatiaux. Si le protocole de sélection est trop faible, en tentant de solliciter les capacités d'adaptation plutôt que la résistance, on peut parfois observer des échecs. La solution qui a été retenue lors de la dernière sélection effectuée (MIR 92) a été de tenter d'établir la tolérance de base d'un faible nombre de candidats pré-sélectionnés, puis de tester les capacités d'habituation de ceux qui semblaient les plus intéressants sur le plan de la candidature.

L'épreuve, toujours fondée sur l'effet de la cumulation des accélérations de Coriolis, a été conçue en conséquence . La session complète de stimulation vestibulaire comporte 8 paliers de vitesse de rotation (entre 30 et 240 °/s). Pour chaque palier les sens horaire et antihoraire sont successivement utilisés. La durée totale de test est de 34 minutes. Les stimulations provocatrices sont produites par des mouvements actifs d'inclinaison de la tête à droite et à gauche, suivis d'une flexion du buste en avant. La périodicité des mouvements est de 5 secondes et le sujet reste dans la position où il se trouve entre deux mouvements successifs (pas de retour en position neutre). Cette épreuve permet une bonne discrimination de la tolérance des sujets, classés auparavant sur la base d'une épreuve soviétique classique en 8 minutes, en traçant sur un diagramme à deux dimensions la position des différents sujets en fonction du score de malaise observé à la fin de l'épreuve contre l'indice de tolérance calculé (figure 3).

Dans toutes les épreuves de sélection, l'évaluation de la sévérité du malaise provoqué par l'épreuve pose un problème. L'auto-estimation de la nausée, qui est un élément fondamental des techniques d'observation, est relativement peu fiable avec des sujets motivés. C'est pourquoi il est nécessaire de faire arrêter l'épreuve par une action volontaire du sujet lorsqu'il atteint le critère de nausée sévère précédant le vomissement, ce dernier terminant en fait très souvent l'épreuve. Ce type d'épreuve bénéficierait bien évidemment de l'utilisation de mesures physiologiques objectives.

En revanche dans toutes les études où la coopération du sujet est acquise "a priori", les échelles d'auto-estimation donnent généralement des résultats satisfaisants et leur fiabilité semble seulement limitée par les variations interindividuelles et la capacité de discriminer entre des seuils de malaise proches.

Le problème de l'évaluation générale de la susceptibilité à partir d'épreuves provocatrices est beaucoup plus complexe. C'est le cas pour la prédiction de la susceptibilité au mal de l'espace où les stimulation de laboratoire classiques rendent très mal compte de la situation particulière rencontrée en microgravité. Reschke (28) a ainsi montré que les corrélations entre la susceptibilité évaluée au moyen de différentes épreuves de laboratoire et la survenue du syndrome au cours du vol parabolique en microgravité étaient très faibles. Plus récemment (29), le même auteur conclut à la nécessité d'utiliser des batteries de tests qui permettent d'obtenir des scores composites plus représentatifs de la susceptibilité au mal de l'espace. Ceci implique la mise en oeuvre de techniques statistiques élaborées et l'utilisation de modèles prédictifs.

Pour terminer cette synthèse sur l'évaluation et la prédiction de la susceptibilité, il est intéressant de revenir sur les résultats statistiques obtenus par Kennedy qui montre que les mesures opérationnelles donnent de très bons résultats. Il faut remarquer ici que, pour ce qui concerne le mal de l'air, l'Armée de l'Air Française utilise en sélection initiale une technique qui, sans être totalement rationalisée pour les cinétoses, se rapproche assez des mesures opérationnelles. L'Armée de l'Air dispose, il est vrai, d'un potentiel de recrutement assez important, qui lui donne une certaine liberté dans ce domaine. La technique consiste simplement à faire effectuer aux élèves pilotes des manoeuvres acrobatiques dans les quinze premières heures de vol et à ne retenir que ceux qui présentent un niveau de performance satisfaisant. Cette sélection globale, où l'effet des cinétoses est implicitement inclus, semble donner des résultats assez satisfaisants, en ne retenant que des sujets non-susceptibles ou capables de s'adapter très rapidement à l'environnement du vol.

5. CONCLUSIONS

Il existe un très large éventail de techniques permettant l'évaluation et éventuellement la prédiction de la susceptibilité aux cinétoses. Ces techniques sont pour la plupart relativement complémentaires, mais aucune ne peut prétendre convenir à l'ensemble des situations rencontrées en milieu opérationnel.

La plupart des auteurs s'accordent à considérer que les questionnaires anamnestiques et les épreuves provocatrices comportent, à l'heure actuelle, les plus grandes potentialités pour l'évaluation. Parmi les épreuves provocatrices, celles effectuées sur fauteuil tournant et utilisant l'effet des accélérations de Coriolis sont les plus communément utilisées.

Deux tendances semblent coexister dans les études en cours. Il s'agit, d'une part, du recours à des batteries de test très complètes et à des traitements statistiques complexes dont l'objectif est d'aboutir à l'élaboration de véritables modèles de prédiction. D'autre part, il existe une tendance qui tente d'améliorer la fiabilité des critères d'évaluation, en recherchant tout particulièrement la mise au point d'indices reposant sur la mesure de variables physiologiques quantitatives.

Il faut remarquer que ces deux tendances sont complémentaires et devraient permettre d'améliorer considérablement la prédiction des cinétoses terrestres, mais aussi celle du mal de l'espace qui, pour l'instant, pose encore quelques problèmes.

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figure 1 : Distribution de la susceptibilité évaluée au moyen du MSQ (Motion Sickness Questionnary) dans une population de 193 candidats cosmonautes.

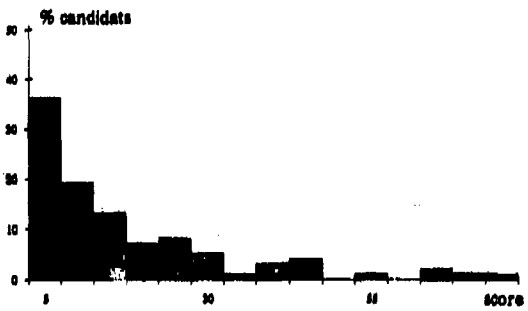

figure2 : Distribution des scores MSQ obtenus par les candidats selon leur classement en trois groupes de susceptibilité (faible, moyenne,forte) à l'issue d'une épreuve provocatrice.

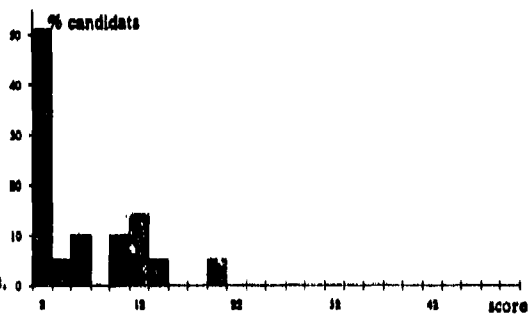

figure 2a : Groupe I, susceptiblité faible.

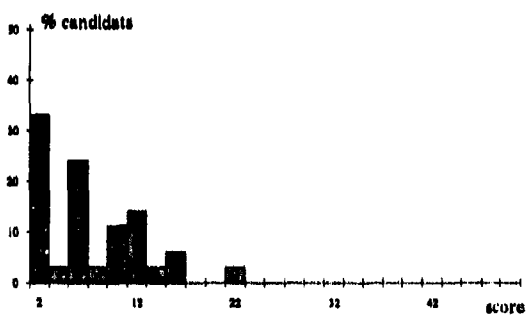

figure 2b : Groupe II, susceptiblité moyenne.

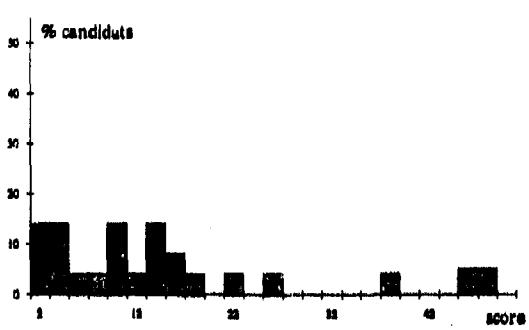

figure 2c : Groupe III, susceptiblité forte.

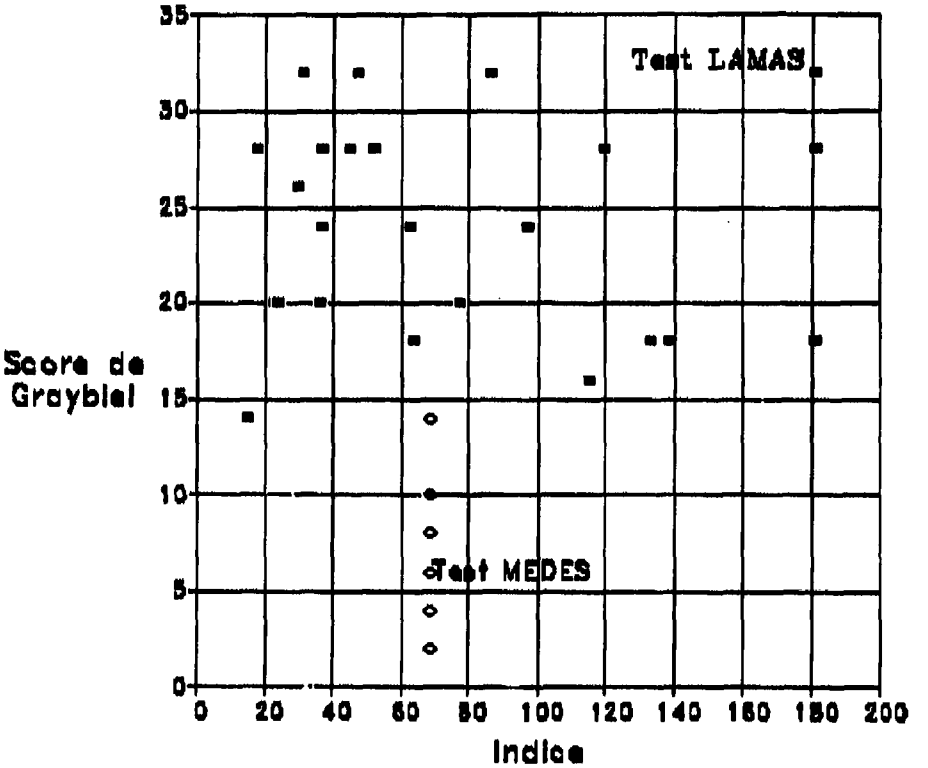

Figure 3: Le diagramme présente le score de malaise à la fin d'une épreuve contre l'indice CSSI calculé en fonction du nombre de mouvements de tête effectués. La comparaison entre les résultats obtenus lors d'une épreuve de 8 minutes (MEDES) et l'épreuve de tolérance de 34 minutes (LAMAS) montre bien la discrimination obtenue entre les différentes susceptibilités individuelles par ce dernier test.

PREVENTION AND TREATMENT OF MOTION SICKNESS: NON-PHARMACOLOGICAL THERAPY

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Introduction.

The concept that conflicting sensory patterns of motion constitute the stimulus that in susceptible subjects gives rise to motion sickness allows rational measures to be taken aimed at minimising sensory conflict and thereby reducing the incidence of symptoms. Such measures add to the effectiveness of prophylactic drugs and may alone be sufficient to prevent motion sickness.

It is well known among sailors and astronauts that continued or repeated exposure to an initially nauseogenic motion stimulus leads to a state of increased resistance to its effect. This provides a spontaneous cure for some individuals and forms the basis of therapeutic programmes to assist others.

This paper deals with the strategies that individuals can adopt to minimise their exposure to nauseogenic stimuli, as well as with adaptation and its application to the treatment of chronic airsickness in aircrew. Also considered are some of the non-pharmacological measures that have been used in the treatment of motion sickness.

The reduction of sensory conflict.

To those of a more adventurous nature a motion stimulus that creates sensory conflict is a pleasurable and exciting experience - initially at least. Much of the popularity of fairground amusements derives from the patterns of motion sensation that cannot be achieved by pedestrian man, and the various rides provide examples of many types of sensory conflict. The fairground operator's treatment for motion sickness is to stop the ride before, at least for the majority of the population, the pleasure gives way to nausea. That way he maximises his profits, while the most susceptible individuals quickly learn to avoid those rides in which enjoyment is too rapidly engulfed by misery. With fairground amusements avoidance is the most reliable prophylaxis. In everyday life, however, for the most susceptible individuals the total avoidance of conflicting sensory stimuli is neither necessary nor possible without imposing unacceptable constraints on travel or leisure activities.

As has been described in a previous paper, sensory conflict can occur between visual and vestibular sensors of motion, between the canal and otolith components of the vestibular system, and as a consequence of low frequency oscillatory motion.

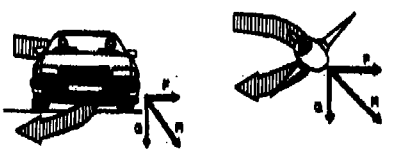
Visual-vestibular conflict. The normal expectation for pedestrian man is that in the presence of head rotations the external visual world will sweep away in the opposite direction; that is it will appear to remain space stable. It is an essential function of the vestibular system to generate compensatory eye movement so as to stabilise the retinal image of a space-stable visual target in the presence of motion of the head. In certain circumstances this reflex is inappropriate and there is consequent sensory conflict.

The navigator who is map-reading while the aircraft is manoeuvring experiences angular motion sensed by the semi-circular canals, but because his field of view is confined to objects within the cockpit, he lacks any visual evidence of rotation, and any vestibular induced eye movement has to be suppressed if visual degradation for the task in hand is to be avoided. Similarly, the susceptible individual aboard ship will experience less conflict if on deck with a view of the horizon, or even the restless sea, rather than the visual world below decks that moves with the ship.

Car sickness is a common problem in children which they are said to 'grow out of'. In fact, physical growth may well be an important factor. A small child confined to the rear seat of a car sees little of the outside world, in particular, the road ahead, that would provide him with the visual accord when the vehicle corners or changes speed. Reading in a car is well known to precipitate motion sickness in susceptible individuals. In this situation the detailed scanning eye movements coupled with the need to suppress vestibular induced eye movements may be an intensifying factor. In a laboratory study a visual task that required a subject to search for numbers within a grid while

undergoing low frequency (0.02 Hz) angular oscillation in yaw was shown to be nauseogenic (Guedry et al, 1982), whereas the task of reading digits sequentially presented at the same location under similar motion conditions did not provoke symptoms (Benson & Guedry, 1971).

Canal-otolith conflict. The state of sensory accord between semicircular canals and otoliths occurs when motion of the head out of the horizontal plane (ie in pitch or roll with the head upright) is accompanied by an equivalent angular change in the direction of the gravitational acceleration as sensed by the otoliths. This sensory linkage between canal and otolith is easily broken in a cornering vehicle whether the turn is flat, as in a car on an uncambered corner, or banked as when cornering on a motorcycle or flying a co-ordinated turn in an aircraft (Fig 1).



F = Inertial Force of radial acceleration
G = Force of Gravity
R = Resultant Force

Figure 1. The forces acting on a cornering road vehicle and an aircraft in a co-ordinated turn. The postural response of the car driver is to lean into the corner whereas the passenger tends to be thrown outwards.

In a flat turn the otoliths sense the resultant acceleration formed by the addition of the gravity vector to the radial acceleration produced by the curved path of the vehicle. This resultant inertial force is directed downwards and outwards, but its outward swing from the vertical as the vehicle enters the corner is unaccompanied by any corresponding rotation signal in roll from the semicircular canals. This conflict can be minimised by a reduction of speed when cornering. The amplitude of the radial acceleration for a given radius of curvature is proportional to the square of the vehicle forward velocity, hence a small reduction in velocity give a disproportionately large reduction in radial acceleration and consequent sensory conflict.

In a co-ordinated turn the aircraft is rolled by an amount which maintains the resultant vector directed at right angles to the aircraft floor. The sensory conflict engendered involves a perception of roll without any relative change in the direction of acceleration sensed by the otoliths. Airline pilots minimise the conflict by limiting the rate of roll to levels approaching the 2 deg.s^{-2} threshold of perception, and by limiting the angle of bank to 30deg so that there is only a small increase, to 1.15G_g, in the intensity of what is perceived as gravity by the passengers.

Low frequency oscillatory motion. In larger ships such as passenger ferries the principal oscillatory motions of the vessel are vertical translation, and pitch and roll rotations (Lawther & Griffin 1986). The centre of roll rotation is well below the water line and therefore gives rise to lateral translation that is greatest for the upper decks. Of greater importance, the pitch motion of the vessel, which occurs about a transverse axis that is roughly mid-way along the length of the ship, results in a considerable increase in vertical oscillatory motion at the bow and stern of the vessel. The peak acceleration amplitude of vertical motion which for medium sized vessels is generally in the frequency range 1.5-3.0Hz can vary by a factor of 10-20 along the length of a 100m long vessel (Griffin, 1990).

To minimise the nauseogenic consequences of this type of motion one of the most effective strategies a susceptible passenger can adopt is to station himself at the position of minimum vertical excursion, over the axis of pitch motion of the vessel. If the ship is rolling heavily, some reduction in the consequent horizontal oscillatory motion can be achieved by being on as low a deck as possible, consistent with the need for a clear view of a stable horizon in order to avoid visual conflict.

The frequency of the vertical oscillation of the vessel, a critical factor in the nauseogenicity of this type of motion stimulus, is affected by the heading of the boat with respect to the direction of advancing waves. This is particularly true of sea-going sailing boats. When beating to windward there is often more pounding of the boat as it meets each wave and, while it may be uncomfortable, it is generally less nauseogenic than when running before the wind. In this condition the wave frequency is reduced and there is an accompanying forward acceleration and deceleration of the boat as it is overtaken by each wave. For larger vessels under power, motion conditions may be more nauseogenic when heading into the oncoming waves (Applebee et al. 1980).

In cars frequent braking and acceleration generate what is in effect a low frequency horizontal oscillatory stimulus and this can also be nauseogenic for susceptible vehicle occupants.

Vertical oscillatory motion is encountered in aircraft when flying in turbulence and may lead to airsickness. In commercial flying this is nowadays infrequent since passenger aircraft fly at altitudes well above the turbulence of the weather. It may occur if the aircraft encounters clear air turbulence at altitude or storm cloud activity on the approach but such problems can often be avoided by re-routing. By contrast, in military flying the vertical oscillatory motion induced by turbulence is a feature of low-level flying which can in certain weather conditions be very severe.

The influence of head movements.

When undergoing rotation, head movement in any plane other than that of the rotation will induce a cross-coupled vestibular signal that is potentially disorientating and also nauseogenic. Aircrew are instructed when manoeuvring in cloud to minimise head movements, principally in the interests of avoiding disorientation. Such advice to aircrew during manoeuvres in clear visual conditions is not appropriate however, because of the need to maintain good look-out when changing heading.

In abnormal G environments head movements are also provocative of motion sickness both in the zero gravity environment of space, (Oman et al, 1986), in parabolic flight ((Lackner & Graybiel, 1984, 1986), or during high G manoeuvres in fast jet aircraft. The elimination of unnecessary head movements in these circumstances will therefore be protective against motion sickness.

It would seem to follow that head movements made in the alternating hyper- and hypo-gravity environment produced by low frequency vertical oscillation would be an additional factor in the provocation of motion sickness. This, however, has not been clearly established. A study of motion sickness induced by vertical oscillatory motion found no potentiating effect from the addition of head movements or whole body pitch and roll movements (McCauley et al, 1976). Initial trials in paratroops of the use of restraints to minimise head movement found significant benefit, but only in conditions of "normal turbulence". In conditions described as "rough" or "violent" no significant differences in the incidence of airsickness were found (Johnson & Mayne, 1953). A similar study (Keist et al, 1985) failed to show any benefit from head restraint, whereas hyoscine 0.65mg taken 1 hour before flight reduced the incidence of vomiting from 31% to 7.5%.

The influence of body posture.

Several experimental studies have investigated the effect of body orientation in relation to the direction of low frequency oscillatory motion.

A study in aircraft undergoing roller-coaster type manoeuvres showed that subjects sitting upright were less likely to become motion sick than subjects sitting with the head and trunk flexed forward to the horizontal (von Baumgarten et al, 1980). A similar study, which used alternating periods of weak acceleration at 0.15g and forceful braking at about 0.8g in an ambulance car, found that subjects were less tolerant of this stimulus if sitting up rather than lying down. These studies would suggest that oscillatory motion is more nauseogenic when applied through the X (dorso-ventral) axis of the body than through the Z (cranio-caudal) axis (Vogel et al 1982). However, in the treatment of seasickness it is well known that seasick mariners are better if made to lie down. It is possible therefore that an oscillatory stimulus is best tolerated in a posture which requires a minimum of postural regulatory activity in order to maintain it. A study to compare the nauseogenic effect of 0.3Hz sinusoidal oscillation at $\pm 2.5ms^{-2}$ applied vertically to seated subjects and horizontally to supine subjects (Golding & Kerguelen 1990). The finding that oscillation in the horizontal plane with the subject supine was less provocative than vertical oscillation is in keeping with this proposed relationship to body posture. However, the findings could also be related to the changed nature of the sensory conflict between the two conditions.

Effect of being in control of the vehicle.

It is well known that the driver of a car or coach is spared the motion sickness that may, as a consequence of his energetic driving, afflict his passengers. Several factors may contribute to the relative immunity of the driver, but probably the most important is his ability to anticipate the effects of his actions in accelerating, braking and cornering. In consequence, the driver will lean into the corner while his passenger tends to be thrown outwards, and likewise, the driver will brace himself in preparation for braking whereas his passenger is initially thrown forwards. An abrupt change in the force environment generates proprioceptive signals that are suddenly at variance with those expected as a consequence of the existing postural regulatory efferent activity. It may be that this constitutes a neural mismatch signal that contributes to other discordant motion signals in the provocation of motion sickness.

A similar benefit from being in control is enjoyed by the pilot of an aircraft as compared with his navigator, though this may be less evident in early training while he is still becoming accustomed to the sensory consequences of his control actions. Likewise seasick crew members on sailing boats may derive some benefit from taking the helm.

A study to investigate the effect of the subject having control over the initiation of a cross-coupled stimulus (Reason & Benson, 1978) used three stimulus conditions. Subjects were rotated at incrementing velocities on a turntable and tilted in roll by 45 deg either passively by means of a mechanical actuator under operator control, or actively using their own muscular effort, or in an active/passive condition in which the subject initiated the actuator-driven chair tilt. Assessment of motion sickness symptoms indicated the passively rotated group to be the most affected, and the active group least so.

A further benefit of being in control may derive from the mental distraction that it provides. There is experimental evidence to suggest that motion sickness symptoms are reduced by mental activity that decreases the subject's awareness of the provocative motion. In a study of vestibular responses to whole body rotation about an earth-horizontal axis (Correia & Guedry 1966), a high incidence of motion sickness was reported. Of the 12 subjects who failed to complete the test procedure on

this account, all were in the sub-group who had been asked to report accurately their sensations of motion, whereas all subjects who were assigned a key press or a mental arithmetic task were able to complete the test.

Adaptation/habituation to provocative stimuli.

In addition to behavioural measures, a further important factor in reducing motion sickness susceptibility is that of adaptation. In this context adaptation refers to the increase in tolerance to a nauseogenic stimulus that occurs over a period of several days or even weeks of repeated exposure. The neurophysiologist would use the term habituation for this phenomenon, and would reserve the term adaptation to refer to changes in the magnitude of the response during the course of the application of the stimulus. Both types of adaptive response can be seen with motion sickness. Exposed to an appropriate level of stimulus over say 30 minutes an individual may show an initial loss of well-being followed by partial or complete recovery indicative of a short term adaptation within the period of the test (Fig 2).

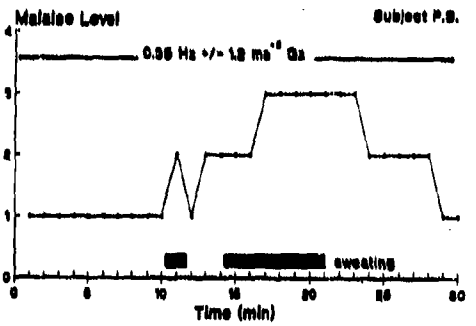


Figure 2. Short term adaptive response to a 30 min exposure to a constant intensity low frequency vertical oscillation. The onset and cessation of forehead sweating reflects the changes in subjective well-being.

Researchers have attempted to use other measures of adaptation such as the rate of disappearance of illusory sensations produced by repeated head movements in a rotating environment (Reason 1972). It is not however clearly established that such tests of perceptual adaptation are predictive of the rate of increase in tolerance to nauseogenic motion.

The phenomenon of adaptation is well known among mariners in whom seasickness symptoms abate during the first few days at sea accompanied by an improvement in locomotor co-ordination aboard ship, a phenomenon colloquially known as 'getting one's sea legs'. Adaptation is also a feature of aircrew training. The familiarisation sortie may give rise to aircrew sickness in 15% of aircrew and flying instructors may have to resist the temptation to show off to the student the full capabilities of the aircraft at this stage in training. Tolerance builds up with continued flying but airsickness may recur with the introduction of new manoeuvres such as steep turns, spinning and aerobatics (Tucker et al, 1985). In space flight the malaise and nausea that may initially be provoked by head movements in the zero gravity environment gradually diminishes, typically over a period of 2 to 4 days.

Similarly in laboratory experiments, the wearing of image-inverting spectacles initially provokes severe visual disorientation and, with continued efforts to carry out normal activities, symptoms of malaise and nausea. After wearing spectacles continuously over several days, nausea is less readily provoked and locomotor activities become more normal (Mikaelian & Held, 1964). Likewise, experimental subjects who spent several days living in a room that rotated about a vertical axis at 10 rpm showed adaptation to both the neuromuscular and nauseogenic consequences of this environment (Graybiel et al, 1969).

A distinction can be made between those environments that produce a consistently reorganised relationship between the various sensory outputs of motion such as the microgravity environment of space, the continued wearing of image reversing spectacles, and to a lesser degree the motion environment of a ship, and those environments to which exposure is brief and intermittent, such as flight involving aerobatics.

In the former group of environments it is hypothesised that new sensory inter-relationships are established that to some extent replace or overlie those that previously existed. As a consequence the return to the normal terrestrial environment is associated with a temporary degree of mal-adaptation and even mild motion sickness symptoms, termed mal de débarquement.

Adaptation to aerobatic flight may be qualitively different and involve an extension of the repertoire of patterns of sensory input that the brain accepts as legitimate.

Adaptation treatment for motion sickness.

The use of adaptation as a therapeutic procedure is most appropriate to those situations in which, following treatment, there is sufficient continuity of exposure to the stimulus to maintain the state of adaptation and also in which the economic or personal consequences of continued motion sickness make a time-consuming treatment programme worthwhile. Though spaceflight fits these criteria it is impossible as yet to predict who will suffer spacesickness and uncertain as to whether tolerance acquired to any earthbound stimulus would transfer to the microgravity environment in space. It is perhaps in the treatment of airsickness in military aircrew that adaptation treatment has proved to be of most value.

In 1968 Dowd described the successful

return to flying of an airsick pilot who over a 1 week period received twice daily sessions of cross-coupled stimulation.

Treatment programmes for airsickness in student pilots were started in 1965 both in the RAF by Dobie and in the USAF by Dowd. Cross-coupled stimulation was used as a means to provoke symptoms of motion sickness and subjects increased their tolerance to this stimulus by incremental exposures. Unlike the US programme, that in the RAF involved from the outset a period of remedial flying before the individual was returned to training. Review of the first four years of the US programme showed that of 75 aircrew treated, 48% had continued in training for the 3 month follow up period. The comparable success rate in the first 50 cases treated in the RAF programme was 85% (Dobie, 1974), and it has continued to be at or above this level. It is therefore tempting to ascribe this difference to the inclusion of remedial flying.

The RAF airsickness desensitisation programme at its present state of evolution consists of a 4 day assessment phase, a 4 week ground phase and a 3 week flying phase. (In practice, owing to the vagaries of weather, aircraft serviceability and aircrew illness, only the assessment phase is of a fixed length).

The assessment consists of a medical interview, vestibular function tests (post rotational and sinusoidally induced nystagmus and positional test), psychometric tests (Eysenck Personality Inventory and Cattell 16 Personality Factors tests) and assessment of initial tolerance to the ground-phase stimuli (cross-coupled stimulation and low frequency (0.35Hz) vertical oscillation). During the ground phase of treatment subjects undergo twice daily sessions of either cross-coupled or low frequency vertical oscillation stimuli (usually the same stimulus in any given week) but with an overall bias towards cross-coupled sessions.

During cross-coupled stimulation the subject sits in an enclosed cab on a turntable and makes head movement sequences to and from each quadrant in pitch or roll in random order over a 30 s period. During the earlier part of the course the rotational velocity of the chair is incremented from zero by 1, 2, or 3 deg.s^{-1} after every head movement sequence. The rate of increment is determined on the basis of initial susceptibility, the more susceptible subjects being assigned lower rates of increment and thus relatively longer session duration. Later sessions are started at higher rotational velocities. Every 30s the subject reports his well being on a 1 to 6 scale; the session is stopped as soon as a rating of 4 (moderate nausea) is reached. Research is currently in progress to study the effect on adaptative rate of stopping the session at level 2 (mild malaise).

Sessions of low frequency vertical oscillation are carried out on a 2m stroke platform oscillating at 0.35Hz. Nausegenicity of this stimulus is intensitified by an enclosure that precludes a view of the stable visual world and by making the subject carry out a visual search task. The severity of the stimulus is also determined by the peak acceleration level. The same level is used throughout each session and is set to a level, based on survival time in previous sessions, that the subject would be expected to tolerate for 20-30 minutes.

Under this treatment regime most subjects show adaptation as evidenced by an ability to tolerate increased stimulus intensities or by longer survival times. Also in the course of adaptation subjects find that following a treatment session, their recovery time from the same level of malaise becomes shorter. By the end of the ground phase the expectation is that the majority of subjects will tolerate 20-30 head movement sequences while rotating at 60 deg.s^{-1} and will survive for 20 minutes vertical oscillation at 0.35Hz ±3.5ms^{-1}. While some exceed this criterion, others show a very poor capacity to adapt (Fig 3). This failure to adapt seems to be more common among aircrew referred from maritime reconnaissance aircraft in which, compared with aerobatic fast jets, the stimulus level is relatively low but continues for 6-8 hours. Aircrew who continue to have motion sickness problems on this type of aircraft are probably failing to adapt, not just between successive sorties, but also within the duration of each sortie.

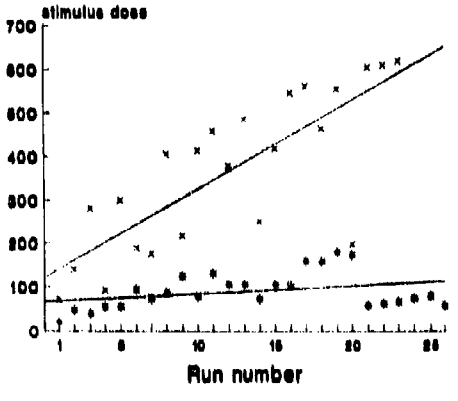


Figure 3. Long term adaptive responses in two individuals exposed to twice daily sessions of incrementing levels of cross-coupled stimulation.

Following the ground phase, pilots and navigators fly a 15 hour course under the supervision of a dedicated pilot. The flying is graded in the introduction of provocative manoeuvres according to the subject's well-being during the sortie. In its early stages the course is similar for both pilots and navigators and both groups are encouraged to take control of the aircraft. In the later stages pilots learn to tolerate increasingly vigorous aerobatic sequences while navigators undertake the navigation of low level routes and target runs (Bagshaw & Stott, 1985).

The transfer of adaptation.

Implicit in the use of ground-based cross-coupled stimulation is the assumption that tolerance acquired to this stimulus will transfer to the motion environment of the aircraft. This proposition has not formally been tested. Much of the experimental work on adaptation (reviewed in Parker & Parker 1990, Stott 1990) tends to indicate that the short term adaptation that occurs over minutes or hours is highly specific to the initiating stimulus, but that a somewhat more generalised adaptation is evident over a period of days. Some degree of correspondence can be discerned between the laboratory stimuli and the aircraft motion environment. Low frequency vertical oscillation is one nauseogenic component of flying in turbulence. Similarly, cross-coupled stimuli can result from a pilot making head movements when the aircraft is in a sustained turn. This is particularly so in the high rates of turn that can be achieved by aerobatic light aircraft or by gliders flying in thermals. However, the cross-coupled stimulus is less severe if the turn is not sustained or if, as in high speed flight, the rate of turn is low.

High performance aircraft manoeuvres often involve high G, and head movements in this environment can also be both disorientating and nauseogenic. It is unclear whether tolerance acquired to a laboratory cross-coupled stimulus transfers to the high G environment. Still less likely is it that cross-coupled tolerance can give protection against the low frequency vertical oscillatory stimulus typical of flight in low level turbulence. Laboratory studies on the transfer of adaptation found no increase in cross-coupled tolerance as a consequence of an increased tolerance to low frequency vertical oscillation acquired by repeated exposure to this stimulus for 2h/day over 15 days (Potvin et al, 1977).

Factors influencing the rate of adaptation.

The rate of adaptation varies widely between individuals both in adaptation to novel motion environments and to laboratory motion stimuli. Although a period of 2 to 4 days is often quoted for adaptation to the space motion environment, some astronauts have experienced space sickness for longer periods. In a review of space motion sickness in Russian missions one cosmonaut suffered space sickness throughout the 15 days of the mission (Matsnev et al, 1983). The persistence of sea sickness symptoms during a voyage is less clearly evidence of slow adaptation since, on account of the variability of the sea state, periods of calm could lead to the loss of previously gained adaptation.

Graybiel and Lackner (1983) compared the adaptive rates of 14 individuals to three different nauseogenic stimuli and found that qualitative estimates of adaptive rate for each stimulus were similar within subjects. A study of the adaptive response to repeated cross-coupled and to low frequency vertical oscillation stimuli in aircrew referred for treatment of airsickness failed to show a correlation between the rates of adaptation to the two stimuli (Stott, 1990).

Although the adaptive rate may be an individual characteristic it may be possible to influence the rate of adaptation in the laboratory by the appropriate choice of conditions. For example, it is likely that if each treatment session repeatedly provokes vomiting this will be counter-productive to the process of adaptation. Several experiments to study the effect of different stimulus profiles in adaption were conducted in the Pensacola Slow Rotation Room (Graybiel et al, 1965, 1969). Subjects spent up to 16 days continuously in this rotating environment. The responses of subjects who began rotation at 10 rpm from the outset was compared with various incremental approaches to the final velocity. Using a protocol of nine increments over a period of 16 days adaptation was achieved in four subjects without motion sickness symptoms other than excessive drowsiness. By contrast four subjects exposed to the abrupt onset of 10 rpm experienced nausea, two of whom vomited, one repeatedly, and it was judged that none had fully adapted by the end of a 12 day period.

In these conditions in which motion sickness is provoked by movement in an atypical environment, for example, wearing image inverting spectacles, in microgravity, or in the slow rotation room, active movements by the subject are likely to be more effective in promoting adaptation than when the subject is passive and moved by external means. It is postulated that voluntary movement generates proprioceptive and tactile sensory signals that feed back to the brain (reafference) and are compared with what is termed an efference copy - an expected sensory pattern based on the initial efferent motor activity (Von Holst 1954). Failure of the reafferent signal to match the efference copy leads to illusory sensations and corrective motor activity and, if a consistent new sensory pattern persists, to an adaptive response.

Anti-motion sickness drugs have been used in association with repetitive nauseogenic stimuli in the expectation that they will facilitate the adaptive process. In a study to test this possibility (Wood et al, 1986) it was found that hyoscine 0.6mg with amphetamine 10mg or hyoscine 1.0 mg alone produced a more rapid rate of increase of tolerance compared with placebo over the 3 days on which the drug was taken. However, when the subjects were tested 1 day after stopping medication, tolerance had decreased towards pre-treatment levels and was significantly less than the level of tolerance acquired by subjects on placebo. This abrupt fall in tolerance was ascribed to a pharmacological rebound effect, but an equally valid conclusion is that scopolamine hinders the process of adaptation. It may be of relevance that hyoscine has been shown to inhibit verbal learning process (Crow, 1978). There is some neurophysiological evidence to suggest that adaptation to conflicting motion stimuli involves what could be considered as cerebellar learning mechanisms.

Autogenic feedback training.

Autogenic training, has been developed as an alternative to repetitive stimulus adaptation to alleviate space sickness (Cowings et al, 1977). The essential problem in attempting to pre-adapt individuals to the motion environment of space is the inability to produce a weightless environment for adequate lengths of time to enable subjects to build up tolerance to movement in zero G.

Autogenic feedback training is described as a combination of biofeedback and autogenic therapy. It is based on the proposition that the acquisition of voluntary control over certain of the autonomic responses that characterise motion sickness would allow an individual to attenuate at will the whole symptom complex and thereby increase his tolerance. Furthermore if the training were aimed at control of the expression of motion sickness symptoms it might be possible to achieve a tolerance to discordant motion that was not confined to a specific type of stimulus. The technique involves both visual display and auditory feedback of several autonomic responses that occur during the development of motion sickness. In the studies carried out by Cowings & Toscano (reviewed in Cowings, 1990), the monitored variables were: heart rate, respiration rate, plethysmography of the finger and of the skin adjacent to the mouth, skin conductance of the fingers and electromyographic activity in intercostal muscles. Subjects were taught to control some of these responses both in the direction of reducing the autonomic response to motion sickness and also in the opposite direction.

In a series of experiments it was shown that tolerance to cross coupled stimulation administered at weekly intervals increased at a faster rate in subjects who received autogenic feedback training than in controls or in subjects given a cognitive task to perform. It was also shown that there was an increase in tolerance to other nauseogenic stimuli namely, head movements during 0.33 Hz ±0.36 G vertical oscillation, and head movements during simultaneous chair rotation and yaw axis optokinetic stimulation.

An assessment of the technique has hitherto involved only one space mission. Two of the four astronauts on the mission received autogenic feedback training. All four astronauts suffered some symptoms of space sickness but these were least in the individual who was considered to have achieved greater autonomic control following treatment.

A similar technique has been used by the US Air Force in conjunction with cross-coupled stimulation as a treatment for airsickness in aircrew (Jones et al, 1985). Of 83 aircrew accepted for treatment, 79% successfully returned to flying and a further 6% were partially successful. This treatment also forms part of the Canadian Air Force program for the treatment of airsickness (Ceresia, 1983).

Alternative treatments.

There has been recent interest in the use of elasticated bands sold as "Sea Bands" or "Acubands", that apply pressure to a point above the wrist, known to acupuncturists as the P6 or Nei-Kuan point, as a prophylactic treatment for motion sickness. Two controlled trials have failed to show any benefit of this treatment (Bruce et al, 1990; Vijtdehaage et al, 1991). Nevertheless, the placebo effect of any form of treatment, prescribed with conviction, may be beneficial to some individuals.

While acupressure is not significantly better than placebo, self-administered electrical acustimulation has been shown to be of benefit when compared with a control group who received no treatment, and with a placebo group in which the device was not switched on but who were told that the frequency of the stimulus made it imperceptible. However, even this experimental protocol does not eliminate the placebo effect of having perceptible, and perhaps distracting, confirmation in the form of an electric shock that the therapeutic device is doing something.

Similar considerations about the placebo effect apply to the conductive strips that dangle from the rear of cars to prevent motion sickness in the occupants by discharging to earth any accumulated static charge on the vehicle. Even if static electricity were to play some part in the development of car sickness, the charge on a hollow metal box is always on the exterior surface while the interior is always at zero potential.

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ASSESSMENT OF DRUG EFFECTIVENESS

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Motion sickness continues to be a problem encountered in aviation, as well as in other means of travelling (1,2). Recent studies (3,4) have stressed the fact that the main treatment is still based on the use of motion sickness suppressants. Although a variety of drugs is available, none of them provides a watertight solution.

Motion sickness as an entity exists as long as men makes use of artificial means of transportation. As long as men suffers from motion sickness methods of prevention and ways of treatment have been developed and are still under development.

Besides advices about food and fluid intake, the use of necklaces, a metal ring around the arms and plugs into the external ear canal at the site opposite the right- or left handedness, pharmacotherapy is as old as the problem itself.

An important issue in the search for drugs against motion sickness is that not only a satisfactorily action and prevention exists, but also that a compound is free from any disturbing side effect. Side effects which include any influence on the working capacity in any respect.
This regards especially crew members who have a responsible task. When it concerns passengers side effects are much less important.

So two different groups can be distinguished in the search for compounds active against motion sickness.

For centuries the search methods for new drugs were restricted to experiments and experiences with drugs in so-called field conditions.

Laboratory instruments are able to provide a means of investigation in order to evaluate qualitatively and quantitatively the effect of a drug on the vestibular system.
It is assumed that an effect on the resistance against motion sickness of subjects has a relation with the resistance against sickness provoked by stimulation on a rotation chair or in a rotation room. It should resemble the sensitivity for motion sickness.
However, the final assessment of a drug is always made in the particular motion sickness condition itself.

The assessment of anti-motion sickness drugs into the laboratory sounds much more attractive, however there is always the lack of reality, the lack of stress as well as to some extend the lack of sway movements and vertical movements.

When a drug is expected to exert a suppressing effect on motion sickness, the available laboratory test methods should not only provide objective measurements about the potency and the duration of action, but should also allow to eliminate adaptive mechanisms by the body, as well as psychic influences. In addition this research model can use a placebo-group, which is usually impossible in clinical studies of this nature.

Old prevention methods concerned behavior activities such as eye fixation at the horizon, and controlled respiration. Also eating onions in order to avoid an abdominal vacuum. Furthermore a whole bunch of advises with regard food and liquid intake as well as the use of a spread of products.
The use of a drug was first mentioned in 1869 in "the Lancet" where in a letter a combination of chloroform and tinctura of belladonna was recommended. This was also the first mention of a belladonna derivative.

A study published in "the Lancet" revealed that between 1829 and 1900 practically everything that could be carried, worn of swallowed was prescribed at one time or another. These prescriptions were a.o. opium, cocaine, strychnine, creosote, quinine, nitrous oxide, amyl nitrite, hydrocyanic acid, nitroglycerine, warm salt water, strong tea or coffee as well as a variety of alcoholic beverages (3).

The sea-land operations in the Second World War initiated the development of modern drugs because of the military necessity.
It became apparent that in the design of motion sickness drug trials careful control was of uttermost importance because of the many influential variables involved.

In experiments several methods were used, all providing a particular combination of movements. Many different devices were constructed imposing a controlled motion upon their occupants.

Method	duration (h)	investigator
small ships	1 - 4	Hill 1945 (6), Glaser 1951 (7)
aircraft	1 - 7	Chinn 1953 (8), Strickland 1949 (9)
life rafts	1	Glaser 1952 (10)
swings	1/3	Smith 1946 (11)
vertical accelerator	1/3	Johnson 1964 (12)
slow rotation room	1/2	Graybiel 1960 (13), Wood 1968 (14), Wood 1970 (15)
troopships Atlantic	48	Gay 1949 (16)
mine sweepers	48	Arner 1958 (17)

However, there were many doubts as to the relevance and validity of the devices in the evaluation of anti-motion sickness drugs. The suspicions were partly confirmed as it appeared that drugs with a proven value against seasickness were relatively ineffective when tested by the motion of swings, accelerations or life rafts.
Furthermore the effect of a drug was usually assessed by means of its effect on symptoms as vomiting, sweating and general feelings of well-being. Real objective measurements were not available.

In considering the efficacy of a drug the size of the given dose is for many reasons an important factor. Usually a drug has a range of doses over which a gradually increasing effect will be observed until a maximum is achieved. Very important is also the time interval between intake and maximum effect. Meclozine, a long-acting drug, active when given in a once daily dose in experimental conditions at sea for 48 hours appeared no more effective than a placebo when given in the same dose, but only 2 hours before exposure to 60 minutes in a life raft. So many examples can be given with regard the moment of intake, the moment of maximum action and the duration of the effect.

In this respect the following diagram (5) gives a good example of the effective blood level to time of experimental observation. The moment the test procedure is applied gives false information in case b (fig. 1).
It means that for every drug to be investigated at a number of intervals after the administration a test has to be conducted.

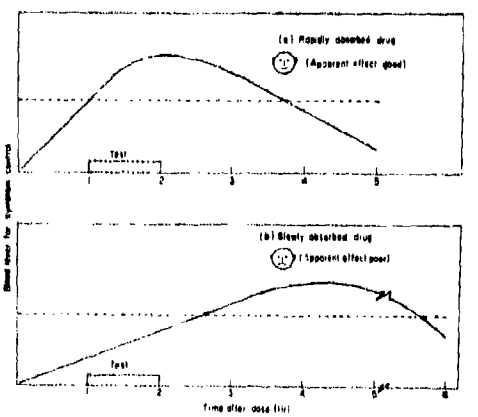

Fig. 1. Diagram relating effective blood level to time of experimental observation.

In the publication of Wood (18) the anti-motion sickness drugs at that time were divided into four groups: anticholinergics, antihistamines, tranquilizers and miscellaneous.
It is nowadays clear that such a classification does not serve any purpose.
The Pensacola investigators, headed by Ashton Graybiel, made a review of the available drugs in the sixties and with the use of the slow rotation room made studies about the effectiveness.

Several laboratory techniques were used in rather recent times.
The cupulometry was first described by Van Egmond (19) and later used for many years as a screening test for the sensitivity of the vestibular system. With this test an individual is subjected to an acceleration on a rotation chair till a constant velocity is reached. Then, following a sudden stop both the length of the provoked rotational sensation as well as the postrotational nystagmus is measured. The change in these parameters is assumed to give insight into the effect of a drug on the vestibular system, and so on the sensitivity for motion sickness.
Cupulometry is not very popular anymore in motion sickness studies, especially since Dobie (20) was not able to confirm the assumption that motion sickness susceptibility could be determined in an extensive study in 1000 human subjects.

With the dial test (21) a stimulus was administered for the controlled production of motion sickness. The test requires the subject to make prescribed settings on a series of five dials situated at fixed positions around his chair. The arrangement of the dials is in such a way, that substantial head an body movements have to be made in order to make the setting. The head movements are assumed to stimulate the nausseogenic coriolis vestibular reaction.
Well-being ratings were made at the completion of each sequence of five settings. The ratings were based on an 11 point scale: 0 = I feel fine, 10 = I feel awful, just like I am about to vomit.
Objective signs and subjective symptoms reports together have to be collected independently.
The method can be applied before as well as following the administration of an anti-motion sickness drug.
It has a great deal to commend it as technique for evaluating drug efficacy because it allows a quantitative assessment of the amount of stimulation required to reach a well-defined pre-vomiting end point.

Several drug studies have been published by Wood and Graybiel (22), Wood (23).
Fig. 2 gives an impression of the results. According to these studies the combination scopolamine amphetamine proved to be most effective.

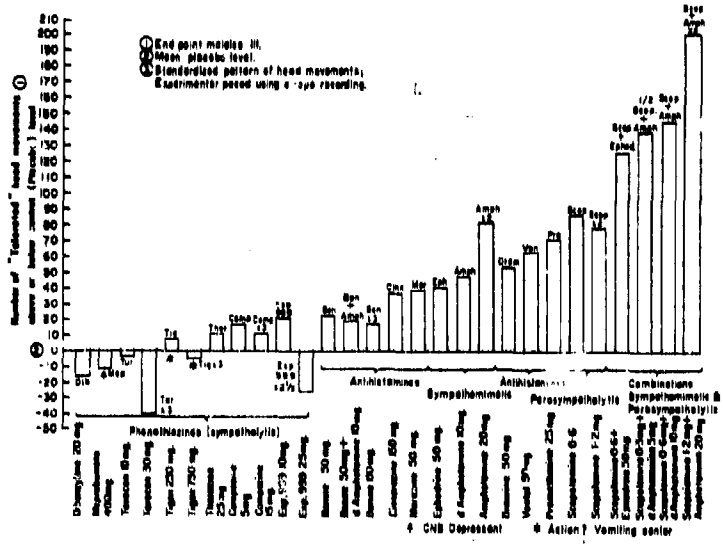

Fig. 2. Effectiveness of antimotion sickness drugs in preventing motion sickness in 50 subjects exposed on 500 occasions in a rotating environment using the Dial Test. From Wood and Graybiel (1968).

It must be emphasized that because of the chosen moments of measurements these results did not fit very well with the experiences in real motion sickness conditions.
According to Parrott (24) transdermal scopolamine is the most effective single drug for the prevention and treatment of motion sickness. Chelen (25) reported that phenytoin increased the tolerance to motion sickness considerably.
As an example of a drug study I present you the data of a trial conducted in our own laboratory.
The effect of the two drugs, of their combination, and of a placebo on angular acceleration-induced nystagmus and on linear acceleration-induced eye movements was examined in normal healthy human subjects. A compound such as Cinnarizine, which specifically suppresses the activity of the vestibular organ, has proven to considerably counteract torsion-induced nystagmus (26). It seems that under the effect of this compound, the labyrinth becomes less sensitive to stimuli, with the effect that reactive nystagmus starts at a later point of the sinus-shaped stimulus and that it stops sooner.
The linear acceleration and deceleration test, specifically on the parallel swing, makes it possible to observe a drug-induced reduction of the deflection of the nystagmus movement (27,28).
Domperidone, too, has an effect on the duration of nystagmus, but apparently via another mechanism of action (27,28). In addition, the pharmacokinetic profile of Domperidone, a compound with rapid absorption and relatively rapid excretion, is clearly different from that of Cinnarizine, which is slower in reaching its peak effect, and which is excreted more slowly. For this reason, it was decided to combine the two compounds and compare their combined action with that of the separate compounds.

Material and methods

Test subjects and schedule: The study was carried out in 25 healthy subjects. Each subject participated four times at weekly intervals. The tests were performed in the morning after the subject had a light meal.
The torsion-swing test: The torsion-swing provokes an angular acceleration which stimulates both horizontal semicircular canals. On the other hand the parallel swing provokes a linear acceleration which stimulates the otoliths.
The torsion swing produces rotating accelerations in alternating directions. In the tests, a 20-s oscillating period was used. The swing was rotated 90° in the horizontal plane and subsequently released. This produced a rotating movement of the swing in alternating directions. The slow rotation time of 20 s had a relaxing effect on the test subjects, while the stimulation, which was supraliminal, only induced a slight sensation of oscillation. The stimulation brought about a clear nystagmus, which was possible to record electronystagmographically.
The head of the test subject was fixated by means of a headrest with two pads. The subjects were blindfolded while the eyes were kept closed. Regular electronystagmo-graphical electrodes were placed on both sides of the head at the temporal bones.
Only eye movements in the horizontal plane were recorded. At the beginning of a sequence a subject was subjected

to the torsion swing test and exposed to five complete oscillations, after which the test medication was administered in tablet form. At fixed points in time, after the administration of the medication, the torsion swing test was repeated. Each time subjects were exposed to five complete oscillations. For the calculation of the effect of the compound on the duration of the nystagmus, the time between the first and last nystagmus movement in a given direction was measured. For each subject, measurements were always based on the same direction of nystagmus.

The parallel-swing test: For the second part of the investigation, a parallel swing was used. This consisted of a load-bearing surface suspended from the ceiling with 4 ropes. The maximum deflection of the swing was 125 cm, maximum acceleration 348 $cm.s^{-1}$, and maximum speed 205 $cm.s^{-1}$. The time required for a complete oscillation was 3.7 s. With the parallel swing, a sinusoidally alternation linear acceleration was produced, which gave rise to vestibular eye movements. As linear acceleration is a specific stimulus for the otoliths, the parallel swing test may be regarded as a test for the otoliths. When a subject is swung sidewards, a sinusoidal eye movement is elicited that is in phase with the swing movements. These movements are caused by stimulation of the otoliths and of the utriculi.

The subject was positioned on the swing with his head fixed in position on a headrest between two clamps. The eyes, which were closed, were covered with a piece of cloth. The swing was then pulled away laterally over a distance of 125 cm and released, resulting in the subject's body making lateral swinging movements. The compensatory eye movements were registered as described above. For each test the subject was swung to and fro 10 times. After subjects had been submitted to the test 3 times at 15-min intervals, the drug under study was administered, after which the test was repeated at fixed points in time.

Before each test, the apparatus was calibrated by having the test subject with fixed head, look in alternating fashion at two fixed points on the ceiling; an eye movement of 20° was made to correspond to a 20 mm deflection on the registration paper.

The swing-induced registered eye movement was a sinus movement. For purposes of calculating the effect of the compound administered, the maximum eye movement deflections were averaged for each subject. Each subject completed the test four times, after receiving a different drug each time. Thus, it was possible to compare changes in the registered eye movements.

Medication: In both parts of the study, randomization was used for the sequence of the four treatments in each subject (placebo (pl), Cinnarizine (C), Domperidone (D) and Touristil (C+D)), as well as for the order of administration to all subjects per study session. Care was also taken that each drug be given with equal frequency in the 4 phases. The investigation was carried out in double-blind fashion, i.e. neither the subject nor the investigator was aware of the composition of the tablets, which were all the same colour, shape, and size.

Results

Intensity of the vestibular inhibition: During both rotation and linear movement, Touristil (C+D) caused a significantly greater reduction ($p<0.05$) of the duration and amplitude of nystagmus and eye deviation only 15 min after the intake. As early as 30 min after intake, Touristil (C+D) performed significantly better ($p<0.01$) than the other medications. The shortening of the duration of nystagmus obtained with (C+D) was exceptional in the rotation test: almost 61% (± 4%) vs. a maximum shortening of 28% (± 6%) for (D), 37% (± 6%) for (C), and 3% (± 5%) for (pl) (Fig. 3).

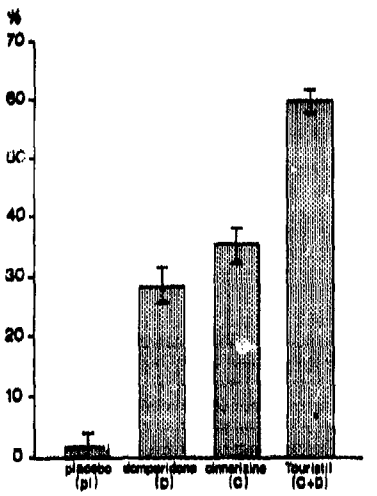

Fig. 3. Vestibular depression (shortening of duration) during stimulation torsion swing. Peak effects of four medications

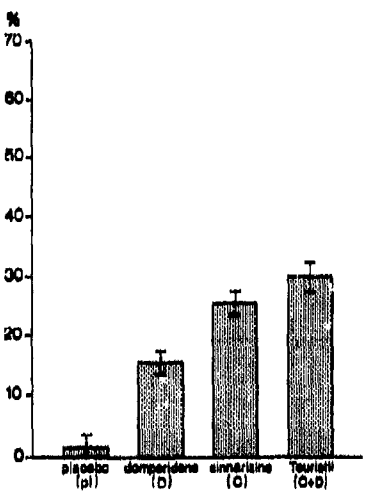

Fig. 4. Vestibular depression (reduction of amplitude of eye movements) during stimulation on parallel swing

The average maximum reduction of amplitude in the parallel swing test was 2 (± 5), 16 (± 4), 26 (± 4) and 30 (± 5) for (pl), (D), (C) and (C+D), respectively (Fig. 4). In increasing order of potency, the following series was obtained: placebo, Domperidone, Cinnarizine and Touristil. Each of these "steps" was statistically significant ($p<0.05$). The median maximum inhibition (Fig. 5) was 4%, 30%, 39%, 62%, for (pl), (D), (C), and (C+D), respectively, in the rotation test. The median amplitude reduction was 6%, 17%, 26%, and 34% in the linear test. As previously, the differences were statistically significant ($p<0.0001$).

Time of maximum vestibular inhibition: Maximum vestibular inhibition was reached after an average of 1 h (± 12 min) with (D), 2,24 h (± 42 min) with (C) and 2.12 h with Touristil (C+D) in the rotation test (Fig. 6). This maximum inhibition occurred at a significantly earlier point in time with (D) than with (C+D) and (C) ($p<0.0001$), while the difference between (C+D) and (D) was not significant (Fig. 7). The maximum effect in the parallel swing test occurred at the end of 2.53 h (± 58 min) for (C+D), 2.14 h (± 35 min) for the placebo, and 1.24 h (42 min) for (C) and (D).

The graphs (fig. 8 and 9) of the course of the vestibular inhibition during the 4-h investigation give a clear picture of the effect of each medication. The placebo effect hardly deviates from the 100% line. Cinnarizine, with its well known inhibitory effect on the vestibular apparatus, is slower in reaching its peak than Domperidone. Touristil (C+D), like Cinnarizine, induces a very strong reduction of the duration of nystagmus at the end of approximately 2 h, however its peak effect is considerably stronger. These curves, which were derived from the two partial investigations (rotations and linear), showed an identical pattern. As already stated, the rotation test evidenced a stronger quantitative effect.

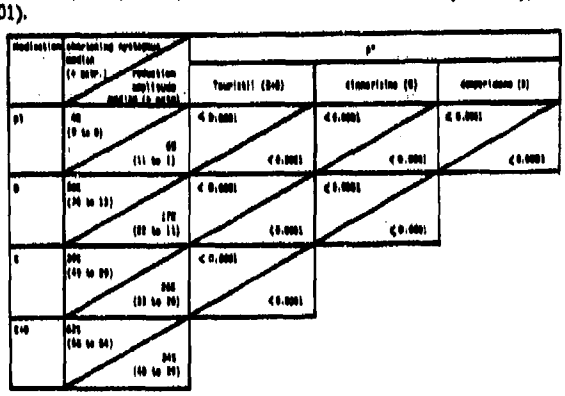

Fig. 5. Shortening of nystagmus (torsion swing) and reduction of amplitude (parallel swing) at time of maximum activity.

The 10% limit of vestibular inhibition: A shortening of the nystagmus by at least 10% in practice is assumed to correspond to an activity in the prevention of motion sickness (29). This level was reached with Touristil (C+D) in 16 test subjects within 15 min and in the total study group (n=25) within 30 min. With (D) the figures were 0 and 21 subjects, respectively; and with (C) 0 and 10 subjects (fig. 7). The mean time (±S.D) to the occurrence of 10% inhibition was 20 min with Touristil (C+D), while the same values for (D) and (C) were, respectively, 35 min and 1 h (fig. 10). These differences were also statistically significant ($p<0.01$).

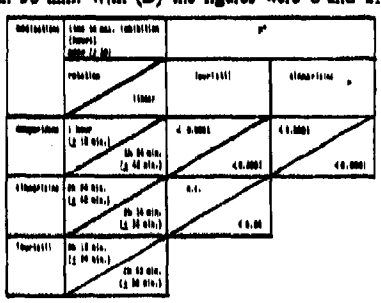

Fig. 6. Time to maximum inhibition with rotation and linear swings. Wilcoxon 2-tailed.

Although measurements were only taken up to 4 h after intake of the test medication, the duration of action of Touristil (C+D) as a medication against motion sickness in the present investigation was longer than that of (C) and (D) (Fig. 11). Again, the differences were significant ($p<0.001$). Contrary to the torsion swing, the evaluation criterium adopted for the same anti-motion sickness activity in the parallel swing test described in this paper was a less-than-10% reduction of the amplitude of nystagmus. A comparison of the data obtained using the two swings shows that the value in the parallel swing test must be around a 5% amplitude reduction. The times recorded for reaching this minimum of 5% amplitude reduction show a rapid effect, similar to that observed in the torsion swing. As before, the differences with (C) and (D) were highly significant ($p<0.0001$) and in favour of Touristil (C+D). In fact, this point was reached at the end of an average of 25 min with Touristil.

Superiority of Touristil to Cinnarizine: Fig. 12 and 13 show that the greatest difference in efficacy between Cinnarizine (C), the more potent of the two compounds, and Touristil (C+D) was seen in subjects who responded poorly to (C) on its own. The Pearson correlation factors were 0.84 for the torsion swing and 0.81 for the parallel swing, and both were highly significant ($p<0.0001$).

Side effects: No side effects were observed or reported during the study.

oral intake	Shortening of nystagmus duration rotation test n with 10%		n with 5% reduction of amplitude, parallel swing test			
	after 15 min.	after 30 min.	after 1 h	after 2 h	after 3 h	after 4 h
placebo	0 / 0	0 / 0	0 / 11	0 / 0	0 / 0	0 / 0
dexpertidone (D; 30 mg)	0 / 3	21 / 0	25 / 88	88 / 22	11 / 22	3 / 13
cinnarizine (C; 40 mg)	0 / 4	10 / 11	21 / 84	25 / 25	22 / 29	25 / 25
fenetelil (C; 60 mg D; 30 mg)	10 / 10	25 / 21	25 / 25	25 / 25	25 / 0	29 / 25

Fig. 7 Number of subjects with significant vestibular depression, i.e. 10% shortening of nystagmus duration or 5% reduction of amplitude. Total number of test subjects = n=25.

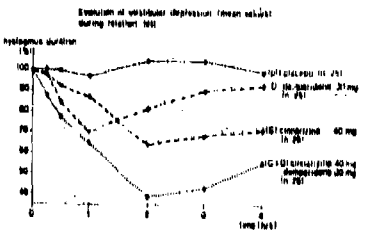

Fig. 8. The change in the duration of a rotatory-induced nystagmus in a time course of 4 h after drug administration.

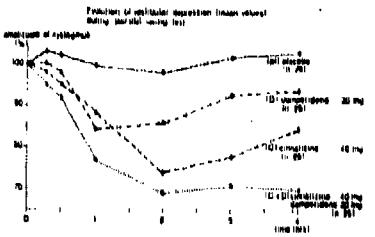

Fig. 9. The change in the amplitude of parallel swing-induced eye movements in a time course of 4 h after drug administration.

	Shortening of nystagmus duration by >10% / 5% reduction of amplitude	p¹		
		placebo	fenetelil	cinnarizine
dexpertidone	30 min. (± 11 min.) / 60 min. (± 13 min.)	< 0.0001 / < 0.0001	< 0.0001 / < 0.0001	< 0.01 / n.s.
cinnarizine	60 min. (± 31 min.) / 30 min. (± 20 min.)	< 0.0001 / < 0.0001	< 0.0001 / < 0.0001	
fenetelil	30 min. (± 7 min.) / 30 min. (± 11 min.)	< 0.0001 / < 0.0001		

Fig. 10. Mean time (±S.D.) until clinical effect (>10% of nystagmus duration) and >5% of nystagmus amplitude. Wilcoxon 2-tailed.

Compound	Median duration inhibition (+ extremes)	
placebo (p1)	0 h	(-)
domperidone (D) 30 mg	> 1 h	(30 to 4)
cinnarizine (C) 40 mg	> 3 h	(> 2 to 4)
Tourisitil (C+D)	> 4 h	(4 to > 4)

Fig. 11. Vestibular inhibition during stimulation on the torsion swing. The medians and the extremes of the duration of the nystagmus are indicated for all four components separately.

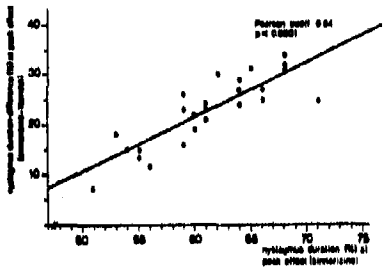

Fig. 12. Correlation between the additional effect of tourisitil and the effect of cinnarizine separately (rotation tests)

Discussion

The aim of the study was to examine the inhibitory effect of Tourisitil, a combination of Cinnarizine 40 mg and Domperidone 30 mg, on the labyrinth. Each of these two compounds exerts a vestibular inhibition separately, albeit through different mechanisms (1-5). The activity of Cinnarizine (C) is rather slow at the onset, and reaches a maximum after 2-3 hours, while the peak effect of Domperidone (D) comes about more rapidly (slightly more dan 1 h). It emerged from the present investigation that the two separate compounds (C) and (D), once in combination in Tourisitil (C+D), had a synergistic effect on the vestibular system in the form of an inhibition. The maximum reduction of about 60% obtained in this study is altogether exceptional in this type of investigation and clearly underlines the potency of the new combination. In addition, the rapid onset of action of Tourisitil is a further advantage of this new medicine against motion sickness (fig. 10). This prompt action, moreover, has long duration, which would be especially useful in cases of a long-lasting endogenous or exogenous excitation of the labyrinth.

Finally, it should be emphasized that both Tourisitil components are safe compounds. In addiction, this study shows that Tourisitil is most effective in those subjects who are not affected by (C), the most potent of the two, when given exclusively (fig. 12 and 13). This can be explained by the different pharmacological properties of the two components: Cinnarizine is a Ca^{++}-entry blocker and is known to be, amongst other things, a direct vestibular inhibitor. It is not known with certainty in what way Domperidone, a peripheral dopamine antagonist, affects the labyrinth. As there is an obvious relationship between the inhibitory action of a drug on the labyrinth and its value as a medication against motion sickness, it may be concluded that Tourisitil is a very potent preparation against motion sickness. In addition, Tourisitil approaches the profile of the ideal drug against motion sickness more closely than any other medication. In this connection the rapid, potent, and long-lasting effects of Tourisitil are of particular relevance.

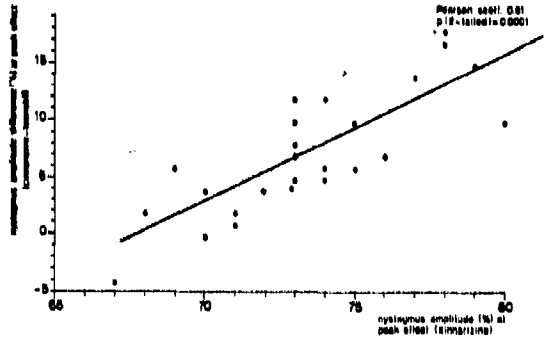

Fig. 13. Correlation between the additional effect of tourisitil and the effect of cinnarizine separately (parallel swing test).

Representation of the data of this study are meant to show you that laboratory models are useful to some extend. However, the final proof must always be given in the conditions where a drug is expected to work.

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MANAGEMENT OF ACUTE AND CHRONIC MOTION SICKNESS

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Introduction.

To the general physician the problem of motion sickness typically presents as a request for advice on how to avoid motion sickness when travelling, or during leisure activities such as gliding or sailing. More urgently, a doctor aboard ship in rough weather may be confronted with the need to treat individuals prostrated by repeated vomiting as a result of seasickness. The military doctor may have to deal with the operational problem of airsickness in trainee aircrew, seasickness in sailors, or motion sickness among troops transferred by air, in enclosed army personnel carriers, landing craft or tanks. Finally, because motion sickness may have financial implications by contributing to the failure rate in a costly training programme, medical advice may be sought in order to minimise the economic problem of wastage in training.

Diagnosis.

Motion-induced nausea and vomiting is seldom a diagnostic problem. Those who tend to be susceptible often know from experience the circumstances that induce symptoms. Most of the available prophylactic medication is available without a doctor's prescription and often sufferers will have found a suitable remedy. Individuals may present to their doctor because drugs have failed to prevent symptoms, because of unacceptable side effects, or because the use of drugs would be contra-indicated, for example, when piloting an aircraft.

The doctor should be aware of other diagnostic possibilities, particularly when from the history it would seem that the intensity of provocative stimulus is low, when vomiting persists after cessation of the stimulus, when prophylactic medication has made no difference to the incidence of symptoms, or when there is not a similar degree of susceptibility to provocative motion in other environments.

Vomiting is a presenting symptom in a wide variety of diseases involving different systems. Common causes include gastroenteritis, migraine, excess alcohol consumption, peptic ulceration, drug treatment side effects, and, in young children, acute infections. It is likely that a tendency towards vomiting as a consequence of an underlying disease could be exacerbated by a nauseogenic motion environment.

The suggestion, often by those who have not themselves experienced motion sickness, that anxiety is the principal factor in the nausea and vomiting of motion sickness is not well founded (Mirabile 1990). Nonetheless, anxiety can of itself give rise to symptoms of anorexia, stomach awareness and vomiting. Phobic states can therefore occasionally be confused with motion sickness. Anxiety may however, worsen an already existing susceptibility to motion sickness. It has been observed in airsickness susceptible aircrew that symptoms can be exacerbated by increased work load pressures within the sortie and also by an excessive anxiety to succeed in training (Bagshaw & Stott 1985).

Prophylactic treatment of motion sickness.

In many circumstances in which motion sickness is a problem it is important to instruct individuals in methods to avoid or minimise the stimulus. Such measures may make the difference between success or failure in the effectiveness of prophylactic drugs or may eliminate the need for drug treatment. When exposure to the stimulus is likely to be frequent, an assessment of the likelihood of useful adaptation may give the sufferer some grounds for optimism.

In a review of drugs used in motion sickness (Brand & Perry 1986), the authors point out anomalies between the results of experimental trials which can probably be traced to variations in the interval between medication and exposure to motion, in the duration and type of motion and in such factors as the mode of administration

Table 1. Anti-motion Sickness Drugs

Drug	Route	Adult Dose	Onset	Duration (hr)
Hyoscine	oral	300-600µg	30 min	4
	patch	200µg+20µg/hr	6-8 hr	72
	injection	200µg	15 min	4
Cinnarizine	oral	15-30mg	4 hr	8
Promethazine				
-theoclate	oral	25mg	2 hr	24
-hydrochloride	oral	25mg	2 hr	18
	injection	50mg	15 min	18
Dimenhydrinate	oral	50-100mg	2 hr	8
Cyclizine	oral	50mg	2 hr	12

and dosage level of the drug. The design of most field trials has signally failed to take account of available information about such variables". If this is true for experimentors, how much more so is it for mere practitoners or their self-medicating patients.

Many of the drugs that are used to treat nausea and vomiting from other causes are ineffective in motion sickness. Thus metoclopramide, widely used for vomiting due to gastrointestinal disorders, migraine, anaesthetic agents and cytotoxic drugs is almost certainly ineffective in motion-induced nausea and vomiting (Kohl 1987), although an earlier study (Von Baumgarten 1980) had indicated some benefit. Likewise prochlorperazine, also used in the treatment of nausea and vomiting, has been shown to have negligible benefit in motion sickness (Wood & Graybiel 1968). A recently introduced drug, odansetron, is a $5HT_3$ receptor antagonist which is highly effective in the treatment of nausea and vomiting induced by radiation and chemotherapy, but has no effect on laboratory-induced motion sickness (Stott et al 1989).

The successful use of prophylactic drugs relies upon a knowledge of their time to peak therapeutic activity, their side effects, and duration of action. It is also useful to know something of the variability of response between individuals to a standard dose.

The protection afforded by drugs in motion sickness is far from complete. A survey of over 20,000 passengers aboard ferries (Lawther & Griffin 1988) found that 26% had taken medication. Of these, 11.4% vomited during the voyage compared with only 8.6% of those who took no medication. Of the established drugs, hyoscine (scopolamine USP) has been in use for the longest time and is the most researched drug. Several of the anti-histamine group of drugs also show anti-motion sickness effects. These include dimenhydrinate and its active moiety diphenhydramine, cyclizine, cinnarizine, and promethazine. Recent reports indicate that phenytoin in a dosage that gives anti-epileptic drug levels and also terfenadine in high dosage have beneficial properties in motion sickness prophylaxis. Table 1 lists the currently used drugs, their dose, and time course of action.

Despite its longevity, hyoscine probably remains the most potent single anti-motion sickness drug. It is rapidly absorbed following oral administration and reaches a peak concentration in the blood after ½ to 1 h. Its relatively short half life of about 2.5 h implies that its duration of action is no longer than about 4 h. It is therefore most suitable for short exposures to relatively intense provocative motion stimuli. Side effects from hyoscine are frequent, in particular light headedness, drowsiness and dry mouth. The drug is not recommended for children in whom the therapeutic margin is probably narrower and who are therefore more at risk of serious toxic central effects of restlessness, hallucinations and psychosis. For similar reasons hyoscine is not well tolerated in the elderly. Additionally it should not be used in patients with glaucoma and only used with caution in patients with urinary retention from bladder neck obstruction. Hyoscine has also been shown to impair vigilance and short-term memory (Brazell et al. 1989), possibly a desirable feature in motion sick travellers but not in the training environment.

Oral hyoscine is a less satisfactory drug for longer exposures on account of the need to repeat the dose every four hours. This shortcoming has been addressed by the development of a dermal patch (Scopoderm TTS, Transderm-Scop) which delivers a loading dose of 200 µg hyoscine and 20 µg per hour for up to 72 hours when applied to the post-auricular skin. An effective drug concentration is not reached until 6-8 hours after application of the patch, though this delay can be overcome by simultaneous administration of a single oral dose (Golding et al. 1991). Drug excretion continues for up to 48 hours after removal of the patch probably indicating that the skin beneath the patch continues to provide a reservoir of active drug (Schmitt & Shaw 1981). The principal use of this presentation of hyoscine must therefore be in exposures to a motion stimulus lasting more than 24 hours, as for example on longer sea voyages.

The efficacy of the hyoscine patch has been studied on a number of trials at sea of varying duration. On 12 hour voyages in coastal patrol vessels (Noy et al. 1984), the index of protection, defined as the difference in incidence of seasickness between drug and placebo groups as a percentage of the incidence with placebo, was 62%, and exceeded that for prophylactic dimenhydrinate. In a sea trial lasting 3 days (Attias et al. 1987), an index of protection for the first two days of 74% fell to an insignificant level on the third day. Although similar sea states were encountered on the three days, the incidence of seasickness in the placebo group was much reduced by the third day, presumably as a consequence of natural adaptation. In a sea trial lasting seven days (Van Marion et al. 1985) the hyoscine patch was tested against placebo during the first 3 days of the voyage. A significant benefit was evident during the first 2 days of the trip but not on the third day. In addition, following removal of the patch, those in the active drug group experienced a significantly higher incidence of nausea and vomiting than in the placebo group. It was concluded that the transdermal hyoscine while initially protective against seasickness, had delayed spontaneous adaptation. In the longest duration study (How et al. 1988) the patch was worn for the entire 18 days spent at sea. Therapeutic benefit was confined to the first few days of the voyage, and was greatest among inexperienced sailors and in more severe sea states. Side effects were few. There were three reports of difficulty in accommodation in the drug group, and 13 reports of skin irritation at the site of the patch, similar numbers occurring in both drug and placebo groups.

The rate of drug absorption is supposed to be limited by a membrane within the patch rather than by the underlying skin. However, studies of excretion rates have found between four and six-fold variations in urinary excretion, and other studies have found variability in response between subjects (Pyykko et al. 1985). As with oral hyoscine the side effects of drowsiness, dry mouth may occur. In addition, with the patch there may be impairment of visual accommodation. This affects particularly hypermetropes (Herxheimer 1956), and the risk increases with the length of time for which the patch is applied (Parrott 1986). Even more than with oral hyoscine, the dermal patch is unsuitable for use in children. Hallucinations and extreme agitation were noted in 5 out of 24 children administered either a whole or half scopolamine patch (Gibbons et al 1984), and in a case report of a 6 year old 20 kg child who developed a toxic psychosis, a plasma level at 24 hours after patch application was some 7 times greater than the equivalent adult level (Ewenhauser & Schwarz 1986).

Cinnarizine an antihistamine which also has calcium antagonist properties has in recent years been widely used in the prophylaxis of seasickness (Hargreaves 1980). Adequate drug levels can be maintained with an 8 hourly dosage scheme and the drug is said to be relatively free of the side effect of drowsiness. However, after a single dose, drowsiness tends to occur after about 5 hours rather than at 2 hours when peak plasma level is reached. Likewise, some indices of performance impairment have shown changes only at 5-7 hours after drug administration (Parrott 1986). Two studies of the prophylactic benefit of cinnarizine using similar protocols, found that a dose of 30mg cinnarizine showed no benefit at 2 hours (Pingree 1989), but after 5 hours was as effective as hyoscine 0.6mg taken 2 hours previously (Pingree & Pethybridge 1989). On the basis of these observations cinnarizine in a dose of 30mg should be taken at least 4 hours before exposure to provocative motion and 15 or 30mg taken every 8 hours as required thereafter.

Promethazine is available in two forms either as promethazine hydrochloride or promethazine theoclate. The 24 hour duration of action of the latter may be of advantage in some circumstances, and would allow for a single daily dosage preferably at night so that the peak sedative effect occurs during sleep. Promethazine belongs to the phenothiazine group of drugs, and, in addition to H_1 receptor antagonism, this group possesses considerable anticholinergic activity, so that marked sedation and dry mouth are frequent side effects.

Dimenhydrinate was the first of the antihistamine group of drugs to be shown to have an effect in motion sickness. In a dose of 50mg it is marginally less effective than 0.3mg hyoscine (Wood & Graybiel 1968), but has a longer duration of action. Its sedative side effects may limit its usefulness.

Cyclizine is said to be less sedating than promethazine or dimenhydrinate, though in a dose of 50mg it was found to be somewhat less effective than the same dose of diphenhydramine (Wood & Graybiel 1968). Cyclizine is no longer available in the UK without prescription on account of its potential for abuse. In excess dose the drug can give rise to hallucinations.

The central sympathomimetic drug dexamphetamine has been shown to have an antimotion sickness effect when used alone and to act synergistically when combined with hyoscine, promethazine, or dimenhydrinate (Wood 1990). It also antagonises the soporific and performance decrementing effects of hyoscine. The combination of hyoscine 0.4mg and dexamphetamine 5mg (Scop/dex) is the primary medication used by UK astronauts to combat space sickness and is probably the most effective drug combination available. However, because of its habituating properties and its potential for abuse, dexamphetamine is a controlled drug. In consequence, its general use in motion sickness prophylaxis cannot be justified. A similar alerting effect without the associated risks can be obtained with ephedrine 15-30mg. When used in combination with either hyoscine or promethazine, laboratory studies have indicated an augmented prophylactic effect over either hyoscine or promethazine alone (Wood & Graybiel 1968), though no clear benefit of hyoscine with ephedrine over hyoscine alone was found in a trial at sea (Tokola et al 1984). As with hyoscine, ephedrine may precipitate acute urinary

retention if there is bladder outflow obstruction, an effect in which synergism can be expected with an anti-cholinergic drug.

Recently introduced antihistamine drugs claim benefit over earlier drugs in having less sedating properties. This is ascribed to an inability of the drug to cross the blood-brain barrier. As a general principle drugs that lack any activity within the central nervous system are likely to be ineffective in motion sickness. In support of this principle the H_1 antagonist astemizole is without benefit in motion sickness (Kohl et al. 1987). However, terfenadine, also said to have no central nervous system penetration, has been shown to possess anti-motion properties following a single dose of 200mg (Kohl et al. 1991). (This dose exceeds the manufacturers recommended maximum of 120mg). Whether at this dose some drug does cross the blood-brain barrier, or whether the principle must be revised, is not yet clear. However, drowsiness is an occasional side effect of terfenadine.

A recent study on the prophylactic use of phenytoin (Chellen et al. 1990) found a tenfold increase in tolerance to cross-coupled stimulation when taken in a dosage (1-1.4 grams over 24 hours) that produces anti-epileptic blood levels. This represents an increase in efficacy by a factor of four over any other single drug in current use. Though no side effects were reported in the seven subjects involved in this study, possible side effects include nausea and vomiting, mental confusion, dizziness, headache and muscle tremor. It may be significant that in excess dose this drug produces signs and symptoms referable to the vestibular system and cerebellum, in particular, ataxia, dysarthria and nystagmus. Cerebellar atrophy may follow larger overdosage. It may therefore be that in the therapeutic dose range the cerebellum is preferentially sensitive to the neuropharmacological effects of phenytoin. At the present time, however, its use in motion sickness prophylaxis remains experimental.

Root ginger has long been used in oriental traditional medicine as a remedy for nausea and vomiting. One laboratory study (Mowrey & Clayson 1982) has found powdered ginger root to be of prophylactic benefit in motion sickness. Two further laboratory studies (Stott et al. 1984, Wood 1990) that used an end point of moderate nausea failed to show any increase in tolerance to cross-coupled stimuli. However, further evidence of a beneficial effect comes from a controlled trial at sea (Grøntved et al. 1988) in which 80 naval cadets were given either 1g powdered ginger root or placebo, and were asked to score the symptoms of nausea, vertigo, vomiting, and cold sweating, every hour for the next four hours. The number of individuals who reported no motion sickness symptoms was equally divided between the two groups. Among the 61% of subjects with symptoms of seasickness there was no significant difference between ginger and placebo for the symptoms of nausea or vertigo. However, the frequency of vomiting and the severity of cold sweating were significantly reduced in the ginger group. Of the seven subjects who vomited in the placebo group five vomited two or more times, whereas the five subjects who vomited after taking ginger did so only once. When vomiting powdered ginger, perhaps once is quite enough! These findings are consistent with the view that ginger may act locally to ameliorate the gastro-intestinal consequences of motion sickness without altering the underlying susceptibility.

Dietary factors.

Reviews of the various dietary measures proposed in the treatment of motion sickness make amusing reading (Reason & Brand 1975), but their diversity is some indication of their lack of value. No effect on the incidence of motion sickness has been found in relation to time of day or the time of meals (Manning & Stewart 1949). It is generally agreed that vomiting is less unpleasant when the stomach contains something to vomit and that lack of food can itself be dispiriting. Equally, it would seem sensible to avoid dietary excesses.

Motion sick aircrew have observed that their susceptibility to airsickness is increased by even moderate amounts of alcohol in the previous 24 hours, and this effect of alcohol has also been observed on tolerance to cross-coupled stimulation during desensitisation treatment (Bagshaw & Stott 1985). For those taking anti-motion sickness drugs, the instructions on the packet warn against the consumption of alcohol on account of increased sedation.

Adaptation

For many individuals whose exposure to provocative motion is infrequent, advice on how to minimise the stimulus of motion sickness coupled with the use of an appropriate prophylactic drug offers the best chance of avoiding the condition. For others, such as week-end sailors and those who fly for pleasure in light aircraft or gliders, repeated use of drugs may be undesirable or, in the case of solo flying, prohibited. Though repeated exposure should lead to adaptation, the frequency of exposure to the provocative motion is often insufficient to promote an adequate degree of adaptation.

Often the dispiriting effect of repeatedly becoming motion sick during what would otherwise be a challenging and pleasurable leisure activity, leads an individual to take up golf, bird watching, or some other pedestrian interest. Before concurring with this course of action, the advice, for example, to a motion sick gliding student might be to undertake a more continuous period of training in which the intensity or duration of provocation could be graded. It could be anticipated that having once gained some degree of adaptation, its re-acquisition after an interval away from the stimulus might be a more accelerated process.

Formal desensitisation treatment, on account of its cost and the amount of time

involved, would not be justified for this group of individuals. It is however possible that a self-administered ground-based desensitisation course might offer some degree of pre-adaptation to someone particularly enthusiastic to succeed. It is simple to generate a cross-coupled stimulus using a rotating office chair kept in continuous motion with the feet, the occupant making roll or pitch head movements. However, rotational velocity is poorly controlled by this method and it is not difficult for a susceptible individual to make himself rapidly unwell. Another readily available device is a conventional swing. This device offers two types of sensory conflict, both of which are features of aviation. There is an alternating acceleration stimulus that has both vertical and horizontal components, whose frequency of oscillation can, by appropriate choice of rope length, be brought into the nauseogenic range (4m for 0.25 Hz, 2.25m for 0.33 Hz). In addition, because in a freely swinging pendulum the resultant force vector is always aligned with the rope, the subject experiences the alternating angular rotation stimulus without a corresponding change in the direction of the resultant vector, the same conflict that is engendered by coordinated turns in aircraft. To render the swing stimulus comparable to that in the air, the subject would require to sit sideways so that angular oscillation of the swing would act in roll rather than in pitch.

A further device (Aerotrim^{R}), found in some sports centres, consists of three concentric metal rings, the outer one fixed to the ground, the inner two rings free to rotate about axes at right angles to each other, with the innermost ring, about 2m in diameter, configured to carry a standing subject. By appropriate movement of his body, the subject who is tethered by the feet and restrained by a metal hoop at waist level, can rotate about two axes simultaneously. The nauseogenicity of this device is not severe, but probably derives from its capacity to produce cross-coupling of a somewhat uncontrolled nature, and unfamiliar changes in the dynamic force environment.

It has to be admitted that none of these expedients designed to promote adaptation has received experimental validation. In fact, in a controlled study, repeated exposure to swings used in the conventional fashion failed to protect against airsickness (Gibson et al. 1943).

The treatment of established motion-induced vomiting.

Once individuals have reached the stage of vomiting, drugs are less effective and have to be given parenterally and in higher dosage. Even in the period of malaise that precedes vomiting there is a marked delay in gastric emptying time (Wood et al 1987) and the absorption of orally administered drugs will therefore be impaired.

Having vomited, there is often a temporary improvement in well-being, but with continued stimulus malaise worsens and vomiting recurs. Some individuals may become totally prostrated and appear very ill. When this occurs aboard ship, sufferers should be moved to the part of the ship where the provocative motion is at a minimum, and should lie down. Adequate fluid intake is not possible until vomiting has stopped, and to this end anti-motion sickness drugs may be required by injection. Severe motion sickness is frequent aboard life rafts in rough seas and could be a significant factor affecting survival. Although drugs given orally are of no value, buccal absorption of hyoscine, administered as the proprietary dispersible form (Kwells), has been shown to be as effective as generic hyoscine tablets administered orally (Golding et al 1991). An effective blood level can be obtained within 30 minutes.

The transcutaneous route of administration is of no immediate value in individuals already suffering symptoms of motion sickness on account of the 6-8 hour time interval before therapeutic levels of the drug are reached. To achieve a more rapid onset of activity drugs must be given by injection. In an uncontrolled case study of airsickness in 47 individuals who undertook repeated flights involving parabolic manoeuvres, the intramuscular administration of diphenhydramine 50mg, promethazine 25mg and 50mg, and hyoscine 0.5mg were compared for their beneficial effects on reversing severe symptoms of nausea (Graybiel & Lackner 1987). Both hyoscine 0.5mg and promethazine 50mg showed beneficial effects in 72 and 78% of subjects respectively, whereas little or no benefit was obtained from the lower dose of promethazine or from diphenhydramine. The therapeutic efficacy of hyoscine when given intramuscularly is about 6 times that of the same dose given orally (Mirakhur 1978). This difference is a consequence of the first-pass metabolism of orally administered hyoscine in the liver. Thus 0.5mg hyoscine given intramuscularly would be equivalent to about five times the standard oral dose. Such a dose runs the risk of toxic side effects. For this reason the intramuscular injection of promethazine 50mg is to be preferred as the treatment for established nausea and vomiting due to motion sickness.

The treatment of motion sickness in the armed services

The military doctor, perhaps more than his civilian counterpart, has to balance the loss of operational effectiveness produced by motion sickness with the possible detrimental effect on performance from the side effects of drugs, and, if neither alternative is acceptable, to consider the need to suspend or re-role affected individuals.

Motion sickness may have significant economic consequences when it contributes to the failure rate in an expensive training programme. One solution might be to develop some selection test to eliminate those individuals likely to encounter

problems with motion sickness severe enough to result in withdrawal from training. A significant correlation (r=.41) has been shown between failure in flying training and objectively rated tolerance to a sequence of 10 head movements while rotating at 90 deg.s^{-1} (Ambler & Guedry 1966). For practical purposes, however, such a test lacks sufficient selectivity. An alternative might be to eliminate aircrew who are airsick during the introductory sorties of training. This approach fails to take account of the natural adaptive process. On the other hand, it may require 20 - 30 sorties before spontaneous adaptation can be judged to have failed, by which time, given the cost of flying hours, a considerable investment has been made in the individual. The precise policy adopted depends on the availability of suitable applicants for the training programme and on financial resources. Because of the high cost of training, a good case can be made for establishing some form of treatment programme for airsickness. Various schemes have been described (Dowd 1973, Dobie 1974, Grabiel & Knepton 1978, Levy et al. 1981, Kemmler 1983, Ceresia 1983, Giles & Lochridge 1985, Bagshaw & Stott 1985). All involve adaptation to ground-based motion stimuli that are nauseogenic. Some use biofeedback and relaxation techniques. They vary in the extent to which remedial flying is incorporated into the programme. Detailed comparison of results is complicated by differing criteria of success, but it is possible to reduce by 80 to 90% the number of trainee aircrew who would otherwise be eliminated from training on account of persistent airsickness.

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Benson, A. J. (Royal Air Force, Farnborough (England). Inst. of Aviation Medicine.)
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Leger, A. , Sandor, P. , Dalalhaye, R. P. (Centre d'Essais en Vol, Bretigny-sur-Orge (France). Lab. de Medecine Aerospatiale.)
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Magee, L. E. , Kantor, L. , Sweeney, D. M. C. (Defence and Civil Inst. of Environmental Medicine, Downsview (Ontario).)
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NASA-TM-89412; A-87034; NAS 1.15:89412

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Morales, Rogelio, Jr. (Air Force Inst. of Tech., Wright-Patterson AFB, OH.)
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14. Abstract

In aerospace activities, motion sickness, specifically airsickness, continues to be a problem during flying training and in regular operations for aircrew and passengers (e.g. paratroops). Simulator sickness can degrade the effectiveness of simulator training and space sickness reduces the efficiency of astronauts. Seasickness is also of aeromedical concern in so far as it affects aircrew operating from ships and the survivability of ditched aviators.

The Lecture Series has been designed, primarily, to aid practising Flight Surgeons in the performance of their primary care duties. It should also be of interest to others who wish to obtain an overview of recent advances in the understanding of the aetiology and treatment of motion sickness.

The topics to be covered in the 12 lectures are:

1. Clinical features of motion sickness
2. Operational significance of motion sickness
3. Aetiology and neurophysiological mechanisms
4. Physical characteristics of provocative motion
5. Special features of air, space, sea and simulator sickness
6. Selection and assessment of susceptibility
7. Prophylaxis and management.

The Lecture Series will conclude with a Round Table Discussion.

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