UNIVERSITY OF SOUTHAMPTON

Visual acuity, eye movements, motion sickness and the illusion of motion, with optokinetic stimuli.

by

Nicholas Andrew Webb

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Human Factors Research Unit,
Institute of Sound and Vibration Research,
FACULTY OF ENGINEERING AND APPLIED SCIENCE

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ABSTRACT

FACULTY OF ENGINEERING AND APPLIED SCIENCE
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VISUAL ACUITY, EYE MOVEMENTS, THE ILLUSION OF MOTION AND MOTION SICKNESS WITH OPTOKINETIC STIMULI

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Motion sickness and the illusion of self-motion (vection) can be induced by the use of an optokinetic drum (a black and white striped cylinder which rotates around a seated, stationary subject). It has been suggested that vection is a cause of motion sickness in optokinetic drums because the illusion of self-motion, in the absence of real motion, may be a form of sensory conflict. Alternative theories have suggested that motion sickness may arise from eye movements. Motion sickness has been reduced with fixation, where subjects focused on a stationary object in front of the moving stripes, preventing eye movements.

This thesis investigated the correlations between motion sickness, vection, eye movements and visual acuity. Six experiments were conducted. The first compared motion sickness and vection in a real and a virtual reality simulation of an optokinetic drum (with the same field of view). There was slightly greater motion sickness in the real drum, but no difference in vection. Vection and motion sickness scores did not correlate within conditions, indicating that vection may not be the main cause of motion sickness. It was found that visual acuity was significantly correlated with motion sickness, in both conditions. Subjects with poor acuity reported increased symptoms.

In the second experiment subjects viewed a normal optokinetic stimulus on the virtual reality display and the same optokinetic stimulus with a stationary cross in front of the moving stripes (fixation). Motion sickness was significantly reduced with fixation but vection was unchanged. Visual acuity was correlated with motion sickness without fixation, as before, but was not correlated with motion sickness with fixation. The fourth experiment found that motion sickness could be produced with a single moving dot, tracked by the eyes of subjects, presented on the virtual reality display. Motion sickness symptoms were not significantly different with a single or multiple dot display. Vection was significantly higher with multiple dots, where peripheral visual stimulation was increased.

A fifth experiment found that motion sickness was significantly higher when subjects viewed a standard optokinetic drum without vision correction, compared to viewing with vision correction. Visual acuity and contrast sensitivity to higher spatial frequencies were found to be correlated with motion sickness, indicating that a lack of sensitivity at high spatial frequencies, rather than at a wide range of low and high spatial frequencies, were associated with motion sickness. A final experiment measured the slow phase of nystagmus with and without vision correction. It was found that the slow phase velocity was significantly lower with poorer sensitivity to high spatial frequencies.

The results from the experimental work suggest that vection and motion sickness are distinct phenomena, since they can be manipulated independently, and were not correlated in any of the experimental conditions. Motion sickness was not significantly different with a single dot (foveal) or multiple dot display (foveal and peripheral), and was reduced by fixation (where there was no foveal image slip, but large peripheral image slip). It is concluded that foveal image slip may be an influence on motion sickness via an unknown mechanism. Vection is controlled mainly by peripheral image motion and is unrelated to eye movements. Contrast sensitivity to high spatial frequencies influenced the amount of foveal image slip occurring, which in turn influenced motion sickness. A model of the interactions between visual acuity, vection, foveal and peripheral image slip, motion sickness and the slow phase velocity of nystagmus has been developed.

Acknowledgements

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My parents and grandmother also deserve a special thankyou, for their encouragement during the course of my study.

Dedication

I would like to dedicate this thesis to my Grandfather, Arthur Horseman, who was unable to finish his own PhD due to the 2nd world war, but who would have no doubt found all the errors and weak links in my theories in a matter of minutes!

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Glossary of terms

Circular-vection: The illusion of rotation.

Contrast sensitivity: Ability to detect a difference between two spatially adjacent values of luminance. Usually measured with a display on which the luminance varies sinusoidally across the field of view. The contrast can be increased until a difference is perceived. Contrast sensitivity is measured at various spatial frequencies from low to high, where the frequency of the sinusoidal variation in luminance increases.

Electro-oculography (EOG): In each eye, a potential difference exists between the cornea and the ocular-fundus (corneal-retinal potential, 10-30mV; the cornea being positive). The potential difference sets up an electrical field in the tissues surrounding the eye. As the eye rotates the field vector rotates correspondingly. Eye movements can be detected by placing electrodes on the skin in the area around the eyes. This is known as electro-oculography. The resolution is about 1° of visual angle and the technique is suitable for horizontal eye movements within a range of approximately 30°.

Fast phase of nystagmus: A saccadic eye movement used to re-set the eye position after the eye has followed a moving object, or during rotation of the head or visual surround.

Field of view: The visual extent of the view of a subject. Field of view is expressed as a horizontal and vertical visual angle. A restricted field of view occurs when using some virtual reality displays which have small screens, or by wearing blinkers.

Fixation: The act of looking at a stationary object. In the experimental work presented here, fixation was achieved by placing a stationary cross in front of a moving optokinetic background.

Fovea: The central area of the retina. The fovea has the highest visual resolution of the retina at all spatial frequencies. It is usually 30-100 minutes of visual angle. The fovea is the area of the retinal in which there are only cones.

angular eye velocity. Retinal slip can be referred to as foveal slip: the velocity of images on the fovea, or peripheral slip: the velocity of images on the peripheral retina.

Saccade: A saccade is an eye movement of short duration, typically 50 ms. The purpose of a saccade is usually to bring an object of interest into the foveal region of the retina.

Slow phase of optokinetic nystagmus: A type of pursuit eye movement. It occurs when there is motion of the head, or visual surround, in order to maintain high visual acuity by minimising the relative velocity of the object on the retina.

Smooth pursuit: Smooth pursuit is a tracking eye movement in response to motion of an object. The purpose of smooth pursuit is to maintain the object on the fovea. Smooth pursuit is more accurate in response to predicable target motion, than to random motion.

Spatial frequency: The frequency at which luminance varies with distance, in a contrast sensitivity test. There is usually a sinusoidal or square wave variation of luminance.

Vection: The illusion of self-motion. See also 'circular vection' and 'linear vection'.

Vestibulo-ocular reflex (VOR): The vestibulo-ocular reflex serves to stabilise vision during rotational head motion. Eye movements of an equal angular velocity occur in the opposite direction to the head motion in order to maintain high visual acuity.

Virtual reality: A visual display in which images are presented to subjects by the use of two screens in front of the eyes. Virtual reality can be head-coupled, so that motion of the head is sensed and used to update the visual scene.

Visual acuity: The resolution of fine detail, at high contrast. A normal person can usually resolve a visual angle of 1 minute. A number of visual acuity tests exist.

Gain: The gain of nystagmus is defined as the angular velocity of the eye, divided by the angular velocity of the moving target. The gain of the vestibulo-ocular reflex is defined as the angular velocity of the eye divided by the angular velocity of the head.

IRIS: The IRIS is a device produced by the Skalar Medical Company which can measure eye movements by the detection of reflected infra-red radiation from the cornea of the eye. The accuracy of the IRIS system is approximately 1 min of visual angle.

Landolt 'broken ring' test: A standard measurement of visual acuity. The Landolt ring target is usually a 'C' shaped black ring on a white background. The gap in the ring can appear in four different positions (up, down, left or right). The gap in the ring and its thickness are equal to one fifth of the ring diameter. The rings (and gaps) get smaller until a subject can no longer locate the gap in the ring.

Linear vection: The illusion of self-motion in a straight line (usually in the fore and aft direction). For example linear vection occurs during visual simulation of forward motion in a car or aeroplane simulation, or on a train when a nearby train moves.

Motion sickness: The signs and symptoms experienced during exposure to certain types of motion, motion simulators or motion of the visual scene.

Nystagmus: A movement of the eyes that alternates in direction.

Optokinetic drum: A cylinder which is painted with black and white stripes internally. The optokinetic drum rotates around a stationary, seated subject.

Optokinetic nystagmus: A smooth pursuit eye movement, followed by a rapid return saccade. Nystagmus occurs during exposure to rotating visual surrounds or during constant velocity rotation about a vertical axis.

Peripheral retina: The area of the retina outside of the fovea. The peripheral retina has a lower image resolution than the fovea and is responsive mainly to lower spatial frequencies.

Retinal slip: The relative angular velocity at which an image is moving on the retina. The retinal slip velocity is the difference between the angular target velocity and the

Chapter 1. Introduction

Motion sickness is an unpleasant condition, which many people experience at some point in their life. The most common forms of motion sickness occur during the course of travel on ships, small boats, cars, aeroplanes and buses. This 'travel sickness' is widely experienced and takes the form of a number of common symptoms, such as yawning, cold sweating, increased salivation, drowsiness, dizziness, headaches, stomach awareness, nausea and vomiting.

Motion sickness during sea travel has been noted for many centuries. In more recent times motion sickness symptoms have been noted even without movement of the person experiencing the sickness. Sickness has been noted during wide screen cinema presentations, when playing computer games, during exposure to a motion simulator (such as a flight simulator) or more recently during virtual reality simulations. Visually-induced motion sickness, sometimes known as 'simulator sickness', can pose real problems in the use and development of motion simulators or virtual reality for training purposes. For example, virtual reality has the potential to be used for training pilots or for use in the medical training of surgeons. When motion sickness occurs it can prevent some individuals from participating in the training or it may limit the length of time for which training can occur. Perhaps more seriously, the individuals undergoing training may develop strategies to avoid experiencing the symptoms, such as minimising head movements, which may then have an adverse effect on their performance when they move to the real world task (i.e. poor transfer of training).

Visually induced motion sickness has been studied for the past 30 years with the use of an optokinetic drum. This is a cylinder painted internally with black and white vertical stripes, which rotate around stationary, seated subjects who watch the stripes. Motion sickness is common upon exposure to an optokinetic drum. Optokinetic drums are used because they are simple to manufacture and operate, and can be altered to discover more about visually-induced motion sickness, such as whether the speed of the visual stimulus influences the symptoms experienced (or whether eye movements, made in response to the moving stripes, influence motion sickness).

Subjects also tend to experience an illusion of motion known as 'vection' when viewing an optokinetic drum. Vection has been studied in its own right as an interesting phenomenon. Vection has often been linked to motion sickness in the literature, as a cause of motion sickness, although it has not been proven statistically (e.g. Hettinger *et al.,* 1990). Eye movements have also been suggested as a possible cause for motion sickness, with higher frequency eye movements hypothesised to increase motion sickness in response to an optokinetic drum (e.g. Ebenholtz *et al.*, 1994).

The aim of this thesis was to investigate visually-induced motion sickness by using an optokinetic drum and a virtual reality display. In each of six experiments reported in this thesis, vection and motion sickness were investigated to see whether they were correlated. Visual acuity data for subjects were recorded in each experiment to discover whether the ability of a subject to see fine detail at high contrast would affect their motion sickness symptoms. Additionally, eye movements were recorded in the 2nd, 4th and 6th experiments in order to investigate correlations between visual acuity, eye movements and motion sickness.

In the first experiment, a virtual reality simulation of an optokinetic drum was compared with a standard optokinetic drum to see whether the results found in virtual reality were comparable with past results from a normal optokinetic drum. The second experiment investigated whether suppressing eye movements could influence motion sickness and vection (the illusion of self-motion). A third experiment investigated whether artificially blurring the stripes could affect motion sickness or vection. A fourth experiment compared motion sickness and vection when only central (foveal) vision was stimulated (with a single moving dot) or when central and peripheral vision were stimulated (with multiple moving dots). A fifth experiment investigated whether motion sickness was different when subjects watched the optokinetic drum with or without their vision correction (e.g. their spectacles or contact lenses). The sixth and final experiment investigated whether there was any difference in the eye movements of subjects when they viewed the optokinetic drum with and without their vision correction.

A review of the literature is presented before the experimental work. The literature review encompasses areas such as visual acuity, contrast sensitivity, eye movements, the vestibulo-ocular reflex, vection and motion sickness. The review concentrates mainly on research which has been conducted using optokinetic drums.

Chapter 2. Literature review

2.1 Introduction

The purpose of this chapter is to present information relating to optokinetic motion sickness and related subjects such as the illusion of self-motion (vection) and eye movements, in particular optokinetic nystagmus and the vestibulo-ocular reflex. Some background material is presented on the structure of the optical and vestibular systems in order to provide the necessary detail to understand eye movements in response to movement of the surrounding visual scene or movement of the subject. A general discussion on motion sickness and the theories used to explain it is presented but the main body of work consists of motion sickness in response to optokinetic stimuli.

2.1.1 A simple introduction to the anatomy of the eye – the retina

The average human eye is approximately 22 to 24 mm in diameter. The cornea at the front of the eye is made of clear, blood vessel free, tissue and it is through the cornea that refracted light enters the eye. The curvature of the cornea is responsible for approximately 60% of the initial refraction of the light entering the eye, with the lens providing the remaining refraction.

At the back of the eyeball (see Figure 2.1) lies the retina which is comprised of hundreds of millions of nerves distributed into nine layers. The retina consists of "rods" and "cones". These are two different types of light receptors with different properties. Rods are by far the more numerous of the two receptors. There are estimated to be approximately 20 million rod receptors per eye. The rods are black and white receptors, they have no colour sensitivity and function best in low illumination, reaching maximum sensitivity after being in darkness for approximately 30 minutes. They are less responsive to fine detail than the cones which are mixed in with the rods in varying densities and which work best in high illumination.

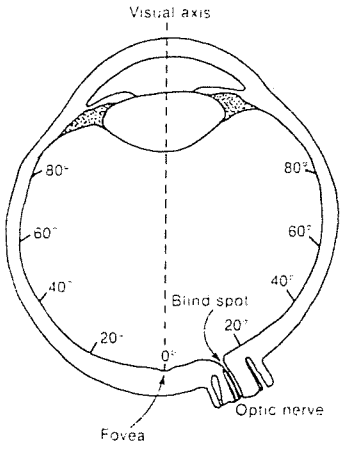

Figure 2.1. Cross section of the eye.

A central area of the retina known as the macula contain only cones, which are responsible for colour vision and the discrimination of fine detail – higher spatial frequencies. The macula is located in the central retina directly behind the pupil. The tiny, central portion of the macula is referred to as the fovea, where cone density is highest and is responsible for our high acuity vision. Figure 2.2 (Ditchburn *et al*, 1973) shows the retina of a left eye as seen through an ophthalmoscope. The spacing of the cones on the retina decreases with distance from the fovea. Figure 2.3 shows how the inter-cone spacing changes with retinal eccentricity. The resolution of the retina is related to the inter-cone spacing, hence the fovea can resolve higher spatial frequencies than the peripheral retina (Polyak, 1941).

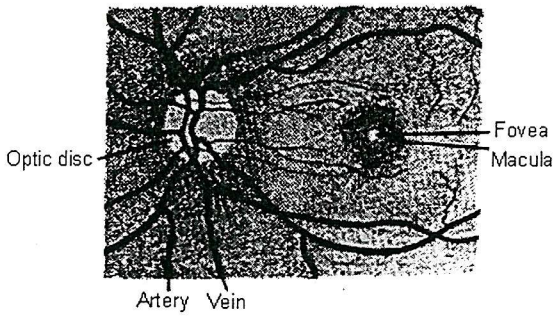

Figure 2.2. The retina seen through an opthalmoscope (from Ditchburn, 1973)

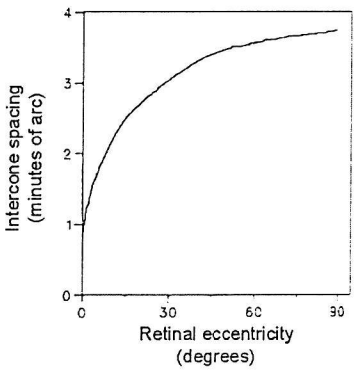

Figure 2.3. Cone spacing against retinal eccentricity (Polyak, 1941).

2.1.2 Visual acuity

Visual acuity is described as the ability of the eye to discriminate fine details (e.g. small print). It has traditionally been expressed as a ratio, such as the familiar 20:20. Twenty-twenty vision is defined as the ability of the eye to discriminate 1 minute of visual angle, which is approximately the limit of human performance. The two numbers in the ratio refer to the measuring distance. If a subject could only resolve 2 minutes of visual angle measured they would be said to have 20:40 vision. The denominator refers to the distance at which a person with normal vision could resolve the same target. The smaller the denominator the better the visual acuity. In practice the subject is not moved, but is presented with rows of increasingly small targets which they attempt to identify until a mistake is made. The visual acuity is often expressed as a decimal or as a fraction, where 20:20 is equal to 100%.

2.1.2.1 Limits on Visual Acuity

The visual acuity of a subject can be limited by the optical characteristics of the eye, such as corneal or lens imperfections, which can be corrected to a certain extent by the introduction of glasses or contact lenses. Neural limits are imposed by the characteristics of the retina, the density of the cones and rods which vary on different parts of the retina, and the inherently noisy signal pathways.

2.1.2.2 Landolt "broken ring" test.

This is a traditional and effective method to measure acuity (used in the experiments presented in chapter 4 onwards. It relies on the ability of the subject to identify the orientation of a "broken ring", otherwise known as a "broken c" (Olzak *et al.,* 1986). The gap can appear in one of four orientations – up, down, left or right. The width of the gap is equal to one fifth of the diameter of the ring. A Landolt broken ring test consists of lines of rings with different orientations of the gap. The subject reads from left to right the position of the gaps in the rings. Figure 2.4 shows a typical example as used in the experiments presented later (from the Keystone visual skills test). The subject would, for example, read the top line as 'left, top, bottom, right' and then read successive lines until a mistake is made. The last correctly completed line is taken as the subject's score for the test and will correspond to a certain acuity ratio (e.g. the bottom line on Figure 2.4 corresponds to 20:15 vision, as measured at the specified test distance). The Landolt acuity measurements can be made at various distances, usually at a near and far point. The two distances in the experimental work presented in later chapters were 0.4m (near) and 4m (far).

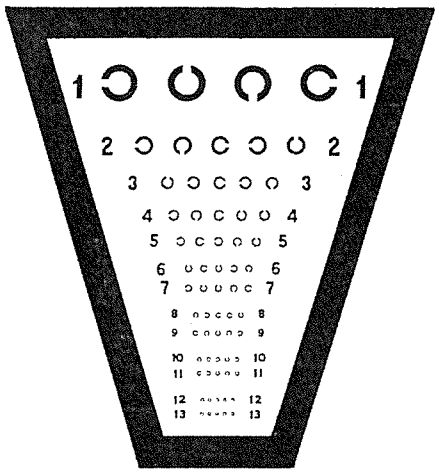

Figure 2.4. Landolt 'broken ring' test of visual acuity.

2.1.3 Contrast Sensitivity

Measurements of visual acuity normally record only the subject's sensitivity to high spatial frequencies at high contrast. A subject with 20:20 vision can resolve 1 min of

visual angle, which is equivalent to one of the highest measured spatial frequencies in a contrast sensitivity test: 60 cycles per degree. Often, particularly with the onset of cataracts in elderly people, vision can be impaired without affecting the responses to higher spatial frequencies. It is possible to lose sensitivity to lower spatial frequencies without losing sensitivity to high frequencies. Somebody with loss of sensitivity at low spatial frequencies may report feelings that their vision is not quite right and a loss of night vision. Tests have been developed to measure the visual response at a wider range of spatial frequencies, in order to gain a clearer picture of visual performance at a wide range of spatial frequencies, rather than at only the high frequencies. A contrast sensitivity test known as the "Arden Test" was used in the fifth and sixth experiments presented in this thesis. In this test, a card is slowly removed from a holder. Each card has a sinusoidal variation across the card of grey to black. The contrast increases down the length of the card. The subject indicates the point at which they can see the difference in contrast (i.e. the card no longer looks grey all over). At the point at which the card is stopped, a number is read off the edge of the card to indicate the threshold of detection of that spatial frequency. Several cards of different spatial frequencies are used in the Arden test. An example is shown in Figure 2.5. A contrast sensitivity test includes a built in test of visual acuity, because eventually a subject will be unable to resolve a spatial frequency even at full contrast. In this instance the visual acuity of the subject has been found (e.g. the limit at which they can resolve fine detail at high contrast).

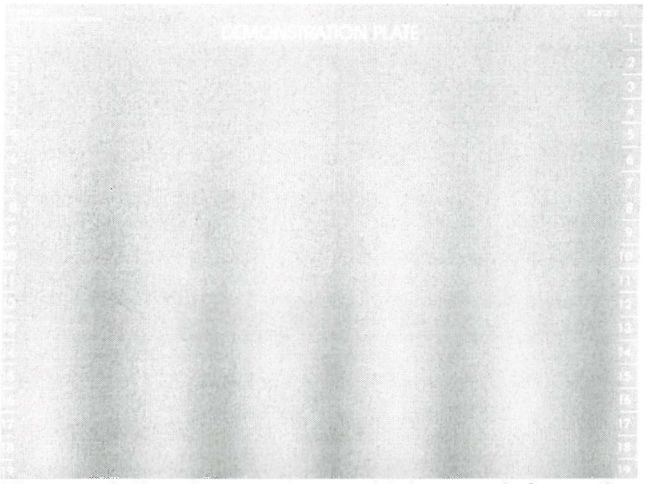

Figure 2.5. Contrast sensitivity card from the Arden test.

Marmor *et al.* (1987) studied the effect of introducing visual lenses in order to blur deliberately the image seen by a subject. A range of lenses were used in order to reduce the visual acuity of a subject to 20:20 (if their initial acuity was better), 20:32, 20:50 and 20:100. The contrast sensitivity of subjects was then measured whilst still wearing the blurring lenses at spatial frequencies of 1.5, 3.0, 6.0, 12.0 and 18.0 cycles per degree. The authors found that contrast sensitivity was impaired at a wide range of frequencies even with modest refractive degradation (e.g. blurring to 20:20

from higher acuities). The loss of contrast sensitivity when blurring vision from 20:20 to 20:100 is not just confined to the width of letters between 20:20 and 20:100 but also decreased sensitivity to lower spatial frequencies. It is concluded that the test of contrast sensitivity should be used alongside standard Snellen acuity tests for clinical purposes. The two tests together offer a greater level of information about a subject's visual capabilities and may help to explain functional disabilities such as trouble seeing at dusk or reading in low light. Contrast sensitivity measurements should be made whenever lenses are used to reduce visual acuity experimentally, in order to fully appreciate the overall effect on vision that is occurring.

2.2 Summary of the vestibular system

The labyrinth, embedded in the temporal bone on each side of the head includes the semi-circular canals, the otoliths (utricle and saccule) and the organ of hearing, the cochlea. The vestibular system consists of the non-auditory components of the labyrinth; the otoliths which are sensitive to gravity, tilt and linear acceleration of the head and the three semi-circular canals which are sensitive to rotations of the head in three axes (Howard, 1986).

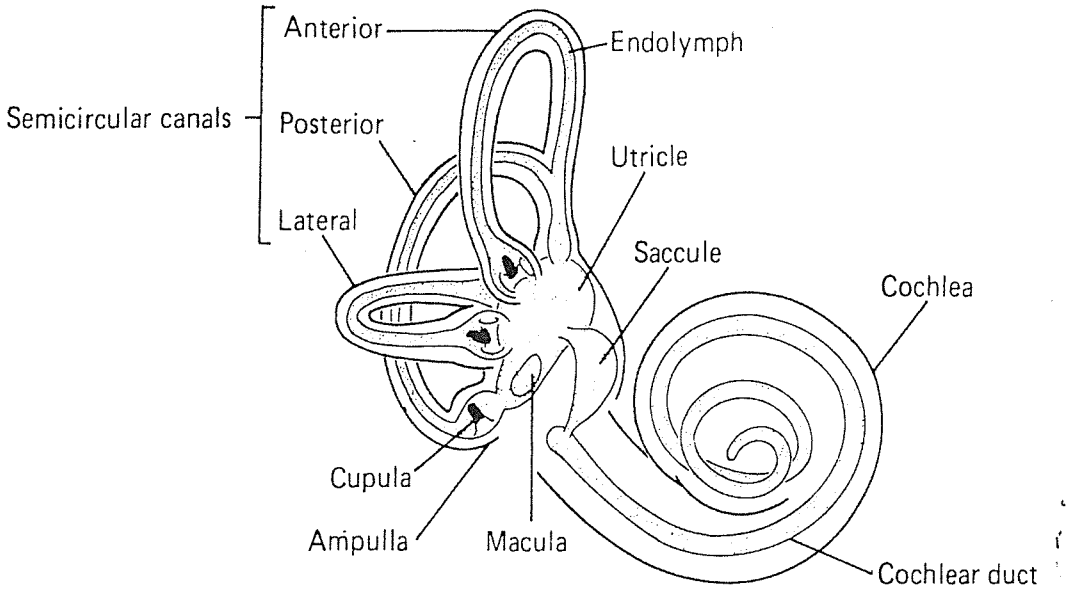

Figure 2.6. The vestibular labyrinth showing the cupula.

The function of the vestibular system is to sense motion of the head. The signals derived from the canals are used to generate appropriate eye movements in response (vestibulo-ocular reflex), control posture, balance, and perceptions of motion and orientation.

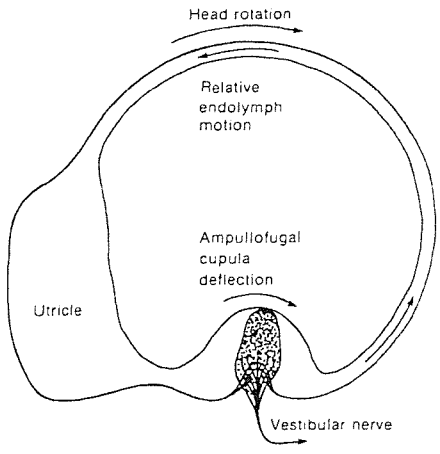

Figure 2.7. Rotation of one semi-circular canal.

There are three semicircular canals within the labyrinth on each side of the head. They are approximately at right angles to each other, in order to be sensitive to acceleration of the three rotational axes of the head. Figure 2.6 shows the structure of the three canals. Filling each canal is a fluid known as endolymph which, when the head moves, lags behind the motion due to its inertia and hence 'flows' relative to the walls of the canal in the opposite direction to that in which the head is turning. Figure 2.7 shows the rotation of one canal. The fluid flow acts on a membrane – the cupula, which forms a seal between the two halves of the canal flow (Melvill Jones, 1993; Robinson, 1981). This pressure, or deflection of the cupula, bends tiny hair cells located at the base of the cupula which causes a signal to be sent to the vestibular nucleus via the eighth cranial nerve.

2.2.1 Dynamics of the semi-circular canals

The dynamics of the semi-circular canals can be modelled quite simply. The force on the cupula can be modelled with the equation for a torsion pendulum. It is then a straightforward matter to identify the nature of the response of the equation to varying frequencies and displacements of the head. The derivation below is from Howard, 1986.

If **H** is the moment of inertia of the endolymph fluid plus cupula and a person rotates the head with an angular acceleration α in the plane of one semi-circular canal then the force acting on the cupula is α**H**. This force displaces the endolymph and cupula by an angle θ. The force is approximately described by the torsion pendulum equation:

$$\alpha H = k\theta + r\frac{d\theta}{dt} + H\frac{d^2\theta}{dt^2}$$

In the above equation, k is the stiffness (position dependent), r is the coefficient of viscous resistance (velocity dependent) and H is the moment of inertia (mass dependent resistance) of the cupula and endolymph. This equation is suitable for simple analysis of the dynamics of the semi-circular canals. More complicated models have been proposed to take into account additional properties of the system, the details of which are unnecessary here.

The human vestibular canal is only about 0.3mm in mean diameter, hence the viscous resistance is high even for moderate velocities and the mass of the endolymph is small. The elasticity of the cupula is also small compared with the viscous resistance. The first and third terms of the pendulum equation are hence very small in comparison to the second term and can be ignored for moderate to high frequencies (in which head movements usually occur) hence:

$$\alpha H = \frac{rd\theta}{dt}$$

It can be concluded that $d\theta/dt$ is proportional to α, in other words the angular velocity of the cupula is proportional to the acceleration of the head. By integrating both sides of the equation it can be seen that the angular displacement of the cupula is proportional to the velocity of the head. This holds for the normal range of voluntary head displacements and velocities. At very low frequencies of head rotation the viscous resistance of the endolymph fluid becomes small compared to the inertia resistance. In this instance the third term of the differential equation becomes dominant and the response becomes proportional to the acceleration of the head.

Constant angular rotation of a subject (i.e. zero acceleration) leads to a decrease in the response from the vestibular system after the initial acceleration period until there is no response. The endolymph fluid decreases its inertial force on the cupula due to the lack of acceleration and the natural elasticity of the cupula causes it to resume its neutral position. The time constant for cupula deflection in humans is thought to be approximately 5 to 7 seconds (Robinson, 1981). Attempts to measure the time constant by measuring the persistence of an oculo-motor response are complicated by the additional influence of a neural response on eye movements. The oculo-motor response with repeated exposure tends to reduce to about 7 seconds which may give the best estimate for the mechanical component of the time constant (the cupula deflection).

In the case of constant angular rotation, a sudden deceleration of a subject will cause the cupula to deflect again, and induce a response of the oculo-motor system (nystagmus) in the opposite direction to that which occurred when the subject was accelerated. This can be disorientating and can induce motion sickness.

2.3 Eye movements

2.3.1 Introduction

The oculomotor system can be analysed more easily than most other movement control systems because it can be broken down functionally into smaller subsystems (Robinson, 1981) which can be analysed individually. This section looks at the main types of eye movements including the purposes of eye movements, the vestibulo-ocular reflex, saccades, smooth pursuit and nystagmus.

2.3.2 Purposes of eye movements

Species have evolved into two categories: animals with and animals without foveas. Afoveate (without a fovea) animals have evolved systems to minimise the amount of image slip occurring on the retina as a whole via an optokinetic (eye movements in response to a moving visual stimulus) and a vestibulo-ocular reflex response (eye movements in response to head movement). The position of the image on the retina in afoveate animals is of lesser importance than in animals with a fovea, the main purpose of eye movements being that of image stabilisation (Robinson, 1981). Animals with foveas have similar eye movements designed to stabilise images, but also add eye movements which are designed to bring objects of interest to the fovea and to hold them there. These are the saccadic (high velocity, short duration jump eye movements from one point to another), pursuit tracking (following a moving object), and vergence (bringing objects at certain distances onto the fovea of each eye) oculomotor subsystems.

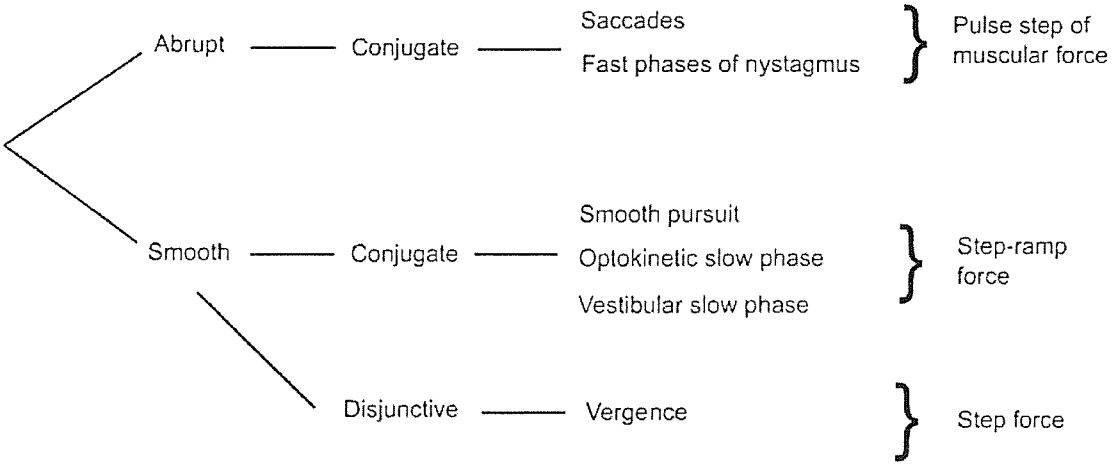

Figure 2.8. Different categories of eye movements (Robinson, 1981).

Eye movements can be classified into two categories: abrupt and smooth. Abrupt eye movements include saccades and the fast phases of nystagmus. Smooth eye movements include pursuit tracking, the slow phase of nystagmus (vestibular or optokinetic) and vergence. Figure 2.8 shows the different categories of eye movements (from Robinson, 1981).

This review deals mainly with optokinetic nystagmus eye movements. Optokinetic nystagmus can be considered to be a combination of a smooth pursuit eye movement (known as the slow phase) followed by a rapid return saccade (fast

phase) to reset the eye position. The components of nystagmus can be shown to have similar properties to slow phases and saccades, measured on their own. This is shown in more detail in Sections 2.3.5 and 2.3.6.

2.3.3 Vestibulo-ocular reflex

The vestibulo-ocular reflex is a fundamental response which enables the eyes to remain space stabilised during head movements and is the most important of the image stabilisation subsystems. Without the vestibulo-ocular reflex it would be impossible to move about and see clearly at the same time. Images would slip across the retina during head movements. Robinson (1981) refers to the case of a physician who lost all labyrinthine function after streptomycin poisoning and was unable to read signs or recognise people in the street without stopping and standing still in order to minimise head movements.

The major contributors to the vestibulo-ocular reflex during rotation of the head are the semi-circular canals. Under certain conditions, for example off-axis rotation (Viirre *et al.,* 1986) or a static tilt (Robinson, 1981) the otoliths can have an effect on the reflex. For the purposes of this review, the term 'vestibulo-ocular reflex' will be used to refer to the canal-ocular reflex and the otolith response will be ignored.

When the head moves, the tiny hair receptors at the base of the cupula send a velocity proportional signal (see Section 2.2.1) to the vestibular nucleus and on to the oculomotor nuclei in order to drive the eyes in an equal and opposite direction to the head movement. The purpose of this response is to reduce slipping of the image on the retina and hence to maintain high visual acuity. The vestibulo-ocular reflex is able to respond to head movements with minimal delay. Eye movements can occur within 10-20 ms from the initial head movement (Virre *et al.,* 1998). The vestibulo-ocular reflex can be quantified in terms of gain and phase, where the gain is the velocity of the eyes in response to head movements divided by the velocity of the head. For sinusoidal stimuli the perfect vestibulo-ocular reflex would have a gain of 1.0 (the eye and head velocities being equal) and a phase of 180° (the eyes should always move in the exact opposite direction to the head motion in order to stabilise vision).

There is an important difference between the vestibulo-ocular reflex gain measured in darkness and measured in light. In dark conditions there is no contribution of

visually-based image stabilisation and the measured gain of normal subjects is of the order of 0.7 at frequencies between 0.05 Hz and 1.0 Hz. At higher frequencies of motion, between 1 and 7 Hz, the gain is closer to 1.0 (Robinson, 1976; Shelhamer, 1994). Attention also affects the gain measured in dark, with higher gains recorded if the subject is made to answer simple arithmetic tests to maintain concentration (Robinson, 1976). Measured under light conditions, the vestibulo-ocular reflex gain will approach 1.0 at most frequencies because of the additional inputs to eye movement control generated by the motion of the visual image, particularly at lower frequencies.

The vestibulo-ocular reflex and optokinetic reflex (optokinetic nystagmus) serve the same purpose - to stabilise images on the retina. They work together in order to stabilise images under many conditions. In the example of acceleration to a constant velocity of rotation, the vestibular response decays after approximately 25 seconds whilst the optokinetic response increases during a similar time period. The optokinetic system takes over the image stabilising task once there is no further contribution from the vestibular system.

2.3.4 Vestibular ocular reflex adaptation

The vestibulo-ocular reflex has a tremendous ability to adapt its response in order to maintain stable vision under changing conditions. An example of this can be changing the relative motion of the visual scene in response to head movements by wearing magnifying spectacles (Demer *et al.,* 1989). The vestibulo-ocular reflex can make gain changes in response to such magnification and regain stable vision. If the spectacles are removed the vestibulo-ocular reflex gain must then re-adapt to its old settings.

Demer *et al.* (1989) studied vestibulo-ocular reflex adaptation with magnifying spectacles. They measured vestibulo-ocular reflex gain by oscillating subjects sinusoidally in darkness at 0.1 Hz and measuring head and eye velocity. Subjects were then exposed to rotations in a lighted room, whilst wearing magnifying spectacles (x2, x4 or x6) and looking at a remote video display. It was found that the initial vestibulo-ocular reflex gain in darkness averaged about 0.7 and that vestibulo-ocular reflex gain increased after viewing through the magnifying spectacles by 7 - 46 %. It was found that significantly more adaptation occurred if the unmagnified

peripheral vision was occluded during the magnified period compared to when it was visible, which raised questions about whether peripheral vision contributed to part of the adaptation process. The above experiment indicated that gain changes occurred but were insufficient to completely adapt to the extreme change of magnification. This was the gain change measured in darkness, with the additional effect of visual input the vestibulo-ocular reflex may have fully compensated for the magnification. The gain change measured in darkness shows that a central change in the response has occurred. Collewijn *et al.* (1983) showed that small changes of gain, of the order of 5-10% could be completely adapted to within approximately 30 minutes. It is thought that more than one different vestibulo-ocular reflex gain can be stored which can be used as the appropriate situation arises (Gauthier & Robinson, 1975). The action of putting on a pair of glasses may immediately switch the vestibulo-ocular reflex gain to the correct gain which has been stored for that situation.

2.3.4.1 Vestibulo-ocular reflex - central vs. peripheral vision

Experiments have been conducted in order to investigate the part of the visual stimulus responsible for driving the adaptation of the vestibulo-ocular reflex. Lisberger *et al.* (1984) investigated vestibulo-ocular reflex adaptation to a stimulus presented to central vision only. Using monkeys, they found that 50-70% of the vestibulo-ocular reflex adaptation to a full visual field could be obtained using only a single spot of light presented to the central visual field.

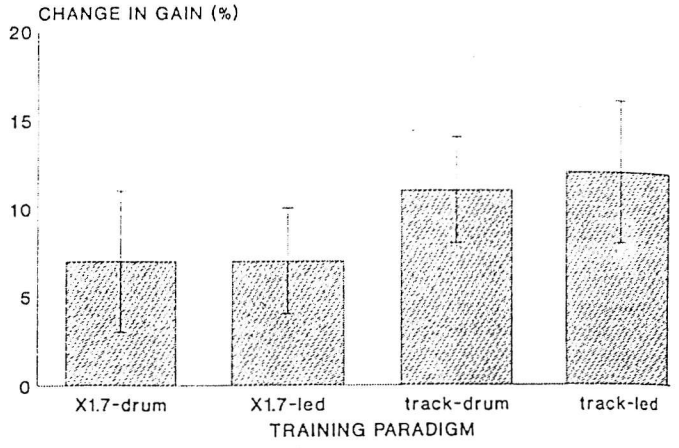

Figure 2.9. Change in vestibulo-ocular reflex gain whilst viewing an optokinetic drum or spot of light (l.e.d.). Subjects were moved in the x1.7 condition but stationary in the other conditions.

Shelhamer *et al.* (1994) found that vestibulo-ocular reflex adaptation occurred without any head motion of subjects. They used a sinusoidally oscillating optokinetic drum at 0.2 Hz in one condition, and found that there was a change in the mean vestibulo-ocular reflex gain from 1.02 before exposure, to 1.13 after exposure. The changes were statistically significant at the $p<0.001$ level, and similar to the gain increases

encountered in conditions where the subject was moved on a rotating chair. They also found no difference in levels of gain adaptation when using a small spot of light as a stimulus instead of a full field of view optokinetic drum condition. This is in contrast to Demer *et al.* (1989) who found that there was less adaptation occurring when the periphery was occluded and Lisberger *et al.* (1984) who found that adaptation was 30% lower when the periphery was occluded in monkeys.

Shelhamer *et al.* (1994) attempted to clarify whether it is the amount of 'retinal slip' (blurring) present on the peripheral retina during motion of the head or visual scene, or a combination of the retinal slip and eye movements that are responsible. They discovered that during a fixation condition where no eye movements occurred, but the visual scene moved behind a stationary spot on which subjects focused, vestibulo-ocular reflex gain changes still occurred but to a lesser extent than those found when the eyes were free to move. They conclude that vestibulo-ocular reflex adaptation is based on a combination of eye movements, retinal slip on the fovea, and a smaller contribution from motion detected on the periphery. The finding that the gain changed without subject motion may indicate that retinal slip alone can be sufficient to drive the vestibulo-ocular reflex adaptation process. Image slip occurs in optokinetic drums because the velocity of eye movements rarely matches that of the drum (see Section 2.3.7.1). Prolonged image slip occurring in the optokinetic drum may give the impression that vestibulo-ocular reflex adaptation needs to occur because image slip over a long period of time (i.e. more than a few seconds) may perhaps only be associated with a visual–vestibular mismatch.

Melvill Jones *et al.* (1979) investigated whether retinal slip was the driving force for vestibulo-ocular reflex adaptation. They studied the vestibulo-ocular reflex response to left-right reversed vision under normal lighting conditions and under strobe light conditions in which the strobe time was short enough to minimise retinal slip detection (4 Hz flash rate - 3μsec flash duration). It was found that the vestibulo-ocular reflex gain (measured at 1/6 Hz frequency, sinusoidal oscillation, peak amplitude of 60°/second) reduced, compared to that measured in the dark before exposure to the reversing goggles, in both the normal and strobed conditions. Further research into the vestibulo-ocular reflex gain adaptation at higher frequencies (Melvill Jones *et al.*, 1981) revealed that the gain was reduced again at the low frequencies in both the normal and strobed conditions. At a frequency of 1.75 Hz the average gain was attenuated by 30% and at 3 Hz by 25% in normal light after a day of

exposure. In the strobe condition no measurable change in the vestibulo-ocular reflex gain was found at 1.75 Hz and 3.0 Hz. The authors conclude that the vector of the image slip on the retina is of importance as the error signal for the adaptation of the vestibulo-ocular reflex but that it is not the only error signal. It may be possible for the brain to interpret the discontinuous sequence of images on the retina as a method for adapting the vestibulo-ocular reflex. This would be predicted to be successful only when the sequence of images were moving in a meaningful way, which would only occur at lower velocities of head movement when a target object on which the subject was focusing would move by small distance on the retina between strobe flashes. At higher velocities the image sequences would appear on the retina in a complex manner which must be difficult to interpret in a meaningful way. This may explain why there was no measurable adaptation in the 1.75 and 3.0 Hz measurement conditions.

It may be the case that the image slip on the fovea during eye movements is the most useful source of information for driving the adaptation process. The motion on the periphery of the retina in a real-life situation, such as tracking a moving object, would have motion cues from different depths of field, whereas the fovea would contain only the object that it is desired to be tracked. This idea is partly confirmed by the results presented above by Shelhamer *et al.* (1994) who found similar levels of adaptation with full or restricted fields and less adaptation with fixation, but they did not draw a definite conclusion from their work on this point.

2.3.5 Saccadic eye movements

Saccades are short duration, high velocity eye movements which serve to rapidly change the position of the eye, usually in order to bring an object of interest onto the fovea or to reset the eye to its primary position in the case of nystagmus. Saccadic eye movements are usually around 50 ms in duration (within a range of 20–120 ms) and with a velocity of 20-600°/second (Hallett, 1986). Afoveate animals use saccades involuntarily simply to reset the position of the eye during vestibular or optokinetic nystagmus. For foveate animals the saccade became more useful as a means of directing the fovea to areas of interest after which other oculomotor subsystems, for example smooth pursuit, developed to help to keep the fovea on the object of interest. An example of a saccade in response to a step stimulus is shown in Figure 2.10.

Saccades can be voluntary (Hallett, 1978) or can at other times occur automatically without any conscious effort, such as during the normal process of reading or during the resetting of the eye position in optokinetic nystagmus. Fast phases of nystagmus are thought to be structurally very similar to saccades and show similar amplitude and velocity characteristics (Ron *et al.*, 1972). Fast phases of nystagmus which are artificially induced, for example by caloric irrigation, show longer durations. It is also shown by (Ron *et al.*, 1972) that alertness influences the duration of saccades with longer durations found for lower levels of alertness.

The size of the smallest saccade is about 3 minutes arc (Haddad *et al.*, 1973) and the largest possible is about 90°. The acceleration of the eye is large – as high as 40,000°/second^2 for a 10° amplitude saccade. Peak velocity is reached roughly one third of the way through a saccade, followed by gradual deceleration. The eye comes quickly to rest at the end of a saccade in order to allow the eye to focus on the new scene. The eye is slowed down by the momentary activation of the antagonist muscles (Robinson, 1981).

Saccades normally fall short of a target, even for small saccades, by roughly 10% of the amplitude of the target jump. A corrective saccade normally makes up the remaining 10% of the distance required to reach the target and occurs with a shorter latency than the initial saccade (Prablanc, 1974). The corrective saccade has a latency of about 130 ms (considerably shorter than the primary saccade latency). It is thought that the saccadic system can sense either before, or just after, the first saccade, that it is too small and initiate the corrective saccade with reduced latency.

Prablanc *et al.* (1974) investigated the occurrence of saccades in response to the sudden illumination of a light source. They found that corrective saccades did not occur if the light was extinguished during the period of the first saccade. Corrective saccades with the usual short latency occur if the light is re-illuminated before the end of the initial saccade, but only if the light is within about 4° of its original position. Saccades with a longer latency occur if a light is illuminated further than 4° from the original light position. It seems that when the target is moved by greater than 4° the full saccadic process must start again because the position of the target differs significantly from that expected by the saccadic system. During repetitive, predictable target jumps the saccadic system is able to move with minimal, or zero, latency to the target (Robinson, 1981).

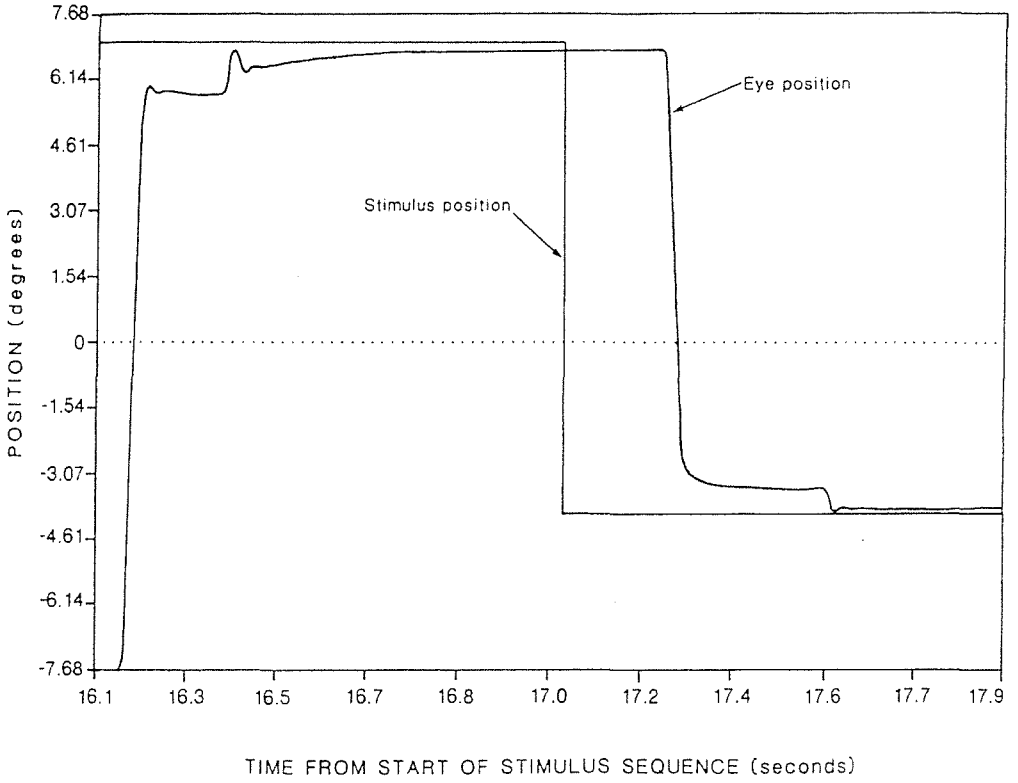

Figure 2.10. A saccade in response to a step stimulus. From Hallett (1986). Note the large primary and smaller corrective saccades.

2.3.5.1 Occurrence of saccades

Saccades occur at about 3 times per second (173,000 per 16 hour waking day). The latency of a saccade following a sudden jump of a target to one side is about 0.2 – 0.23 seconds. It is thought that the typical delay of 215 ms consists of about 55 ms lost in the retina, 25 ms lost in the pre-motor circuits and eye muscles and around 135 ms for central processing and decision making (Robinson, 1972).

2.3.6 Smooth pursuit eye movements

Pursuit eye movements are smooth tracking eye movements which are designed to keep the target object on the fovea and hence to maintain high acuity whilst tracking. The durations of smooth pursuit eye movements are usually more than 200 ms, making them easy to distinguish from saccades, and achieve maximum velocities of 30-100°/second. Smooth pursuit is normally associated with tracking a small target on the fovea whilst ignoring the motion of the background on the peripheral retina.

The main stimulus for pursuit appears to be the velocity of image slip on the retina but the pursuit system can also respond to the position of the target with respect to the fovea. For instance, a small after-image placed near the fovea which a subject is told to look at results in a smooth pursuit movement. The after-image, of course, always moves ahead of the fovea thus not providing any retinal slip velocity information (Robinson, 1965).

2.3.6.1 Structure of smooth pursuit

One simple way to study the response of the smooth pursuit system has been to study the response to a 'ramp stimulus', where a previously stationary target commences a horizontal movement with a constant velocity. This type of stimulus is used because one can study the initial reaction of the eye and the steady response. Line 1 in Figure 2.11 shows an average of 14 responses to a 10°/second ramp stimulus (from Robinson, 1965). The eye movement begins after a delay of 125 ms. Under a rate of muscle force of 21.7 g/sec the eye accelerates at a mean value of 60°/sec^2 to reach a velocity of 6.1°/sec and a displacement of 0.38°. This process takes 112 ms before it is interrupted by a saccade, the purpose of which is to rapidly catch up with the stimulus. The saccade occurs 237 ms after the initial stimulus motion and has an amplitude of 1.24°. An error of 0.7° still remains between the eye and target. The eye now leaves the saccade at a smooth pursuit velocity of 12.2°/sec which it maintains for the next 200 ms. The error between eye and target is

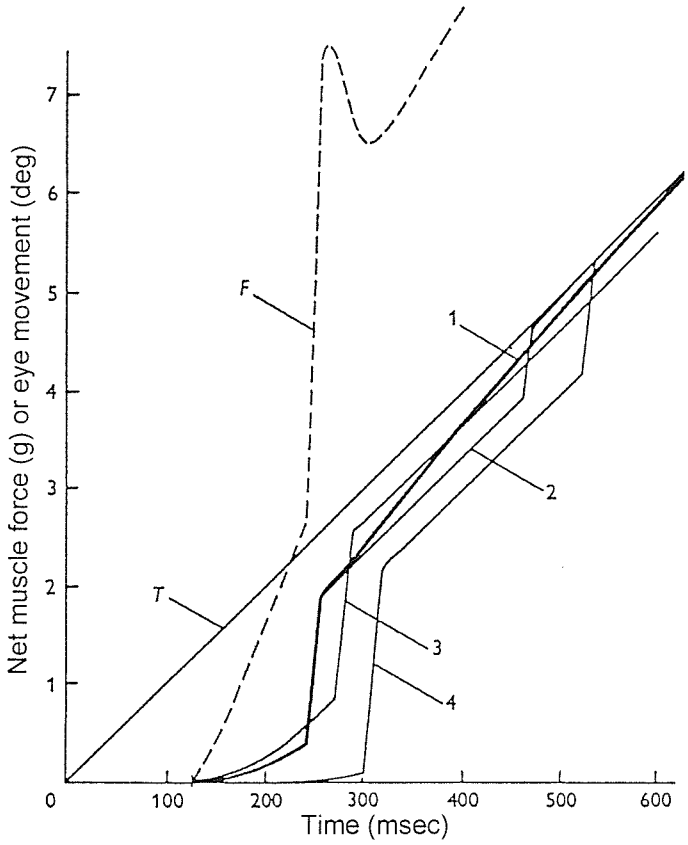

Figure 2.11. Different types of pursuit response to a ramp stimulus. From Robinson, 1965. 1-4 are different responses. F is the net muscle force.

now almost zero and the eye maintains velocity at 10°/sec in order to match the target velocity from this point onwards.

Figure 2.11 also shows other responses which occur less frequently: response 2 (occurring 31% of the time) requires a second corrective saccade because the initial smooth pursuit does not occur at a velocity above the target velocity. In response 3 the saccade occurs later, after the initial smooth pursuit and response 4 (occurring 10% of the time) shows no early smooth component and a large saccade occurring quite late in the process. In Figure 2.11 'F' is also shown, which is the eye muscle force.

Knowler *et al.* (1978) studied smooth pursuit responses with varying target velocities. They found that the velocity of the target was almost perfectly matched at low velocities (around 2°/second) but that the velocity did not quite match the velocity of the target at velocities of 5°/second or more. The eye tended to lag behind the target at a velocity of about 95-97% of the target for the 5°/second stimulus. Variations of the size and shape of the target and the background (either black or striped) made no significant difference on the pursuit velocities observed. Practice was shown to increase the velocity and resulted in a more perfect matching of the target and eye movement and hence to decrease retinal slip. Knowler *et al.* (1978) proposed that the ocular system may need a small amount of residual foveal slip in order to help maintain the pursuit movement. Matching the velocity perfectly (a gain of 1.0) would eliminate foveal slip, which is the necessary error signal used to drive corrections to the smooth pursuit eye movement.

Michael *et al.* (1966) measured the pursuit response to stimuli of varying predictability. They generated signals of varying bandwidth from Gaussian random noise centred about the desired test frequency. The most predictable stimulus is a sine wave, which effectively has a bandwidth of zero, but as bandwidth increases the signal becomes less predictable. Five bandwidths of noise were produced of 0.05, 0.10, 0.20, 0.50 and 1.00 Hz and these were centred around the test frequencies which were 0.3, 0.7, 1.0 and 1.5 Hz. The root mean-squared values of the stimulus amplitudes were kept constant within a small error range to help avoid any influence amplitude may have on predictability. The accuracy of the eye movements was quantified by taking the mean phase shift of the eye movement response (measured by electro-oculography) and comparing it with the stimulus signal. It was found that at

the lowest frequency of 0.3 Hz there was no significant difference in the phase shift with increasing bandwidth. This may have been due to the ease at which tracking could be performed at such a low frequency. At all the higher frequencies an effect of decreasing accuracy with increasing bandwidth was noted. These findings were statistically significant at the $p<0.001$ level. The authors conclude that predictability of stimuli may help maintain high visual acuity in day to day life during head movements, which are generally predictable from previous experience.

2.3.7 Nystagmus

Nystagmus is an eye movement response in reaction to motion of the visual surround or to vestibular input during rotation of the head. It consists of a slow phase which tries to minimise retinal slip velocity by matching the speed of the moving surround (similar to smooth pursuit), and a fast phase (saccade) to reset the eye position. It has developed to maintain stability of vision during self-rotation and consists of vestibular nystagmus (driven by higher frequency head rotations) and optokinetic nystagmus (which is driven by the continuous visual input during head movements rotations or constant body rotation). The two systems are complementary in normal life because they are both stimulated by head movements under self-rotation (Robinson, 1981). During constant rotation, the signal from the vestibular system returns to zero and the optokinetic system dominates the control of eye movements completely. Constant rotation of the visual surround is not encountered during everyday life, but experiments whereby the visual surround is rotated around a stationary subject can reveal useful information about the functioning of the optokinetic system, with applications in moving image systems such as film projections, motion simulators and virtual reality. The system used to move the visual surround has traditionally been the optokinetic drum (a black and white striped drum which rotates around a seated subject and excites all of the visual field.)

2.3.7.1 Optokinetic nystagmus

The pursuit component of optokinetic nystagmus in man is difficult to distinguish from smooth pursuit. Muratore *et al.* (1979) asked subjects to pursue a small spot of light which moved at 50°/sec against a dark background and against a striped background. The spot moved in a sawtooth fashion: moving smoothly before jumping

back to its starting position. After two minutes the stimulus was stopped and in 8 out of 11 subjects an after-nystagmus was observed which was similar to that observed during exposure to a full optokinetic drum rotation. The after-nystagmus consisted of a nystagmus which occurred in the original direction and faded with time.

Afoveate animals (without a fovea) such as the rabbit need a large portion of the retina to be stimulated in order to elicit an optokinetic response (Dubois *et al.,* 1979). Figure 2.12 shows the time course of the onset of optokinetic nystagmus in man, monkey and rabbit. The difference in the initial rise in the velocity of the slow phase in the different species is explained by the varying influence of the fovea in the different species. Animals with foveas: the monkey and human, have a smooth pursuit system which is able to change eye velocity very quickly and aid tracking. It is thought that the pursuit system supplements the optokinetic system in order to boost

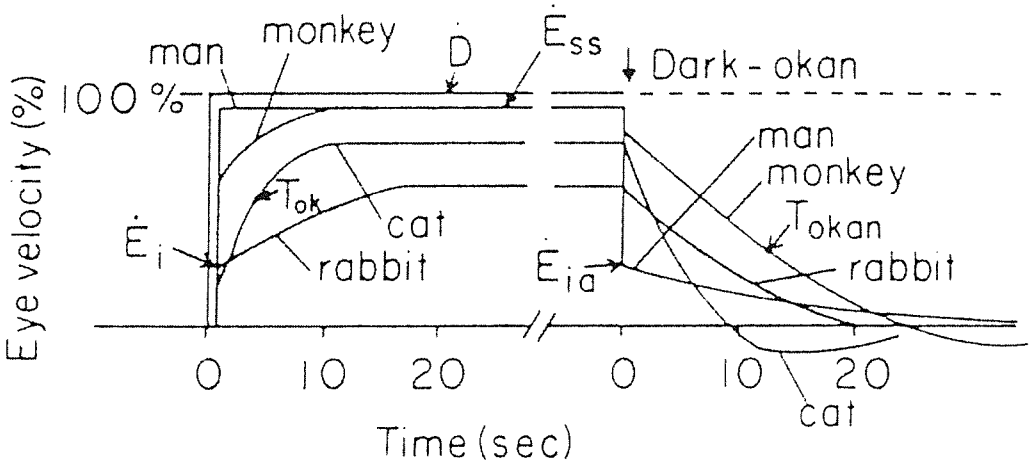

Figure 2.12. Time course of nystagmus and after-nystagmus in man, monkey, cat and rabbit. In response to a suddenly illuminated optokinetic stimulus from Robinson, 1981.

the velocity of slow phase eye movements to help match the target velocity (Van Die *et al.,* 1986). It has also been discovered that patients with a deficient pursuit response but a preserved optokinetic response exhibit the same slow build up of optokinetic nystagmus as the rabbit (Yee *et al.,* 1979). The dominance of the pursuit system in humans is further revealed by asking subjects to fixate on a stationary point, for example a cross in the centre of the visual field (Brandt *et al.,* 1973). Nystagmus is completely suppressed during fixation, indicating that the pursuit system is dominating despite the majority of the retina being excited by the moving stimulus.

2.3.7.2 Optokinetic nystagmus - peripheral and foveal stimulation

Van Die *et al.* (1986) studied eye movements in response to optokinetic stimuli presented to the central and peripheral retina. The stimulation of the central or peripheral retina was achieved by masking the unwanted part of the visual scene. The masking systems tracked the horizontal eye movements and thus prevented stationary edges from suppressing eye movements (i.e. there were no fixation points). In addition, scotopic viewing conditions were used whereby a very low level of illumination was used so that the central retina would not be stimulated. Three patients with a unilateral central retinal scotoma were also studied (they had very poor central vision in one eye).

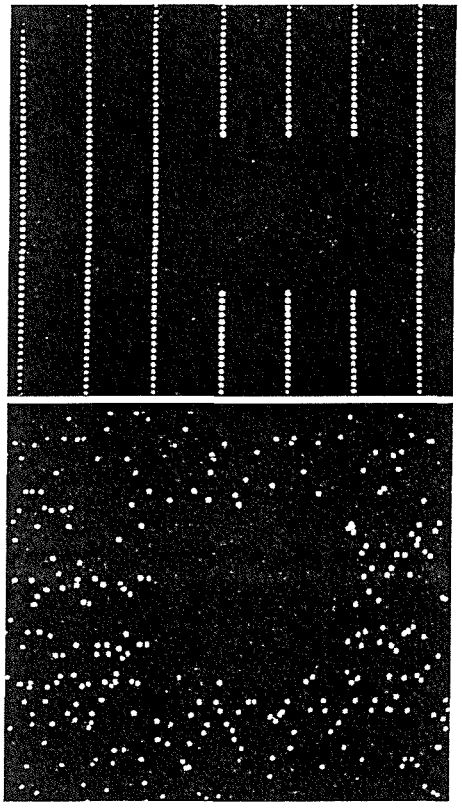

Figure 2.13. Stimulation of the visual field – minus the fovea; area – Cheng *et al.* (1975). With random dots and lines of dots.

In each of the conditions above it was found that the velocity of the slow phase of nystagmus was lower when the central retina was not stimulated. This was the case whether masks were used, scotopic vs. photopic illumination, or when subjects with a central retinal scotoma in one eye viewed the stimulus with the affected eye compared with the normal eye.

The velocity of the slow phase eye movements was expressed in the form of gain: the velocity of the eye divided by the velocity of the drum. In all the cases measured gain was near 1.0 at very low velocities and fell with increasing speed of the stimulus.

Van Die *et al.* (1986) concluded that that the peripheral and central visual systems can produce compensatory eye movements (nystagmus) in response to the visual motion, but that there is a decrease in gain in those conditions when the central retina is not involved. They point out that a previous finding of Hood (1967) that the gain is predominantly controlled by peripheral vision and that there was a steady decrease in gain when the periphery is excluded could have been due to the stationary blinkers that were used in the experiment which would prevent eye movements occurring over a range of more than a few degrees, and may also have acted as a fixation

target. The same foveal dominance for optokinetic nystagmus was found by Cheng *et al.* (1975) who found that the gain of nystagmus reduced as a visual stimulus was moved an increasing distance from the fovea (see Figure 2.13). Dubois *et al.* (1979) also found that blocking the central retina reduced the nystagmus gain more than by deleting peripheral vision.

Murasugi *et al.* (1986) studied the effect that occluding various parts of the visual scene had on the gain of the slow phase of nystagmus. They predicted that the presence of edges in the visual field are enough to reduce nystagmus gain even if they can be made to move with the eyes, which may have been a phenomenon responsible for the reduction in gain found when blocking the fovea as in the above experiments (Van Die *et al.*, 1986 and Cheng *et al.*, 1975). With a display which was 60° wide moving at 30°/second it was found that nystagmus gain was reduced by placing a pair of stationary vertical bars close together. The gain of the slow phase of nystagmus increased as the bars were moved further apart symmetrically about the centre of the display. In a second experiment optokinetic nystagmus in response to a moving field of dots was recorded with a full field condition, a condition with a central horizontal band deleted, the whole display deleted with the exception of a 15° central rectangle, a 15° frame, 15° separated vertical lines and a 15° central rectangle deleted (see Figure 2.14).

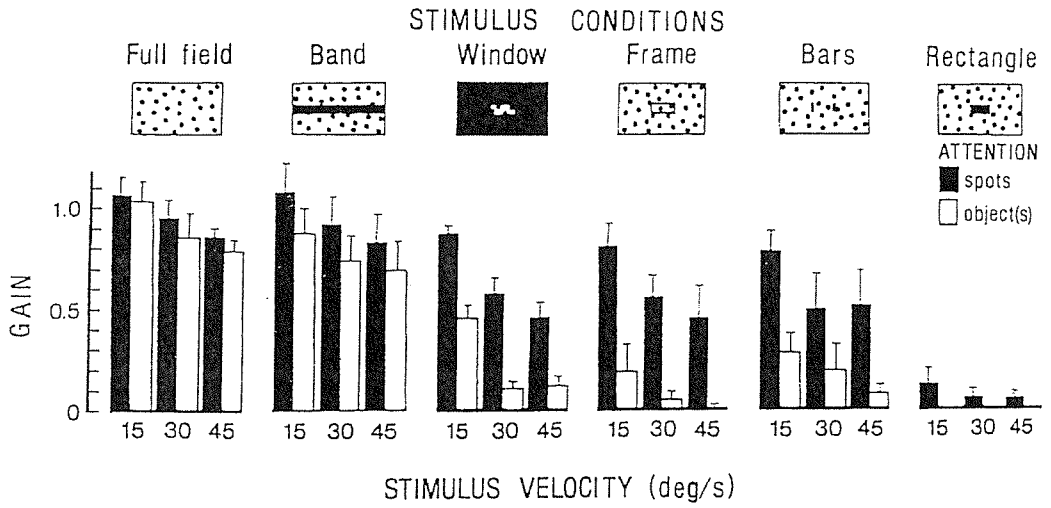

Figure 2.14. Gain of eye movements in response to different visual conditions. Murasugi *et al.* (1986). The black and white bars show the response when the subject was instructed to look at the object (e.g. the black horizontal band) or at the moving dots.

It was found that deleting the central band did not have a significant effect on the slow phase gain compared with full field stimulation, but that by the addition of stationary vertical edges in the rectangle condition (Figure 2.14) that nystagmus was almost completely abolished. The other conditions in which there were stationary vertical edges but central motion was present also the reduced the slow phase gain but did not abolish the nystagmus. The authors conclude that a combination of deleting central vision and the presence of stationary edges are necessary to abolish optokinetic nystagmus.

Howard *et al.* (1984) found that nystagmus gain was reduced at target velocities over about 30°/second by a central band similar to the one used by (Murasugi *et al.,* 1986) above, with no stationary edges. In a second experiment the relative contrast of the peripheral and central displays were controlled by Howard *et al.* (1984) in order to test whether the relative visibility of the stimulus in the central and peripheral retina was responsible. They point out that the statement that the fovea is more important in driving nystagmus is meaningless if it is purely because the periphery cannot see the stimulus. It was found that even with the relative visibility of the stimuli matched, the gain of nystagmus was reduced by deletion of a central band. A third experiment blurred the edges of a restricted visual display and compared the optokinetic nystagmus generated with a wider angle display. It was found that there was no difference in the nystagmus gain generated when the edges were blurred and did not allow for fixation and suppression of nystagmus to occur. Howard *et al.* (1984) concluded that by blurring the edges of smaller displays, nystagmus with a similar gain could be generated. This allowed for the possibility that optokinetic research could be carried out using small screen monitors and other limited visual field displays so long as the edges were blurred.

It appears that is not just the presence of stationary edges which is responsible for the reduction in gain of nystagmus. The results presented by Van Die *et al.* (1986), for scotopic viewing conditions and subjects with central retinal scotoma, showed that the gain of optokinetic nystagmus was reduced in these two conditions where no stationary or moving edges could have been visible.

The studies by Murasugi *et al.* (1986) and Howard *et al.* (1984) showed the strong effect stationary edges have on reducing optokinetic nystagmus. It is clear that attempts to restrict the field of view of a display by introducing masks with sharp stationary edges may have effects of the nystagmus characteristics, perhaps by

reducing the gain or completely abolishing the eye movements. However it is probably not the case that stationary edges are the only way to reduce nystagmus. There appears to be clear evidence for a dual mode of action of optokinetic nystagmus: a passive mode influenced by the peripheral retina (which has a low gain response) and a pursuit component which acts to boost the gain and also to increase the speed of response of the eyes in response to sudden motion of the visual surround. As shown in Section 2.3.7.1, Robinson (1981) points out the differences in optokinetic nystagmus response in foveate animals, such as humans and monkeys, and afoveate animals, such as rabbits or guinea pigs. The optokinetic nystagmus response of the rabbit is slow to build up to its peak gain. In monkeys and humans the ability of the eye to engage in pursuit boosts the speed of gain increase so that the eye reaches a peak gain in a matter of seconds. Foveate animals also achieve consistently higher gains throughout the exposure compared to afoveate animals which do not have the pursuit reflex.

2.3.7.3 Nystagmus and visual acuity

Post *et al.* (1979) measured the slow phase velocity of nystagmus of subjects, in response to a moving optokinetic drum, with normal vision and with visual blur caused by blurring lenses. They found that the velocity of the slow phase was higher with blurring lenses but state that the effect was expected from the magnifying effect of the lenses. The authors state that eye movements were eventually suppressed when a lens of high power was used and the image was 'too degraded to be resolved as a moving grating'. They did not find any difference in vection with blurring lenses. Precise details as to the powers of the lenses were not available. It may not have been possible to find an effect of visual acuity on the slow phase velocity of eye movements because of the magnifying effect of the blurring lenses. It may have been the case that the slow phase velocity of nystagmus with visual blur differed from that which would be expected from a moving stimulus of the same velocity. Because the stimulus velocities were not matched it was impossible to verify whether or not this was the case. Marmor *et al.* (1987) point out that visual blurring caused by the use of lenses affects contrast sensitivity to a wide range of spatial frequencies, not just the high spatial frequencies. An experiment which measured slow phase velocity of nystagmus, with individual visual acuity and contrast sensitivity scores measured for a variety of subjects, would help to determine whether the slow phase velocity is

dependent on visual acuity or on contrast sensitivity to a wider range of spatial frequencies.

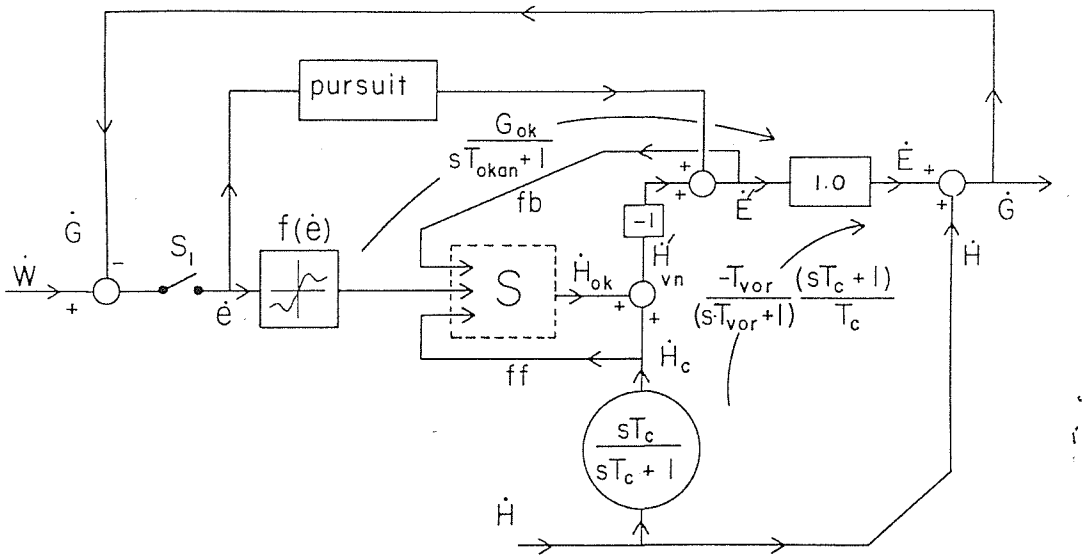

Figure 2.15. Simple model of nystagmus. Robinson, 1981. T_c=cupula time constant, s=the Laplace operator, T_{vor}=vestibulo-ocular reflex time constant, T_{okan}=the time constant of after-nystagmus, G_{ok}=optokinetic gain function. Further explanation is available in the text.

2.3.7.4 Model of nystagmus

Robinson (1981) proposed a model of the slow phase of nystagmus which took into account the foveal pursuit response. It also allowed for head movements as well as movements of the visual surround, such as an optokinetic drum. Shown in Figure 2.15 the model has a number of key features. The input on the left-hand side W shows the 'world velocity', the angular velocity of the visual world with respect to the subject. The summing junction on the left shows that the retinal slip velocity (e) is the difference between the world velocity and the angular velocity of the eye in space (G). Normally W is zero (the world does not move) but in the case of an optokinetic drum W is the drum velocity. The summing junction on the right expresses that the velocity of the eye in space (G) is the sum of the eye velocity in the head (E) and the head velocity in space (H). S is an unknown system by which retinal slip is transformed into an optokinetic signal H_{ok} which is input into the vestibular nuclei (vn), where it constitutes an eye velocity signal.

The finding that the visual system can be split into its various component parts allows for models such as this to be developed. This model is designed to look at the pursuit

component of nystagmus in terms of eye and target velocities and to ignore the saccadic components.

The model is a negative feedback model where retinal slip velocity is used to drive pursuit and optokinetic eye movements which act to reduce retinal slip. The pursuit component in nystagmus is shown at the summing junction just before the eye movement occurs. This model does not show which of the two components driving the eye movements, H_{ok} or the pursuit component, is dominant. It may be possible to split the visual input into foveal and peripheral components to allow for these to be taken into account separately in the model (i.e. with fixation). The model then may be useful in predicting the response of eye movements in experiments where the foveal and peripheral stimuli differ, which are mentioned above in Section 2.3.7.2 (e.g. Van Die, 1986 or Howard, 1984). A proposed model of this nature is presented in the final section of this review.

2.3.7.5 Optokinetic after-nystagmus

After exposure to optokinetic stimuli, 'after-nystagmus' occurs in normal subjects. This is a nystagmus which continues in the same direction as previously but with a lower gain which slowly decays to nothing. Usually it is measured by turning out the lights in the optokinetic drum and observing eye movements as they decay naturally. If lights are not extinguished at the end of a trial the subject will often report that the stationary drum is moving in the opposite direction to that in which it had previously been turning (as the eyes move over the stationary drum) (Brandt *et al.,* 1974). Fletcher *et al.* (1990) tested the relationship between retinal slip velocity (the velocity of image motion on the retina) and optokinetic after-nystagmus in normal subjects by measuring eye velocities in response to known optokinetic drum velocities, from between 10-220°/second and then measuring the velocity of the slow phase of after-nystagmus induced by this motion. It was discovered that the velocity of after-nystagmus increased with increasing retinal slip velocity up to a peak at around 100°/second at which point the after-nystagmus velocity either decreased or reached a plateau. When subjects were made to fixate on a stationary cross and presented with retinal slip velocities of the same order as in the standard condition it was found that after-nystagmus was severely diminished or absent in subjects. It was hypothesised that the development of after-nystagmus relies on the 'charging' of a velocity-storage mechanism which helps to maintain nystagmus during exposure and which dissipates gradually after exposure ends. The velocity storage component of

nystagmus in man appears to rely on foveal slip rather than slip on the retina as a whole. This was confirmed by the study by Muratore *et al.* (1979) mentioned in Section 2.3.7.4, where it was found that a small spot of light stimulating only the fovea could generate after nystagmus with a similar gain and decay as that found by full field optokinetic stimulation.

2.3.8 The role of 'extra-retinal' signals

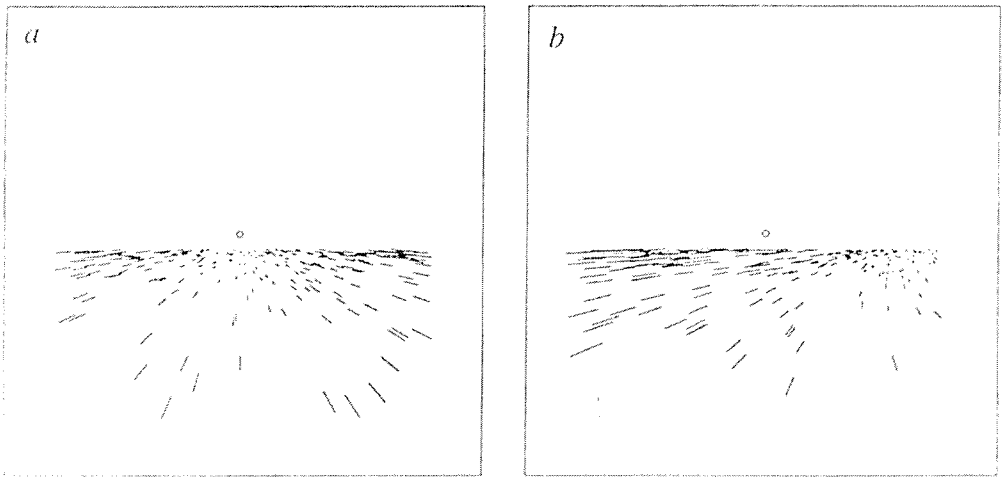

Figure 2.16. Examples of visual flow fields (from Royden *et al.,* 1992).

It has been shown that signals exist that encode information about the nature of eye movements that are occurring. Known as 'extra-retinal signals', it is hypothesised that the brain receives a copy of the signal which is also sent to the eye muscles to move the eye. This signal allows the brain to track the position of the eye with respect to the head and visual surrounding. It allows the interpretation of information about the relative motion between the head and the environment which is not available purely from the pattern of motion on the retina, for example making eye and head movements whilst walking. Royden *et al.* (1992) performed an experiment to show that extra-retinal information is necessary to correctly interpret heading direction when eye movements occur. They used two conditions. In the first an optical flow field was shown on a computer monitor which simulated radial expansion of dots from a focus of expansion. Subjects were allowed to move their eyes by following a pointer on the screen and were instructed to indicate their perception of heading (which direction they felt they were moving in). In the other condition, subjects were not allowed to move their eyes, but simulated eye movements were added into the

pattern of motion presented on the screen, so that the image on the retina was the same as that in the real eye movement condition. Subjects again indicated their perceived direction of motion. Examples of the flow fields are shown in Figure 2.16. Condition 'a' shows translational motion simulation: simulated forward motion towards point of expansion, shown with a circle. Condition 'b' shows translational plus rotational motion.

The hypothesis was that subjects in the simulated eye movement condition would not be able to correctly identify their heading without the extra-retinal signals that occur during eye movements. They found that the average errors in heading estimation were 1.5 and 1.9 degrees for rotation rates of 2.5 and 5°/sec respectively for the real eye movements condition, and 9.8 and 17.3 degrees for the simulated eye movements condition. This may have indicated that the perception of heading was more accurately judged in the eye movement condition.

These results were in contradiction to a previous study (Warran *et al.,* 1990) which showed that heading estimation was equally accurate with or without eye movements. This study used lower simulated eye rotation rates (below 1°/sec). This led to the conclusion that extra-retinal signals are only needed above a certain threshold, say 1°/sec. Below this velocity the brain may be able to interpret the combination of translational and rotational information in the retinal pattern correctly. Above this velocity the brain needs the additional information that extra-retinal signals give, to enable the effect of the eye movements to be filtered out of the visual signal and the heading to be correctly estimated.

Wertheim (1981) commented on the relativity of perceived motion, whereby stability of the visual world is perceived during eye movements despite the visual scene moving on the retina (where an eye movement is intended and not affected, for example, by an external force on the eyeball). The information present on the retina itself cannot supply the necessary information to choose between perception of motion of the world or of the eye. Extra retinal signals are needed to interpret the nature of the motion. It has been suggested that the visual world is perceived to be stationary when the extra-retinal signal generated during an eye movement is equal and opposite to the retinal signal (i.e. they cancel out). Wertheim extends this idea by showing that the world is only perceived to be stationary when the two signals do not differ by more than a 'just noticeable difference'. Subjects were asked to pursue a small circular target on a screen which moved with a triangular waveform (e.g. at a

constant velocity back and forth between two points on the screen). The subject adjusted a potentiometer which increased motion of the background texture, synchronised in time with the motion of the circle. The amplitude of the background motion increased as the potentiometer was increasingly turned. Subjects indicated when motion of the background was first perceived and then turned the potentiometer back to a point where the background motion was no longer perceived. This value was taken as the threshold value for the perception of background motion. The results showed that the threshold velocity for the background motion increased linearly as the speed of the moving circle increased. The result supported the hypothesis that during smooth pursuit the threshold of perception of motion of an object increases proportionally to ocular velocity and the perception of motion depends on the perception of a just noticeable difference between the extra-retinal and retinal signals. It was shown that the extra-retinal and retinal signals could vary through a range of values where no perception of background motion was visible because the difference did not exceed the just-noticeable difference.

2.4 Vection

Vection is the term given to perceptions of self-motion induced by a moving visual scene. There are two forms of vection commonly investigated: i) circular vection - the illusion of rotation and ii) linear vection - the illusion of travelling in a straight path. Vection occurs in the opposite direction to the stimulus direction and occurs either in addition to the perceived object motion or instead of the object motion. On occasions when the perception of self-motion dominates to the extent that the object appears stationary the vection is said to be 'saturated'

2.4.1 Circular vection

Traditionally, circular vection has been studied by the use of optokinetic drums: black and white striped cylinders which rotate about a stationary subject. Usually the drum rotates at a constant angular velocity, for example 5 revolutions per minute. Optokinetic stimuli, such as an optokinetic drum, allow for three perceptual interpretations: (i) that the optokinetic drum is moving and the subject is stationary (ii) that the subject is moving and the optokinetic drum is also moving (in the opposite direction) (iii) that the drum is stationary and the subject is moving. A number of

studies have been completed to discover the visual and psychological aspects involved in circular vection.

2.4.2 Field of view

A standard optokinetic drum excites all of a subject's visual field (about 180° horizontally and 120° vertically). This has been found to create a compelling illusion of motion. The field of view is defined as the horizontal and vertical angle subtended at the subject's eye by the display. Brandt *et al.* (1973) investigated the effect of field of view on the intensity of circular vection by masking parts of the visual field. They found that masking central vision with circular masks of up to 120° in diameter did not significantly reduce the perception of circular vection but presenting a small visual field of 30° centrally reduced the perception of vection so that in a number of cases the subjects perceived only motion of the optokinetic drum.

The intensity of circular-vection experienced could be proportional to the area of the visual field stimulated. Restricting the visual angle to 60° or 30° makes a significant reduction in the area of the visual field stimulated. If this same reduced area was presented only in the peripheral visual field would it produce more vection than in the central field? Post (1988) replicated Brandt *et al's* (1973) study and equated central and peripheral displays in terms of area. Vection was experienced in both cases and it was found that there was no significant difference between the vection intensity in each condition. It was concluded that the area of stimulation was more important than the position in the visual field. A potential problem with these results is that placing a 60° pattern in the central visual field will be exciting the peripheral visual field as well, because the fovea occupies about 1-2°. Hence using equal areas in central and peripheral locations resulted in similar levels of vection. Brandt *et al.* (1973) tried a different method to show that vection was dominated by the peripheral field. They had a large moving field (black and white striped optokinetic drum) in the periphery and a small central field moving in the opposite direction. By measuring eye movements with electro-oculography they found that the subject's eyes were tracking the stripes in the central field, but that they were experiencing vection in the opposite direction: expected from the motion perceived on the peripheral retina. This showed that the peripheral field is dominant for circular vection and also helped to disassociate vection from eye movements.

Stern *et al.* (1990) also showed that a restricted visual field reduced circular vection and that fixation on a stationary cross 10cm from the drum, straight ahead of the subject reduced vection slightly but not so much as the restricted field condition (see Figure 2.17). There were no eye movements in the fixation condition, nystagmus was suppressed by the action of focusing on the stationary cross. Brandt *et al.* (1973) did not find a similar reduction in vection experienced with the presence of a stationary circle and Pyykko *et al.* (1985) found that there was no association between the reports of self motion during caloric nystagmus and the presence or absence of nystagmus at any particular moment.

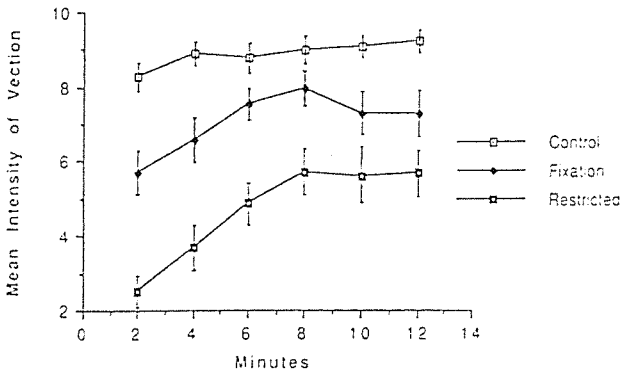

Figure 2.17. Vection experienced with fixation and restricted field of view. (Stern *et al.*, 1990)

Graaf *et al.* (1990) showed that it was the angular velocity of an optokinetic drum that determined the perceived speed of circular vection (the speed at which subjects sensed they were moving), and not the temporal frequency (i.e. the number of stripes passing per second). By manipulating the spatial frequency of the stripes on the drum and the speed of the rotation simultaneously, they were able to maintain the same temporal frequency for different drum speeds (e.g. by doubling the number of stripes and halving the drum speed). Subjects indicated their experience of vection by rotating a small handle at the same angular speed as they felt they were moving. It was found that the angular velocity of the drum was the factor influencing perceived speed of vection, hence people may use a combination of spatial and temporal characteristics of the stimuli to judge the velocity.

2.4.3 Aubert-Fleisch paradox

It has been noted that a moving object is estimated as faster (by a factor of about 1.5) when it is perceived with fixed gaze as compared to when followed by the eyes (Fleisch, 1882; Aubert, 1886). It has been suggested that when the brain is relying solely on retinal information, (i.e. when the eyes are stationary), there is an over estimation of the stimulus speed. It is not understood why holding the eyes still should cause the brain to over estimate the speed of the stimulus. Graaf *et al.* (1991)

performed an experiment to see if the same illusion occurred during vection. They predicted that subjects would experience vection at different subjective speeds with and without fixation of the eyes. They used an optokinetic drum and were able to project a small cross onto the drum from the chair on which subjects were seated. They found that subjects experienced an apparent acceleration of their perceived vection speed when the cross appeared, and a deceleration when the cross disappeared and their eyes tracked the stripes again. In a second experiment the subjective vection speeds were measured in separate sessions so as not to allow direct comparison of the two conditions. In this situation vection speeds were estimated as being the same with or without the cross. This helps explain a contradiction in the literature where Dichgans *et al.* (1973) found no difference between the vection experienced in the two conditions measured separately. It seems that subjects need 'back to back' comparisons in order to sense the difference.

2.4.4 Linear vection

Andersen *et al.* (1985) challenged the theory that the peripheral visual field is entirely responsible for experiences of vection. In a series of experiments they found that linear vection (in this study, simulated motion in the forward direction) could be induced by small visual angles in central vision only. Visual angles of 7.5°, 10.6°, 15°, 21.2° were used together with varying speeds. Subjects were exposed to a radially expanding pattern of dots, simulating forward movement through space filled with dots. They pressed a button when experiencing vection and released it when they felt stationary. Results showed that vection occurred even at the smallest visual angle of 7.5°. This led them to propose a theory that there are two modes of visual processing. An ambient mode (peripheral vision) which is primarily sensitive to low spatial frequencies and requires a large area of involvement and a higher order processing mode sensitive to complex motion information such as depth and stereoscopic cues. It was suggested that the higher mode would be more susceptible to suggestion, such as viewing a display whilst sitting in a vehicle capable of motion (Andersen *et al.* 1985). Telford *et al.* (1993) found that there was significantly more vection experienced when the display was shown through a window in a booth, as in Anderson *et al.'s* (1985) experiment. They attribute this to the edges of the window acting to give extra depth information (i.e. the occlusion edges specify the moving visual display as the background).

2.4.5 Discussion

As a conclusion it may be hypothesised that peripheral vision may be more important for the interpretation of circular motion than for linear motion. Brenner *et al.* (1994), point out that object motion on the periphery is usually a result of a tracking motion of the eyes, or eyes and head, to follow a moving object with the intention of keeping the object in central vision. During tracking, the surrounding environment moves on the periphery of the retina. When a user is presented with an environment that moves on the periphery of the retina, which is not caused by tracking eye movements, and particularly if the whole visual field is excited, it gives the illusion of motion in a circular path.

Royden *et al.* (1992) investigated the perception of heading (i.e. the perceived direction in which subjects felt they were travelling). The simulation consisted of a radial expansion of dots from a focal point in the distance. This was similar to those used by Anderson *et al.* (1985) who investigated linear vection with small fields of view. Royden *et al.* (1992) point out that people perceive heading in linear (forwards) motion by interpreting the point of expansion of the dots. For linear vection it seems likely that the central visual field may be more important than the peripheral visual in following the trajectory of the dots from the point of expansion.

2.5 Motion sickness

Motion sickness is a phrase used to refer to a wide range of unpleasant symptoms experienced during exposure to motion of the body or in response to motion of visual images without concurrent motion of the body. The symptoms experienced range from dizziness, headaches, dry mouth, excess salivation and cold sweating to stomach awareness, nausea and at the extreme end of the scale vomiting. Research has been systematically conducted over many years into the various forms of motion sickness, often by laboratory simulations in order to investigate various forms of motion and the related motion sickness experienced. This review will consider the research that has been conducted into visual motion sickness which included exposure to optokinetic drums, virtual reality, research into eye movements and the

vestibulo-ocular reflex. The theories which have underpinned motion sickness research are presented.

2.5.1 Sensory conflict theory

Sensory conflict or cue conflict theory was developed as a way to explain and predict situations in which motion sickness may arise. Reason and Brand (1978) explained that motion sickness arose when the inputs from vision, the vestibular system and the proprioceptor system were at variance with one another and hence at variance with what was expected from past experience. In its simplest form sensory conflict refers to a mis-match between some or all of the sensory inputs by which we balance and sense motion. Reason and Brand suggest that the brain, from birth, builds a 'neural store' which holds various models of the motion environment encountered. It is when the various motion inputs are at variance with those expected from this 'neural store' that motion sickness arises, until the neural store has been able to update to account for the new motion input combination encountered. This updating

Table 2.1. Categories of sensory conflict. Griffin (1990).

	Category of conflict	
Type of conflict	Visual-Vestibular	Canal-Otolith
Type I	Visual and vestibular systems 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 expected vestibular system	Canals signals in the absence of an expected otolith signal
Type IIb	Vestibular system signals in the absence of an expected visual signal	Otoliths signals in the absence of an expected canal signal

of the 'neural store' can explain the reduced motion sickness experienced on repeated exposures, for example when a sailor has been at sea for many weeks. It is possible to categorise sensory conflict into six groups based on the different motion inputs which are at variance with one another. Table 2.1 (from Griffin, 1990) shows

these groups. Examples of the different types of exposure which may lead to the conflicts in Table 2.1 are shown in Table 2.2.

Table 2.2. Types of sensory conflict and situations where these occur. (Griffin 1990).

	Category 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 an optical device 'Pseudo-Coriolis' stimulation	Making head movements whilst rotating (Coriolis or cross-coupled stimulation) Making head movements in an abnormal environment which may be constant (e.g. hyper or hypo-gravity) or fluctuating (e.g. linear oscillation) Space sickness Vestibular disorders (e.g. Ménières disease, acute labyrinthitus, trauma labyrinthectomy)
TYPE IIA A signals in the absence of expected B signals	Cinerama sickness Simulator sickness 'Haunted Swing' Circular vection	Positional alcohol nystagmus Caloric stimulation of the semi-circular canals Vestibular disorders (e.g. pressure vertigo, cupulolithiasis)
TYPE IIB B signals in the absence of expected A signals	Looking inside a moving vehicle without external visual reference (e.g. below deck in a boat) Reading in a moving vehicle	Low frequency (< 0.5 Hz) translational oscillation Rotating linear acceleration vector (e.g. barbecue spit rotation about an off-vertical axis)

2.5.2 Visual causes of motion sickness

A potential cause of sensory conflict with moving visual scenes is a conflict between the visual and vestibular system, whereby the visual system signals in the absence of expected vestibular signals. According to Table 2.2 this will tend to occur in cases where there is simulated motion in the visual display but no actual motion of the viewer (see type IIa, Table 2.2). In these cases there would be an expected vestibular input which should match the visual input, but the vestibular input is

missing in these situations. There may also be delayed information between the visual and vestibular systems, for example if there is a delay between a head movement and the visual scene updating (type I) in virtual reality systems, or magnification problems where the visual scene moves faster than is expected from the speed of the users head movement (see Section 2.6.2).

2.5.3 Discussion of sensory conflict theory

Sensory conflict theory is at its best when it is desirable to predict if a particular situation will be nauseogenic. It is not able to predict which of two nauseogenic situations will cause the greater sickness or account for the individual differences in motion sickness among subjects and within subjects on different occasions. In some cases it may even be unable to correctly identify nauseogenic situations. For example in Table 2.2 circular vection appears under Type IIa conflict, where it is stated that there is a visual input in the absence of expected vestibular input. In this case however, it could be argued that there should be no conflict between the visual and vestibular systems, because the vestibular system would not be excited during constant speed rotation (see Section 2.2.1). However, situations where circular vection is produced, such as during exposure to an optokinetic drum, can cause considerable motion sickness (Stern *et al.,* 1990). Further research into the individual situations where motion sickness occurs is necessary to create models for these situations which enhance or replace the sensory conflict models.

2.5.4 Motion sickness and vection

Sensory conflict theory appears to implicate vection (visual system indicating motion in the absence of vestibular signals) as the cause of motion sickness with optokinetic displays. Studies concerning vection often assume a link between the vection measured and the potential for the device producing the vection to cause sickness. Studies have measured both vection and motion sickness, such as Hettinger *et al.* (1990) who exposed subjects to a flight simulator. In this case it was shown that all subjects who experienced motion sickness had also experienced vection. They stated that vection was hence a necessary prerequisite for motion sickness. However, some subjects experienced vection and no motion sickness. They did not attempt to measure vection on a variable scale but simply categorised it as either

having occurred or not, so no attempt to find correlations between individual motion sickness scores and vection was made.

Similarly, Hu *et al.* (1997) measured vection and motion sickness incidence with varying numbers of stripes in an optokinetic drum. They found that a particular frequency of stripes (24 pairs of black and white stripes) caused maximum sickness, maximum vection and also the highest frequency of nystagmus eye movements amongst the trial groups. The assumption was made that the maximum vection in this condition was also responsible for the maximum motion sickness. However there were no correlations presented of individual motion sickness and vection scores. The literature available does not appear to provide any conclusive proof of a direct link between vection and motion sickness, although it is often implied.

2.5.5 Alternative theories of motion sickness

Treisman (1977) proposed an evolutionary explanation for the development of motion sickness among humans and animals. The explanation was an attempt to explain why motion sickness responses could have persisted in animals and humans despite millions of years of evolution and there being no obvious benefit from vomiting in response to motion. It would be expected that the motion sickness response would have been eliminated by evolution if, indeed, there was no benefit. Treisman explains that in every moment of waking life an animal or human must organise its movements in response to the surrounding world. Head and eye movements must be co-ordinated and responses such as the vestibulo-ocular reflex must be continuously calibrated in order to prevent images slipping on the retina. The brain must also monitor neck movements and feedback from the limbs. These 'calibrations' require fine tuning which can be disturbed in certain circumstances. The hypothesis of Treisman is that motion sickness arises from repeated challenges to re-determine the calibrations of the various senses. Such challenges may arise when a subject is placed in a novel situation or in a situation where "one input is repeatedly misleading in what it predicts for the other". It is explained that this theory differs from the sensory conflict or sensory rearrangement theory in that the conflict does not arise between current inputs and those expected from past experience but from a situation where two closely coupled systems, for example the visual and vestibular system, are forced to make continuous comparisons in order to perform a task. The reason promoted for this causing nausea and vomiting is that the constant calibration of the senses can be disturbed by toxins present in the diet which often have an effect on

the vestibular system and may disturb the vestibulo-ocular reflex. In this case it would be a positive evolutionary advantage to vomit in order to rid the body of the remaining undigested toxins.

2.5.5.1 Eye movement theories of visual motion sickness

Ebenholtz *et al.* (1994) *proposed* a hypothesis that nystagmus may be responsible for motion sickness, as an alternative to vection being the cause of visually induced motion sickness. This was based on some empirical evidence that retrobulbar anaesthesia (anaesthetising the muscles behind the eye) significantly reduced sickness after surgery. They suggested that movements of the eye muscles may elicit afferent signals that stimulate the vagus nerve which, due to its proximity to the vestibular nuclei, may result in stimulation of the vestibular system. They point out that labyrinthine defective subjects (i.e. those without a functioning vestibular system) do not experience any symptoms of motion sickness when exposed to optokinetic stimuli even though they still experience vection (Cheung *et al.*, 1989). They use this evidence to suggest that a functioning vestibular system is a necessary requirement for the symptoms of motion sickness to occur and that the input from eye movements is a likely cause of this vestibular stimulation.

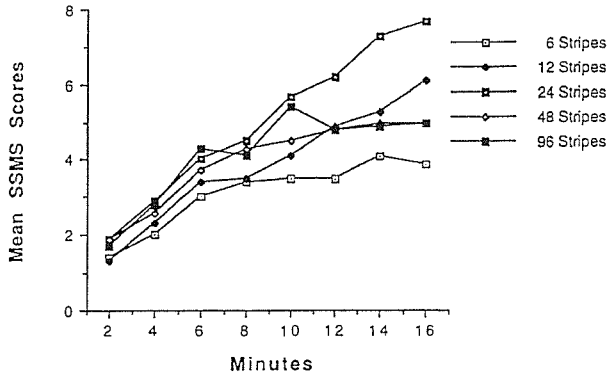

Figure 2.18a. Motion sickness scores for different stripe patterns. Hu *et al.* (1997).

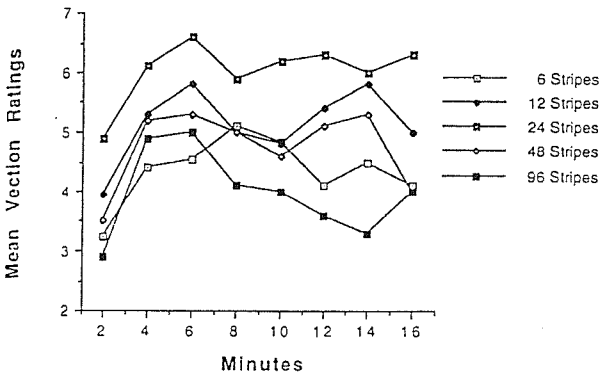

Figure 2.18b. Vection ratings for different stripe patterns. Hu *et al.* (1997).

Hu *et al.* (1997) attempted to test the above hypothesis by exposing subjects to different spatial frequencies in an optokinetic drum. They hypothesised that different numbers of stripes painted around the inside of the drum would cause different frequencies of nystagmus. Those subjects showing the highest frequencies would

experience more vection and hence greater symptoms of motion sickness because they would have made the greater number of eye movements. Nystagmus frequencies were measured for one minute in each of the conditions (6, 12, 24, 48 and 96 stripes around the drum). Motion sickness symptoms were measured in separate sessions of 16-minute exposures to the optokinetic drum. They found that 24 stripes elicited the highest average frequency of nystagmus across subjects, and also the highest ratings of sickness. The authors did not state whether there was a correlation between individual subject's nystagmus frequencies and motion sickness. They reported that there were significantly more symptoms of motion sickness in the condition where subjects viewed 24 stripes compared with 6 and 96 stripes, but presumably there were no significant differences between 24 and 12 or 48 stripes. Figure 2.18a shows the motion sickness ratings and Figure 2.18b shows the vection ratings for the various stripe patterns.

In a separate experiment Hu *et al.* (1998) measured nystagmus, with 87 subjects for a total of 16 minutes, in response to an optokinetic drum spinning at 60°/second. Vection was assessed at two-minute intervals during exposure and an average score calculated. Eye movements were recorded with electro-oculography and an average frequency calculated for each minute. It was found that there was a positive correlation between the average nystagmus frequency per minute and average vection. It was also found that there was a positive correlation between nystagmus frequency each minute and overall average motion sickness symptoms. A correlation between vection and motion sickness was not mentioned, hence presumably was not found. The authors concluded that vection was the cause of motion sickness and that vection was influenced by nystagmus frequency.

2.5.6 Discussion

The hypothesis of Treisman has some advantages over sensory rearrangement theory. It makes the case that situations where there is a difference in closely coupled sensory systems which need to be calibrated can result in motion sickness. This may enable experiments to be designed which can quantify these differences and test the hypothesis directly.

The eye movement hypothesis of Ebenholtz (1994) is difficult to test directly. The experiment by Hu *et al.* (1998) attempted to study eye movements in response to an optokinetic drum. The study showed that eye movement frequency influenced motion sickness. There was a higher incidence of vection and of motion sickness when the frequencies of nystagmus were higher. They did not show a direct correlation between vection and motion sickness. The authors state that subjects were instructed to look at the stripes if nystagmus was absent during the exposure (e.g. when subjects may have not been focusing on the stripes). It is not clear whether these periods (when nystagmus was absent) were accounted for in the average frequency of nystagmus calculation. If the analysis consisted of counting the total number of saccades and dividing by time to give an average frequency, then those subjects who had the greater number of 'non-focusing' periods may have had the lower frequencies. Not focusing on the stripes (or perhaps closing the eyes altogether) may have resulted in the lower sickness levels found with lower frequency. It is not possible to comment further, because of the lack of information concerning the procedure of the electro-oculography analysis.

The hypotheses of Ebenholtz *et al.* (1994) and Treisman (1977) may be useful in focusing research on aspects of the visual system and eye movements rather than focusing on vection only, which is implied as a cause of motion sickness by the sensory conflict model. The research may need to be more clearly defined and the correlations between eye movements, visual characteristics of subjects, vection and motion sickness symptoms should be systematically controlled and investigated.

2.6 *Motion sickness as a result of visual stimuli.*

2.6.1 Introduction

This section deals with visually based motion sickness studies such as magnification of vision, vestibulo-ocular reflex adaptation and motion sickness, the effect of anti-motion sickness drugs on visual motion sickness and motion sickness with optokinetic stimuli.

2.6.2 Motion sickness and vestibulo-ocular reflex adaptation.

Situations in which there is retinal slip and an adaptation of the vestibulo-ocular reflex gain, such as motion of the head with magnified vision, have been shown to be highly provocative stimuli which induce oscillopsia (the perception of motion of the visual world without concurrent eye movements, for example at the end of an eye movement, the world may appear to continue moving for some time) or motion sickness in subjects exposed (Demer *et al.,* 1987, Melvill Jones *et al.,* 1981). In these cases it is reported that oscillopsia and motion sickness symptoms decreased once vestibulo-ocular reflex adaptation had taken place sufficiently to reduce retinal slip. Demer *et al.* (1987) measured the response of the vestibulo-ocular reflex before and after adaptation to 2.2X magnifying glasses. The vestibulo-ocular reflex gain increased after a 15 minute exposure to sinusoidal rotation at 4 Hz, amplitude 30°/second. The vestibulo-ocular reflex gain measured in darkness increased from a mean of 0.74 to 0.83 after exposure. The vestibulo-ocular reflex in light was also measured and found to increase from a mean of 1.07 to a mean of 1.37, the increase being larger due to the visual influence on the reflex. There was a concomitant improvement in the dynamic acuity of subjects of between 30-100%. Subjects typically reported reduced oscillopsia with increased adaptation and improved visual acuity.

Melvill Jones *et al.* (1979) made a discovery whilst measuring the vestibulo-ocular reflex gain with reversed vision in stroboscopic light. No subjects reported symptoms of motion sickness whereas all subjects in a similar condition in normal light reported severe nausea (further information in Section 2.3.4). A second experiment was conducted (Melvill Jones *et al.,* 1981) which also found that no subjects experienced

motion sickness with strobed light. The authors suggested that motion sickness may arise from the process of adaptation of the vestibulo-ocular reflex gain to novel visual stimuli. They suggest that the reduced adaptation found at higher frequencies of the vestibulo-ocular reflex response with strobed light may hence reduce motion sickness.

Draper (1998) conducted a series of experiments which investigated motion sickness in conditions which called upon the vestibulo-ocular reflex to adapt. The experiments were devised using virtual reality whereby the head movements of subjects and the corresponding virtual reality visual images in response to the head movement were not perfectly matched. The visual image would move at a velocity of 0.5X (minimised), 1X (neutral) or 2X (magnified) head velocity which gave an equivalent effect to magnifying glasses, as discussed previously. Draper made the hypothesis that vestibulo-ocular reflex adaptation would take place in the 0.5X and 2X visual magnification conditions. The process of vestibulo-ocular reflex adaptation to changes in image magnification would result in motion sickness and there would be a correlation between the vestibulo-ocular reflex adaptation of the subjects and the motion sickness symptoms. Significant adaptation did take place in the gain in the 0.5X and 2X conditions. There was no significant change in the neutral condition. Motion sickness occurred in all three conditions, but was significantly higher in the 0.5X and 2X magnification conditions compared with the neutral condition. There was no significant difference in the motion sickness incidence between the two magnified conditions. Only weak correlations were found between the magnitudes of the gain changes in the vestibulo-ocular reflex and the motion sickness scores. Only nine subjects were tested in the experiment, which may have been too low to observe significant correlations with something difficult to measure and as variable as vestibulo-ocular reflex gain.

2.6.2.1 Effect of anti-motion sickness drugs on the vestibulo-ocular reflex gain

Pyykko *et al.* (1985) studied the effect of scopolamine and dimenhydrinate, two commonly used anti-motion sickness drugs on the different types of nystagmus: nystagmus induced by caloric irrigation, vestibular nystagmus induced by rotation of the subject and optokinetic nystagmus induced by watching an optokinetic drum spinning. They studied the frequency and the gain of the nystagmus in each case. During caloric nystagmus there was a significant difference in the gain of the

nystagmus between a placebo condition and the two active drug conditions where the maximum slow phase velocity was 30°/second with placebo, 23°/second with dimenhydrinate, 26°/second with one TTS-scopolamine and 21°/second with two TTS-scopolamine (double dose). There were no significant differences in the frequencies of nystagmus. There were similar reductions in the gain of the nystagmus in the rotatory test where the gain found with placebo was 0.75, 0.67 with dimenhydrinate, 0.74 with one TTS-scopolamine and 0.56 with two TTS-scopolamine. The changes in gain were statistically significant between the different treatment conditions. Pair-wise comparisons were significant between conditions except between placebo and one TTS-scopolamine. Figure 2.19 shows the gain changes for optokinetic stimulation with various drugs.

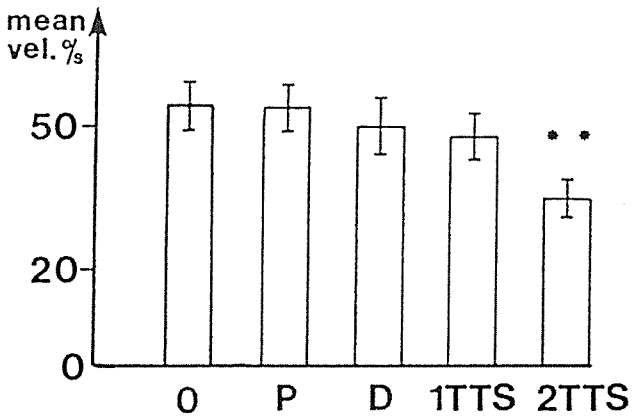

Figure 2.19. Changes in optokinetic nystagmus gain with various anti-motion sickness drug combinations. O=baseline, P=placebo, D=Dimenhydrinate, 1TTS= one TTS scopolamine and 2TTS = two TTS scopolamine.

Mean optokinetic nystagmus gain in response to drum motion at a constant velocity of 90°/second with placebo treatment was 54°/second, 50°/second with dimenhydrinate, 48°/second with one TTS-scopolamine and 35°/second with two TTS-scopolamine. In pair-wise comparisons there was a significant difference between the gain in the two TTS-scopolamine and against placebo and dose response relationships existed in the one TTS-scopolamine and two TTS-scopolamine conditions. The authors state that the most consistent results were found with two TTS-scopolamine treatment. The drugs do appear to be active on the vestibular system and influence the gain of eye movements. This may explain part of their action in reducing motion sickness (see below).

Further experiments were conducted by Pyykko *et al.* (1985) into the effect of the anti-motion sickness drugs on motion sickness symptoms. They studied the response of subjects to a Coriolis test whereby subjects inclined their heads forward or backwards about 20° every fifth second whilst being rotated at a constant velocity inside an optokinetic drum. The results showed that subjects experienced lower

motion sickness when treated with active drugs as oppose to placebo. The influence of anti-motion sickness drugs to standard optokinetic stimulation without any motion of the subject was not investigated.

Gordon *et al.* (1996) tested the vestibulo-ocular reflex gain of subjects who were either rated as highly susceptible (39 subjects) or not susceptible (30 subjects) from a motion sickness history questionnaire. The vestibulo-ocular reflex gain was evaluated by a sinusoidal harmonic acceleration test at frequencies of 0.01, 0.02, 0.04, 0.08, 0.16 Hz. The vestibulo-ocular reflex gain was significantly higher in subjects susceptible to motion sickness at 0.02 and 0.04 Hz and the phase lead was significantly lower at 0.01, 0.02, 0.04 and 0.08 Hz than non-susceptible subjects. Gordon *et al.* (1996) also report a previous study (Shupak *et al.,* 1990) where it was found that the vestibulo-ocular reflex gain of non-susceptible subjects was lower after one month of regular sailing at frequencies of 0.01 to 0.08 Hz compared to susceptible subjects. The authors conclude that subjects who are more susceptible to motion sickness have a more intense vestibular response than those who are less susceptible. The findings that less susceptible crew members of a navy ship had lower vestibulo-ocular reflex gains after one month of sailing may indicate that they had adapted better to the conditions than susceptible crew members, rather than indicating a natural susceptibility to motion sickness. Further study will be needed to verify whether the test can be used as a predictive measure of motion sickness susceptibility. The finding of lower gains among less susceptible subjects appears to be consistent with the findings of Pyykko *et al.* (1985) where they found that the effect of anti-motion sickness drugs on the vestibular system resulted in lower gains of caloric, vestibular and optokinetic nystagmus. The effect of anti-motion sickness drugs on gain and the lower gain of less susceptible subjects may be helpful in the reduction of motion sickness on ships and in transport systems where there is a restricted external visual scene or no external vision for reference. In this case a lower gain of the vestibulo-ocular reflex would help to minimise slipping of the images on the retina, as the eyes move in response to rotational motion of the head but the visual scene stays stationary relative to the head. In an optokinetic drum the occurrence of a lower slow phase gain may not be of any benefit because in this instance the lower gain would actually increase the slipping of images on the retina as the eyes attempted to track the stripes.

view was too narrow for nystagmus, the slow phases may possibly have been interrupted by the edge of the display, or perhaps subjects were able to look at the edge of the display in order to stop their eyes from moving. Foveal dominance in optokinetic nystagmus has been demonstrated by Van Die *et al.* (1986) and in several other studies, which would have lead to the prediction that the restricted visual field should not suppress nystagmus. Van Die *et al.* found that nystagmus was dominated by central vision until the visual field was restricted to below 20° or lower in which case the nystagmus was suppressed, they point out that stationary edges may be responsible for suppressing nystagmus in very small central visual fields. Murasugi *et al.* (1986) found that stationary edges were the most important factor in the suppression of nystagmus. Stern's experiment found reduced motion sickness with reduced field of view, but this may not have been corrected for the possibility of suppression of nystagmus by the stationary edges. So, in effect, it may have been another form of fixation where the subject could choose consciously, or perhaps unconsciously, whether to fixate or not. Stern *et al.* (1990) attributed the increased sickness in the full field condition to increased vection in that condition. Again, there were no correlations presented of individual motion sickness symptom scores against individual vection scores, so although the condition causing the greatest vection also had the greatest motion sickness incidence, it cannot be assumed that vection and motion sickness are directly related from these particular results.

2.6.3.3 Rotation speed of the drum

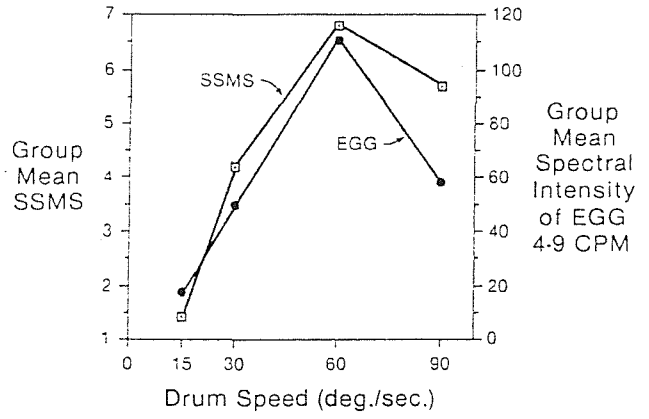

Figure 2.21. Motion sickness symptoms at varying drum speeds.

Hu et al. (1989) recruited 60 subjects and split them into four groups with different drum rotation speeds in each. The four speeds were 15°/s, 30°/s, 60°/s and 90°/s. The showed that only one person reported nausea in the 15°/s group, five people reported nausea in the 30°/s group, eight people in the 60°/s group and six people in results the 90°/s group. They attributed the increased symptoms of sickness to increased experiences of vection as the speed increased. The 60°/s speed was said

2.6.3 Factors affecting motion sickness with optokinetic drums

2.6.3.1 Introduction

This section presents studies which have investigated optokinetic drums or similar stimuli and the variation of motion sickness with varying conditions, for example field of view, fixation and speed of the stimulus.

2.6.3.2 Field of view

Stern *et al.* (1990) tested three groups of subjects: a control group who observed the entire visual field in an optokinetic drum, a restricted visual field group who observed only the central 15° and a fixation group who viewed a centrally located target, designed to suppress nystagmus. Stern *et al.* (1990) hypothesised that both the restricted field and the fixation group would experience less vection than the control group and hence experience fewer symptoms of sickness. A second hypothesis was that 'the fixation group would experience more vection than the restricted visual field group and, therefore, would experience more symptoms'.

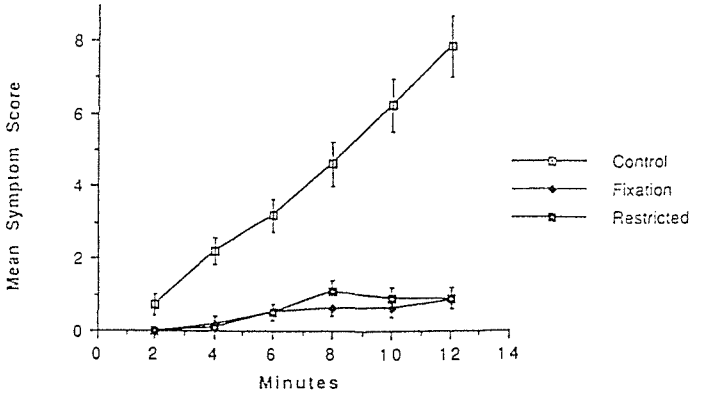

Figure 2.20. Symptoms of motion sickness in an optokinetic drum with fixation and restricted visual field (Stern *et al.*, 1990).

The reports of vection were lowest in the restricted field group, higher in the fixation group and the control group experienced the highest vection, as hypothesised. There were no cases of nausea in either the fixation group or the restricted field group, but the overall trend was a lower incidence of symptoms in the restricted field group. Nystagmus was suppressed in the reduced visual field condition and greatly suppressed during the fixation condition. Stern *et al.* (1990) state that the lower vection experienced in the restricted field condition compared to the fixation condition but the greater nystagmus in the restricted field condition is evidence of the 'partial dissociation of vection and nystagmus, thereby partially dissociating eye movement conflict from self-motion cue conflict'. The suppression of nystagmus in the restricted visual field condition may have indicated that the field of

to be the point vection was saturated. At the 90°/s speed, subjects experienced a severe blurring of the stripes and were said to have experienced a less compelling illusion of vection. Hu *et al.* (1989) suggest that the variation of vection with varying speeds of drum rotation may account for the variation in motion sickness symptoms experienced. No data for correlations between individual vection and sickness scores were shown.

Research into eye movements occurring with various selective stimulation of the retina, for example the fovea and peripheral retina (as discussed in Section 2.3.7.2) showed that the gain of nystagmus decreased with an increase in the speed of the optokinetic drum (e.g. Van Die *et al.* 1986, Cheng *et al.* 1975). At the very high drum velocities the subjects experienced a severe blurring of the stripes, presumably because the gain of the slow phase of nystagmus would be approximately 0.5-0.6 (Howard, 1984) at this velocity. There would a slipping of the image on the retina at a velocity of 36-45°/second for this range of slow phase gain which would account for the severe blurring experienced.

In a similar experiment, which did not use an optokinetic drum but used a military flight simulator (Sharkey *et al.,* 1991), it was found that the 'global visual flow rate' influenced symptoms of motion sickness. Global visual flow was defined as the velocity of the simulated flight divided by the altitude. Lower altitudes result in higher global visual flow rates. Essentially 'global visual flow rate' is a measure of the speed with which images move across the screen on which the simulation is presented. Higher global visual flow rates (i.e. higher velocity of images) were found to significantly increase the symptoms of motion sickness.

2.6.3.4 Fixation

As mentioned in Section 2.5.5.1, Ebenholtz *et al.* (1994) proposed a hypothesis that nystagmus may be responsible for motion sickness. In the experiment conducted by Stern *et al.* (1990) there was a reduction in motion sickness when eye movements were suppressed by the method of fixation (looking at a stationary object in front of the stripes) and as mentioned above there was also a reduction in sickness in the restricted visual field condition where it was possible that fixation was also taking place. It could be argued that the reduced nystagmus was responsible for the reduction in motion sickness, as hypothesised by Ebenholtz (1994) although there is

also a reduction in the motion of images on the fovea during fixation, so that only the peripheral visual field is stimulated. An effect of a reduction of foveal motion on sickness is an alternative possibility. It was shown by Shelhamer *et al.* (1994) that image slip on the fovea was possibly the most significant error signal in the adaptation of the vestibulo-ocular reflex to continuous motion of the visual surround (i.e. optokinetic stimulation without motion of the subject). It was also shown (Van Die *et al.*, 1986, Howard 1984 – see Section 2.3.7.2 for a full discussion) that the fovea is dominant in controlling optokinetic nystagmus. The error signal for the control of nystagmus and for the adaptation of the vestibulo-ocular reflex appears to be, in both cases, image slip on the fovea. It might be argued that foveal image slip might also be an error signal which has an effect on motion sickness. The reduction in motion sickness with fixation here opens up the idea as a possibility, as does the increased motion sickness with increasing speed of the optokinetic drum (Hu *et al.*, 1989) where foveal image slip increases with increasing drum speed (because of reduced optokinetic nystagmus gain with increasing drum speed).

Prothero *et al.* (1999) proposed that motion sickness in virtual reality displays occurs as a result of a sensory conflict between rest frames selected from the motion cues found in the simulation and the true motion of the observer. The rest frame is defined as a reference frame which an observer perceives to be stationary. In normal life we naturally assume the environment to be stationary and perceive ourselves to be moving, but the brain could equally perceive that we are stationary and everything else in the environment is moving. Generally the nervous system will select the rest frame which simplifies the calculations of the motion of objects. In the case of optokinetic drums most subjects perceive themselves to be moving and the drum to be stationary because we have come to expect the external environment to be stationary from experience. In virtual reality, the rest frame is taken as the visual stimulus on the screen because it occupies the entire vision of the subject. There is a conflict between this rest frame and the actual motion of the subject, who is usually stationary.

Prothero *et al.* (1999) conducted an experiment to test the rest frame hypothesis. They recorded an optokinetic stimulus to video tape by placing a camera on a tripod and rotating it at 60°/second (the recording was made on a university campus, so subjects watching the recording saw the buildings of the university moving on the screen). The resulting recording was played to subjects via a virtual reality display system which, in one condition, was used as normal (occluded condition) and, in a

second condition, was used with a see-through screen where the subjects could see the room in which the experiment was occurring through the screen as well as the visual display (see-through condition). Prothero *et al.* (1999) call this an 'independent visual background'. They predicted that the 'independent visual background' condition would reduce symptoms of ataxia and motion sickness by providing an independent rest frame which was consistent with their actual body motion (i.e. stationary).

The results of an initial study showed that motion sickness symptoms were significantly lower in the see-through condition and that there was significantly lower ataxia in the see-through condition. A second experiment was devised which attempted to increase the focus of the subjects into the optokinetic recording, to prevent them from just staring through the display at all times. The recording was made as before by spinning a camera on a tripod at the same speed (60°/second) but on each cycle of the camera, somebody in front of the camera would hold up a different coloured flag, each time the camera was pointing in his direction on its rotation cycle. When the subjects watched the video playback on the virtual reality system they had to call out the colours of the flag at each cycle to ensure that they were looking at the video display. The results showed that there was no difference this time in motion sickness scores between the two conditions or in post exposure ataxia. The motion sickness scores were significantly higher after exposure than before exposure, indicating that the stimuli had a bona fide motion sickness effect.

The difference between the two experiments appears to be due to the nature of the task which forced subjects to pay attention. It is possible that in the first experiment the subjects were looking through the display and focusing on the background. In this case they would be fixating and largely ignoring the visual stimulus. This possibly accounts for the finding of reduced motion sickness and ataxia in the see-through condition in this first experiment. In the second experiment the motion sickness incidence was not significantly different. This may indicate that the subjects when forced to look at the moving display did not find any benefit from the see-through display. The simplest way to find out whether subjects were ignoring the content of interest (the visual display), would be to measure eye movements using electro-oculography to determine whether nystagmus eye movements were occurring in each condition.

Prothero *et al.* (1999) conclude that the see-through display was beneficial in reducing motion sickness and suggest that the rest frame may be selected by peripheral vision at a subconscious level so the 'independent visual background' could be presented purely in the peripheral vision. Another conclusion could be that subjects were likely to be fixating their eyes in the first experiment which would account for the reduced sickness and were unable to do so when forced to look at the display more actively. It could be dangerous to assume that by placing an additional rest frame into peripheral vision alone can reduce motion sickness, when the motion perceived on the fovea would appear to be more important in influencing eye movements, motion sickness and vestibulo-ocular reflex adaptation (Howard, 1984; Stern *et al.,* 1990; Shelhamer *et al.,* 1994).

2.6.3.5 Habituation

It has been shown that habituation occurs with visual stimuli causing motion sickness. That is, the symptoms become less severe on repeated exposures in much the same way as people become accustomed to real motion (e.g. on ships). It was shown (Hu *et al.,* 1997) that all subjects exposed to an optokinetic drum adapted to the exposure whether or not they continued exposure whilst experiencing severe nausea. Seventeen highly susceptible subjects were split into two categories: one in which the exposure was stopped immediately on sensation of nausea, and one in which the subjects continued for 16 minutes even whilst experiencing nausea. It was found that the number of sessions required (16 minutes each, with two days in-between) to fully adapt (i.e. to not feel any stomach awareness or nausea during the 16 minute period) was not significantly different between the two groups.

In a similar experiment, Zhao *et al.* (1999) found that habituation did not occur if subjects were exposed to an optokinetic drum rotating at 60°/sec with 30 minute intervals. In this case susceptible subjects were sensitised to the optokinetic stimulus and reported increased symptoms over three sessions. In this case it was found that symptoms lingered from the previous exposure and it was concluded that it is not possible to habituate over short time periods where symptoms have not fully subsided between exposures.

2.6.3.6 *Previous susceptibility to motion sickness*

Hu *et al.* (1996) recorded motion sickness symptoms from subjects exposed to an optokinetic drum for 12 minutes. Past experience of motion sickness was recorded using a motion sickness history questionnaire (Reason, 1975). It was found that previous susceptibility to motion sickness was highly correlated with the symptoms generated by the novel stimulus, the optokinetic drum. The authors conclude that visually induced motion sickness may share a similar physiological basis to motion sickness more commonly encountered, for example that arising in ships and motor transport. Previous susceptibility to motion sickness has not been consistently measured in optokinetic drum experiments. More detailed study of this matter may help to understand the underlying physiological mechanisms in greater detail.

2.6.4 Alternatives to optokinetic drums

Kramer *et al.* (1998) presented optokinetic stimulation on a virtual reality head-mounted display. In several experiments, eye movements were recorded in response to a traditional optokinetic stimulus or laser target and to a simulation of the same type, presented on the virtual reality display. It was found that pursuit eye movements of similar gain and phase were generated by a laser pointer and the virtual reality system. Optokinetic nystagmus was generated with similar properties to that found in the normal optokinetic drum with similar gain and a gain which decreased with increasing speed of the stimulus. After-nystagmus was also generated with similar gain and decay properties in both conditions. The authors conclude that virtual reality is a useful tool for the study of optokinetic stimuli and eye movement responses. They point out some drawbacks of their particular equipment, namely that subjects were unable to wear glasses in the virtual reality condition and that their particular hardware system was not fast enough to enable real-time head-tracking (the head-mounted display was merely used as a wide field of view monitor system). Used in this way it provided a flexible and cost effective way to present unlimited experimental paradigms.

2.7 *Other visual motion sickness experiments*

2.7.1 Introduction

This section presents some additional motion sickness work based entirely or in part on visual stimuli. These are studies which do not easily fit into the above sections but which have relevant findings.

2.7.2 Sudden deceleration during on-axis rotation

Lackner *et al.* (1979) performed two experiments to evaluate the influence of vision on motion sickness during constant patterns of vestibular stimulation. The stimulation consisted of accelerating subjects from rest at $20°/s^2$ to 300°/s clockwise, maintaining them at 300°/s for 30 seconds, and then rapidly decelerating them to a stop in 1.5 seconds. During exposures, subjects were able to see the room in which the experiment was conducted. It was found that subjects tolerated fewer sudden stops when they had their eyes open for the duration of the exposure. They were able to tolerate more sudden stops when they had their eyes closed only during the sudden stop, and were generally found to suffer from significantly fewer symptoms of motion sickness if they had their eyes closed at any stage of the motion profile. The part of the motion causing the most discomfort to subjects was the sudden stop stage of the stimulation. During the constant velocity stage, there may be no movement of the fluid in the semicircular canals of the subject, which are thought to respond to acceleration at very low frequencies of head movement (see Section 2.2.1). During the sudden stop, the semicircular canals would be signalling changes in angular velocity in the anti-clockwise direction. The experimenters measured a nystagmus with a fast phase to the left, during and after the sudden stop as would be expected from the sudden vestibular signal. This would be in conflict with the pattern of visual stimulation which would still show a clockwise decelerating motion during the stop, and would be stationary after the stop. This was the major source of conflict and sickness among subjects, as they perceived the world to be still turning, even after motion had stopped, and for as long as their nystagmus continued. It was also found that shutting the eyes during the constant velocity period resulted in a rapid reduction of nystagmus and, in some cases, the nystagmus had ceased completely during the constant velocity period. During the periods of sudden deceleration the pattern of visual motion on the retina would have been similar to that experienced in the

optokinetic drum at high velocities (Hu *et al.,* 1989) where the images were slipping over the retina at fairly high velocities. This occurs because nystagmus is occurring with a slow phase to the right despite the motion of the world to the left. The slipping of images on the retina would be highest during the deceleration and persist for some time after the subject had come to rest. This may explain some of the increased motion sickness occurring when the eyes were open.

2.7.3 Nauseogenicity of head-mounted displays versus computer monitors

Howarth *et al.* (1996) compared the nauseogenicity of a head-mounted display with that of a computer monitor, in both cases used simply as a display device with no head tracking. The visual presentation was simply a game of computer chess which the participants played for one hour. It was found that there was a highly significant difference in motion sickness incidence, with more sickness found using the head mounted display. The authors suggest that the motion sickness arises through the conflict caused by head movements occurring without any corresponding motion of the visual scene on the head-mounted display. This is probably true, in the sense that the visual information would slip on the retina during head movements because the eyes will move in response to the head motion (vestibulo-ocular reflex) which will cause a slipping of images on the retina because the visual scene remains stationary with respect to the head.

2.7.4 Motion sickness reduction found with prism spectacles

Vente *et al.* (1998) report an interesting phenomenon whereby children who were prescribed prism spectacles according to a principle known as the 'Utermöhlen method' found a reduction in the motion sickness symptoms experienced during day-to-day car travel. The prism glasses were originally designed to treat people with Ménières disease but were also found to improve the mechanical reading ability of children with learning problems. The study was not concerned with motion sickness exclusively but was one part of a wider questionnaire concerned with the differences found before and after prescription of the prism glasses. The findings were triggered by the spontaneous reports of reduced motion sickness symptoms which were very common among the children who were treated. The questionnaire responses were found to indicate that nausea and incidence of vomiting was reduced after

prescription of the prism glasses. This study was unable to produce any statistics from the particular questionnaire that was used, or precise details on the design of the prism glasses, but is included here as an interesting curiosity.

2.8 Discussion

This literature review has studied three distinct areas of research: eye movements (optokinetic nystagmus, the vestibulo-ocular reflex), vection and motion sickness. The objective of this review was to bring the key elements from within each of the subject areas together in order to increase understanding of the phenomenon of motion sickness in response to moving visual stimuli.

One area of apparent significance which appears in many studies of optokinetic nystagmus and also in the study of the vestibulo-ocular reflex is the difference found in eye movements with peripheral or with foveal stimulation. Section 2.3.7.2 explained the research into optokinetic nystagmus which showed, in a variety of experiments, that the gain of nystagmus is higher when the fovea is stimulated and that small stimuli in central vision are adequate for high gain nystagmus, providing stationary edges are not visible if the field of view is restricted. It was also shown by Muratore *et al.* (1979) that 'after-nystagmus' can be generated with a single point of light tracked by the fovea which has similar characteristics to the after-nystagmus generated by a full field optokinetic drum.

The foveal dominance idea is further extended by the study of fixation. Nystagmus can be completely suppressed by the action of fixating on a small cross in front of a moving optokinetic drum (Stern *et al.* 1990). Stationary vertical edges close to the fovea also act as fixation points and suppress eye movements. The very fact that eye movements can be suppressed by focusing the fovea on a small part of the visual field which is stationary, whilst there is rapid motion elsewhere all over the peripheral visual field indicates that the fovea can dominate the control of eye movements. This also makes sense in a logical analysis of the purposes of eye movements – animals with foveas must be able to fix on an object of interest which they want to track the motion of, for example a bird flying past, whilst ignoring the consequential motion of the background moving in the opposite direction on the periphery.

The idea of foveal dominance also appears in the study of the vestibulo-ocular reflex, although the results are somewhat less clear cut. Studies presented in Section 2.3.4 showed that the vestibulo-ocular reflex gain could change in response to optokinetic motion without movement of the subject. It was shown that even if there was only a small point source of light moving to stimulate the fovea this could result in similar gain changes as found with a wider field of view. Retinal slip velocity (in particular foveal slip velocity) was proposed as the error signal used to drive the adaptation process (Shelhamer *et al.,* 1994). Vestibulo-ocular reflex gain adaptation was reduced by fixation, although not completely abolished, which may indicate that peripheral motion may also act as an error signal. In the strobe light experiments of Melvill-Jones *et al.* (1981) adaptation was found to be minimised under strobe conditions which would have severely limited the occurrence of retinal slip. Some adaptation was found at very low frequencies which may indicate that position data formed by discrete images falling on the retina can be used to drive the adaptation process at these low frequencies, where the appearance of images would be predictable.

A similar process could be occurring in these studies of vestibulo-ocular reflex gain adaptation as is happening in optokinetic nystagmus, whereby the foveal influence dominates but, in the absence of foveal input, the peripheral field can influence and control eye movements with lower precision (i.e. lower gain). Stimulation of the fovea alone can completely control the process of adaptation to similar levels found with full field stimulation (Shelhamer *et al.,* 1994) but in the absence of foveal slip the peripheral field does also appear to have an influence to a lesser extent (Demer *et al.,* 1989, Lisberger, 1983).

Vection was discussed in Section 2.4. It has not been shown in the literature that there is a direct correlation between vection and motion sickness. It is implied in the sensory conflict theory of Reason and Brand (1975) that motion sickness with visual stimuli occurs as a result of a conflict between actual and perceived motion cues. This has probably influenced a number of authors to believe that vection and motion sickness are related as cause and effect. It is implied in many papers that this may be the case but without any direct evidence provided to back up the claim. It is not enough merely to state that the condition with the most vection also provoked the most motion sickness without providing individual correlations between subject vection and motion sickness scores. There do not appear to be any correlations of this nature present in the literature. The factor which has been shown to affect

vection perception appears to be the field of view of the display and specifically whether or not the peripheral visual field is stimulated. Vection has been shown to be dominated by peripheral vision (Brandt *et al.,* 1973).

Motion sickness experiments using optokinetic drums (as presented in Section 2.6.3) show that motion sickness is reduced with fixation (Stern *et al.,* 1990; Prothero *et al.,* 1999). This may be due to the reduction of eye movements (Ebenholtz, 1994) or due to the reduction of image slip on the fovea. Pinpointing which of these two possibilities has the most influence on motion sickness is an area for further research. The reduction of image slip on the fovea may be the most likely because it can help to explain not only the effect of fixation but also the increase in motion sickness with increasing drum speed, where foveal slip increases at higher speeds. It also seems logical from the results of experiments with magnified vision whereby motion sickness symptoms and ataxia are reduced after a subject has successfully adapted their vestibulo-ocular reflex gain to the magnification factor of the glasses. In this case foveal slip would occur until the point at which adaptation had fully occurred. Eye movements before and after adaptation would not be greatly different, except with very high levels of magnification. As discussed above, retinal slip and particularly foveal slip velocity, appears to be the main error signal used for the vestibulo-ocular reflex gain adaptation, so it is a possibility that motion sickness is influenced in some way by the amount of foveal slip.

The significance of the dominant influence of the fovea on eye movement control raises the intriguing possibility that a subject's visual acuity, or contrast sensitivity to high spatial frequencies, may influence their eye movements. Visual acuity is effectively a measure of the quality of the fovea in resolving fine detail. If the foveal acuity is low, perhaps the fovea has less influence on eye movements? If eye movements are in some way influencing motion sickness then the possibility emerges for a mechanism by which a subject's visual acuity could affect motion sickness, via the proposed influence on eye movements. By measuring subject's visual acuity it may be possible to find out if there is any influence of visual acuity of motion sickness and in what way it has an effect. Post *et al.* (1979) found no variation in slow phase eye movements with the addition of blurring lenses, but did not match the stimuli for velocity (see Section 2.3.7.3).

Visual influences on motion sickness appear to occur in other traditional forms of motion sickness research. The action of anti-motion sickness drugs on the gain of

eye movements, together with the finding of lower vestibulo-ocular reflex gain responses in subjects who were less-susceptible to ship motion, should be treated with caution. They may allow a possible route for visual influences to enter more traditional motion sickness, as part of a much wider picture. It is beyond the scope of this thesis to study this in detail but it could provide avenues for research in the future.

The main conclusions to be drawn from this review are that vection and motion sickness need to be measured separately in order to confirm whether they are mechanisms which are linked or occur independently. The visual characteristics of subjects need to be known, and eye movements should be recorded wherever possible, to ensure that the potential influences of visual characteristics and eye movements are controlled or quantified.

2.9 Model of the factors influencing optokinetic motion sickness

A model of the slow phase of nystagmus by Robinson (1981) was presented in Section 2.3.7.3. Robinson's model acts as the basis for the model presented in this chapter (Figure 2.22). Head movements have been removed to simplify the model, because exposure to optokinetic drums usually involves a completely stationary subject, often with the head immobilised. Head movements can be re-introduced if necessary.

There was a single input into the model presented in Section 2.3.7.3. The model presented in this section has two inputs, in order to allow the foveal and peripheral retina to be viewing different velocities of visual motion (e.g. during fixation, where velocity is zero on the fovea, but not zero on the periphery of the retina). The two inputs are 'foveal image velocity' (the angular velocity of the image which is tracked by the fovea – in °/s) and 'peripheral image velocity' (the angular velocity of the image which is viewed by the peripheral retina). In the case of fixation, the foveal image velocity is zero. For an optokinetic drum with a stationary subject and no fixation, both inputs are equal to the speed of the drum. The two summing junctions on the left hand side of the model show that the angular velocity of image slip on the fovea and on the peripheral retina are found by taking the difference between the angular image velocities (foveal and peripheral) and the angular velocity of the eye movements (E).

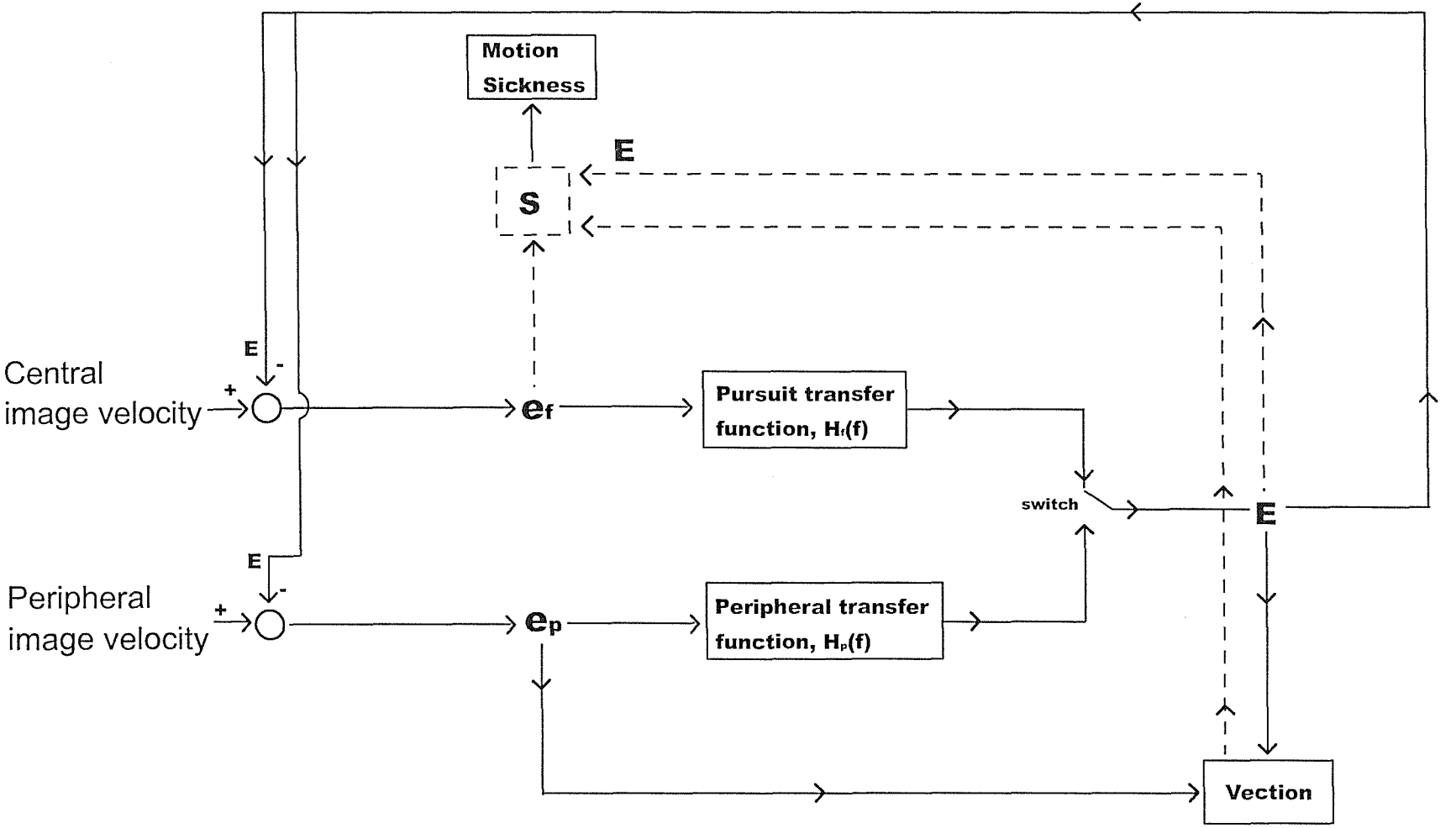

Figure 2.22. Initial model, version 1. Based on Robinson (1981).

The model is a simple negative feedback system, by which an increasing eye velocity acts to decrease foveal slip velocity.

The 'foveal pursuit transfer function' is shown to generate a slow phase velocity signal, from the foveal slip velocity detected, to control the eyes. The 'peripheral tracking transfer function' is also shown to generate a slow phase velocity signal from the peripheral image slip velocity detected. Under most circumstances the foveal pursuit transfer function dominates the control of the slow phase velocity (see Section 2.3.7.2). The switch allows either the 'foveal pursuit transfer function' or the 'peripheral tracking transfer function' to dominate the eye movement velocity (E). This is normally switched to the foveal path but, in the case of artificially blocking the fovea, or central retinal scotoma (e.g. Van Die *et al.,* 1986), the peripheral path can be used. The peripheral control of eye movements has been shown to have a lower gain response compared to the foveal response, hence if the peripheral system is dominating eye movements, slow phase velocity (E) will be lower.

Vection was shown to be dependent on the velocity of the drum (Graaf, 1990) and also controlled mainly by the detection of motion on the peripheral retina (Brandt *et al,* 1973). This is shown by modelling vection as dependent on peripheral image slip velocity and the slow phase velocity of nystagmus. The velocity of the drum can be calculated from peripheral image slip velocity and eye velocity by the equation:

$$D = E + e_p$$

D is the velocity of the drum, e_p is peripheral image slip velocity and E is the eye movement velocity. It can be seen that, for a constant drum velocity (D), an increase in eye velocity (E) will reduce image slip (e_p), or that a reduction in eye velocity will increase image slip. Hence the hypothesis that vection will be dependent on the velocity of the drum also generates the hypothesis that vection will be independent of slow phase velocity.

Motion sickness is added into the model with 3 possible inputs: (i) vection, which is shown with a dotted input line to show that it is uncertain (ii) eye movements (E) themselves, which can be decreased by fixation (decreasing motion sickness) and (iii) foveal image slip (e_f) which is also decreased by fixation and hence reduces motion sickness. 'S' is the unknown mechanism by which motion sickness arises from one or all of the possible inputs: vection, eye movements or foveal image slip.

Inspection of the model shows that the three potential inputs into motion sickness cannot all be true. It was shown that vection is assumed to be independent of eye movements in this model, hence vection and eye movements cannot both influence motion sickness. The experimental work will help to discover whether vection, eye movements or foveal image slip are the most important factors in the influence of motion sickness and to verify whether vection is truly independent of eye movements.

2.9.1 Influence on the first experiment

The model shows an uncertain link between vection and motion sickness. Recording detailed vection ratings and motion sickness scores each minute will allow the two to be tested for correlations, among subjects. If vection is not found to be an influence on motion sickness, experiments which directly test the other possible routes (of eye movements and foveal image slip) can be developed.

Chapter 3. Apparatus and experimental procedure

3.1 Introduction

This chapter describes the apparatus used in the experimental work presented later in the thesis, including the optokinetic drum and virtual reality apparatus used to display moving images to subjects. Other experimental procedure is also described.

3.2 Moving image systems

3.2.1 The optokinetic drum

The optokinetic drum was a cylinder of 1m diameter and 1.2m high supported by a steel frame and counter-balanced by a 90 kg weight. The inside of the drum was covered with alternate black and white stripes each subtending approximately 8° at the subject's eyes. The drum was lit by a 12V, 20W halogen bulb, located at the centre of the drum 20cm below the roof of the drum. The seat of the drum could be raised or lowered in order to ensure that subjects were level with the centre of the drum.

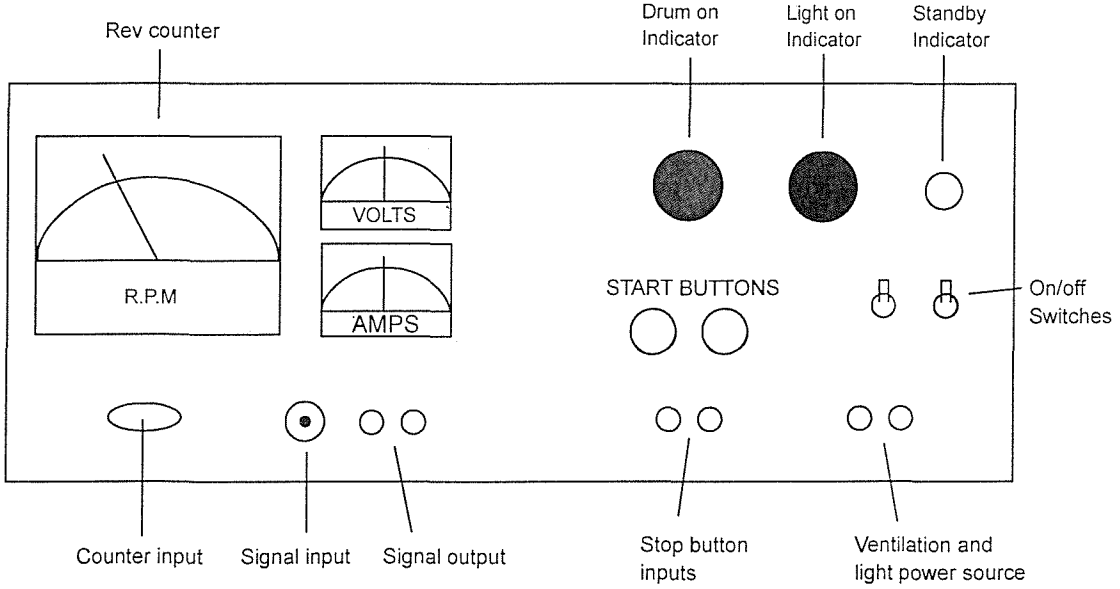

Figure 3.1. The optokinetic drum control box.

3.2.1.1 The drum controls

The optokinetic drum was controlled by a unit specially built in the Human Factors Research Unit. This allowed control over the speed of the drum and also contained two safety features to ensure that accidental operation was not possible and that the drum could be stopped quickly when desired (i) The drum could only be started by pressing two buttons simultaneously (see Figure 3.1) (ii) both the experimenter and subject had an emergency stop button which immediately halted the drum, if pressed. Motion input to the drum was from a standard signal generator, which was used to generated a constant speed of drum rotation in the clockwise direction (as seen from above) for the three occasions when the drum was used in this thesis, but could be used to create sinusoidal motion if necessary.

Figure 3.2. A subject shown sitting in the optokinetic drum seat. The drum is in the raised position to allow access to the seat. The head restraint is not shown.

3.2.1.2 Seating

The seat was situated so that the subject's head was situated at the centre of the drum. The wooden backrest was 1.2 m high and had three slits cut into it – one in the centre of the backrest and two 15cm on each side of the centre slit. The two slits on each side allowed a strap to be placed around the head of a subject to minimise head movements in the drum. A subject is shown in the seat of the drum, with the drum in the raised position in Figure 3.2.

3.2.1.3 Ventilation

The drum contained a ventilation system consisting of a 12W fan and a ventilation tube which drew air into the drum from the room in which the drum was situated. The tube was fixed to the back of the seat backrest so that the end of the tube was level with the top of the seat. Drum temperature typically varied by 1° during the course of an experiment when using the ventilation compared to a variation of 3-4° without the ventilation (Holmes, 1998).

3.2.1.4 Monitoring

It was possible to monitor subjects inside the drum by placing a small video camera on the floor, pointing up into the drum and relaying images to a video screen outside the drum. In this way it was possible to ensure that subjects had their eyes open during exposure.

3.2.1.5 Luminance and contrast of the stripes

The luminance of the stripes with the optokinetic drum in its down position was measured using a Minolta luminance meter. The luminance of the black stripes was 1.44 candelas/m^2. The luminance of the white stripes was 31.28 candelas/m^2.

There are many different ways to express contrast. The following are two of the common ways. It is possible to use the above luminance values to calculate any other measure of contrast if necessary. The contrast ratio (maximum luminance divided by minimum luminance) was 21.72. Modulation contrast (or Michelson contrast) was 0.91 (max – min / max + min).

3.2.2 The virtual reality system

This consisted of a Virtual Research VR4 head mounted display. This model displayed moving images that were the same as that seen on a computer monitor by sampling the image sent to the monitor (Deltascan pro system). Full screen Microsoft AVI video files could be displayed on the computer using Windows Media Player and hence also seen by the subject on the virtual reality display. The same images were always presented monocularly, that is the same image was seen by each eye simultaneously. Figure 3.4 shows a diagram of the connections between computer and the virtual reality system.

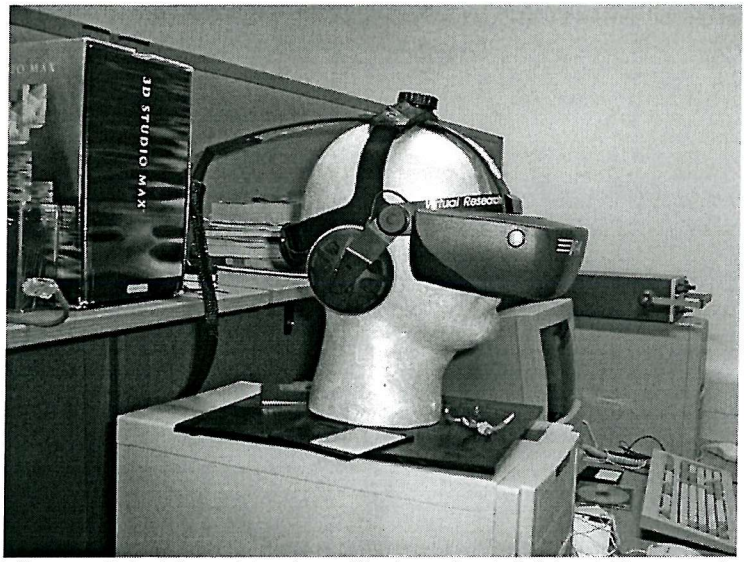

Figure 3.3. The virtual reality headset (Virtual Research VR4).

The VR4 headset had a field of view of 48° horizontally by 36° vertically and a focal point of approximately one metre. The distance between two eye-pieces could be adjusted by the subject to match their inter-pupillary distance. The Virtual Research VR4 head-mounted display is shown in Figure 3.3.

Video file production was carried out using Kinetix' 3D Studio MAX software version 1.2. This software allowed video files to be created of any object with any material, texture or colour applied to the object. In the case of creating a simulation of an optokinetic drum, a cylinder was created with a black and white striped texture applied. A 'virtual' camera was placed at the centre of the drum and a series of key-frames were created with the drum at different angular positions. The video file was created automatically by the software, where each frame was calculated with reference to the key-frames (i.e. the position of the drum at each frame was extrapolated from the key frames). The result was a video file of moving black and white stripes as would be seen in a real optokinetic drum. The video files were all

created at 60 frames per second. The video files were played back to subjects monocularly (both eyes saw the same image sequence) on the Virtual Research VR4 virtual reality head mounted display. The advantage of playing back pre-prepared video files was that the experimenter could control exactly what was seen by subjects

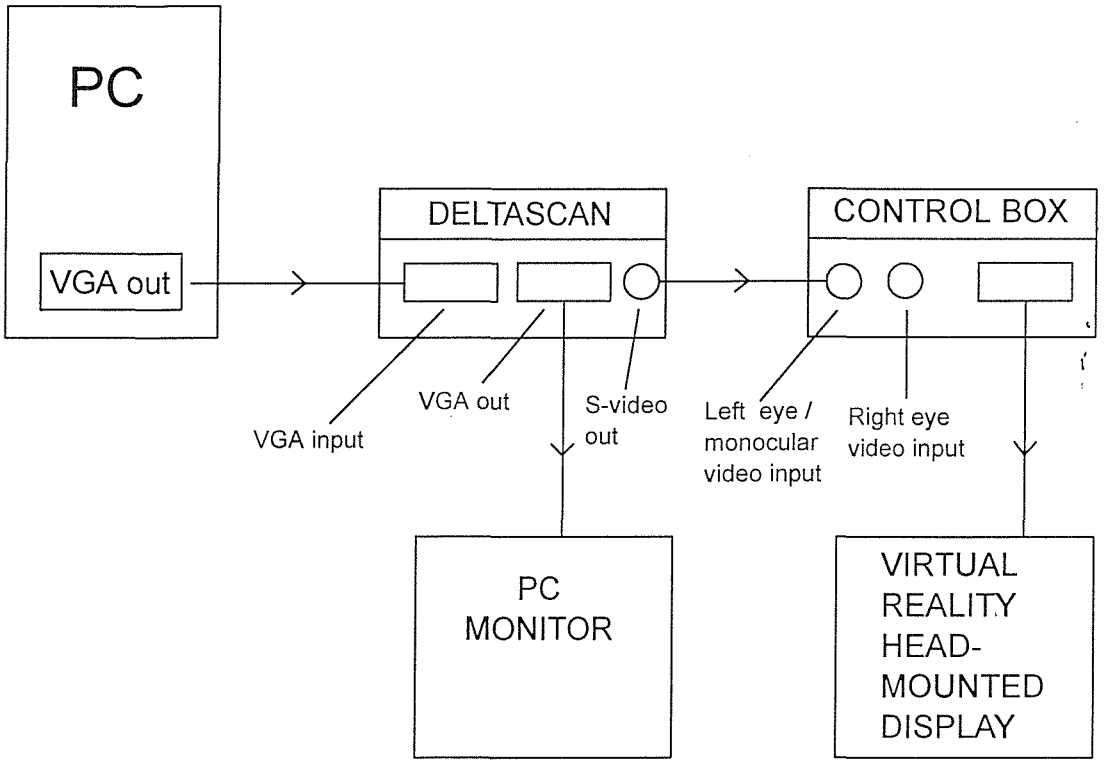

Figure 3.4. Diagram of the connections between the computer (PC), Deltascan (video signal sampler), the virtual reality head-mounted display and computer monitor. The Deltascan system samples the video output from the PC and sends copies to the computer monitor and the virtual reality system.

and there were no problems associated with virtual reality displays such as time lags in the updating of images where head movements are made.

3.2.3 Luminance and contrast of the stripes

In the virtual reality simulations of the optokinetic drum, the luminance of the black stripes was 1.65 candelas/m^2 and the luminance of the white stripes was 30.53 candelas/m^2. The contrast ratio was 18.5 and the modulation contrast (Michelson contrast) was 0.90. Luminance was measured by focusing the Minolta luminance meter through the eyepiece of the virtual reality head-mounted display. For the purposes of the measurement the whole screen was either filled a single black or white stripe to ensure that the luminance meter was focusing on the correct colour.

3.3 Vision testing equipment

Vision tests were completed using two pieces of equipment (i) Keystone visual skills profiles (ii) The Arden test of contrast sensitivity.

3.3.1 Keystone visual skills profiles

This equipment allowed a variety of visual tests to be performed on subjects at two viewing distances, 0.4 metres (2.5 dioptres – 'the near point') and 4 metres (0.25 dioptres – 'the far point'). The tests consisted of various cards which were inserted into the card holder individually. The tests used included tests of simultaneous perception (to determine whether both eyes are used at the same time), vertical and horizontal muscle balance tests, which indicated whether there was a tendency for one eye to drift higher than the other (vertical hyperphoria), for the eyes to cross (esophoria) or to not converge at the correct distance (exophoria). There were also tests of colour perception, to indicate the presence of colour blindness and tests of visual acuity, which used the Landolt broken ring test. The visual acuity tests were performed binocularly and with each eye separately.

Separate testing cards were used for the near and far points. Figure 3.5 shows a subject using the Keystone system, set at the far point.

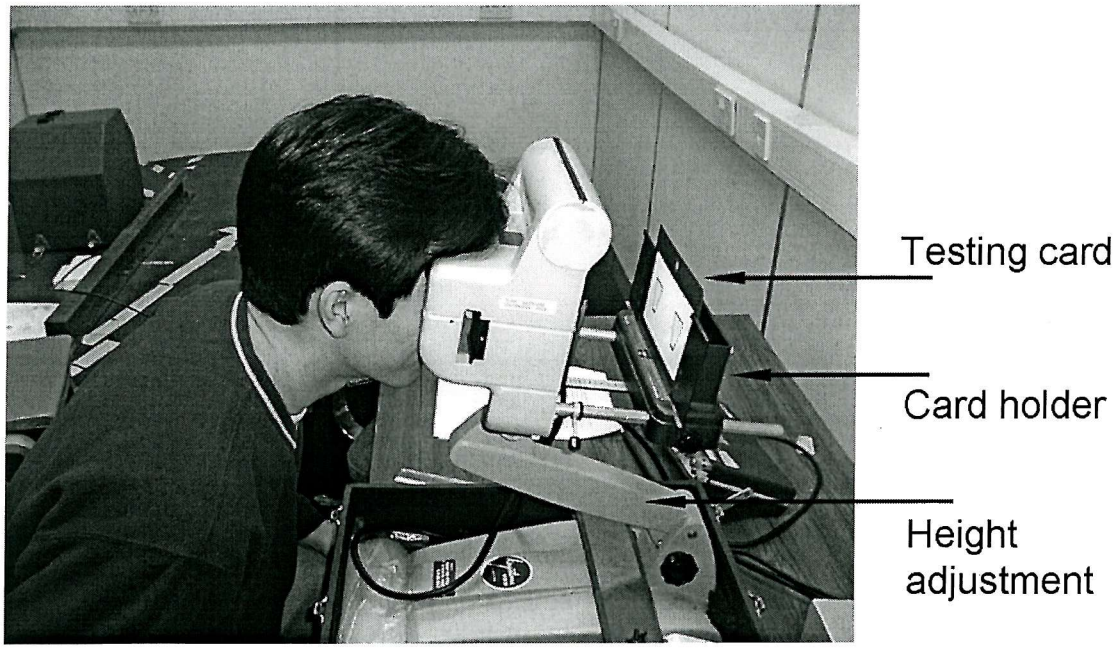

Figure 3.5. A subject undergoing vision tests with the Keystone visual skills profiles testing equipment. Card holder is set to the 'far point'. The holder can be moved towards the subject to the 'near point'.

3.3.2 The Arden test of contrast sensitivity

A test known as the "Arden Test" was used in order to obtain information about the contrast sensitivity of subjects to a broad range of spatial frequencies, not just sensitivity to high spatial frequencies at maximum contrast, as measured by the visual acuity tests used in the Keystone visual skills profiles.

In the Arden test, a card was slowly removed from a holder. Each card had a sinusoidal variation across the card of grey to black. The contrast increased as the card was removed from a holder, up until the point at which a subject could see the difference in contrast (i.e. the card no longer looked grey all over). At the point at which the card was stopped, a number was read off the edge of the card to indicate the contrast sensitivity to that particular spatial frequency. The spatial frequencies used were 0.3, 0.6, 1.25, 2.5, 5 and 10 cycles per degree, when viewed at 0.50 metres, as per the Arden test instructions. An example of a card is shown in Figure 3.6.

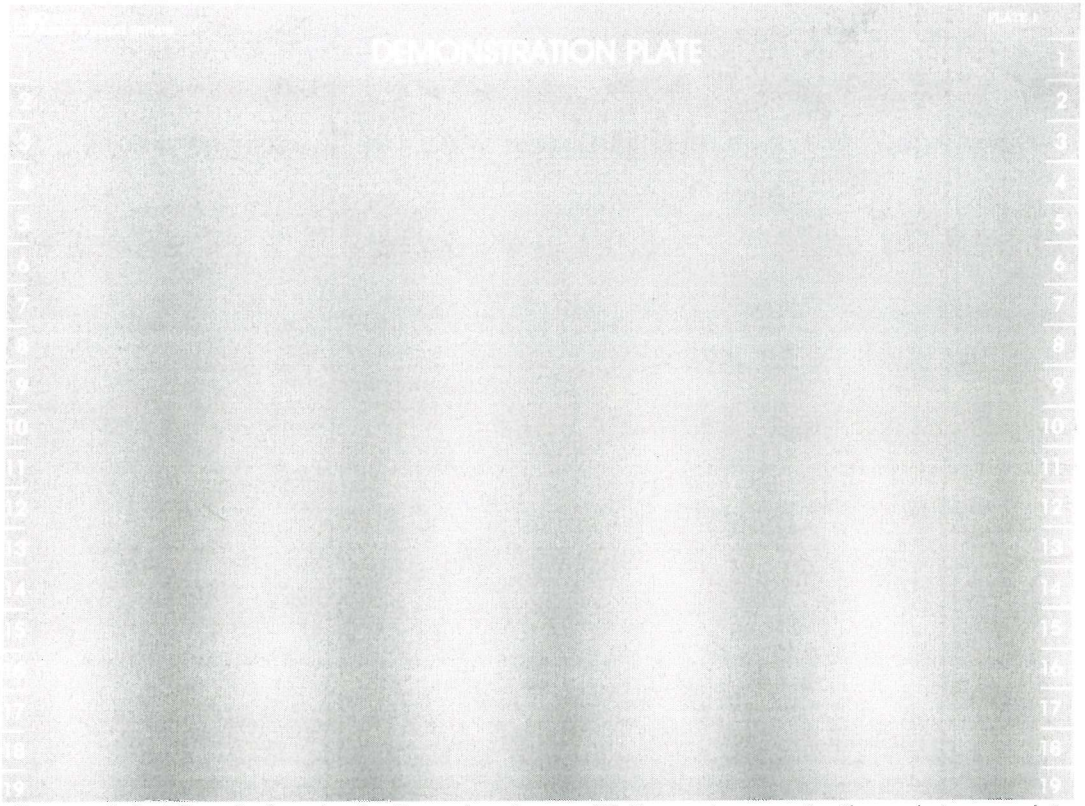

Figure 3.6. The Arden test of contrast sensitivity – demonstration plate used to demonstrate the test to subjects. The difference in contrast is exaggerated on this demonstration card.

3.4 Eye movement measurements

3.4.1 Electro-oculography measurements

Eye movements were recorded in experiments 2 and 4 (Chapters 5 and 7) by the means of electro-oculography. The connections between the equipment used are shown in Figure 3.7. Three disposable electrodes were attached to each subject, the positions of which are shown in Figure 3.8. The signal from the electrodes was sent to a device called the 'Hortmann electro-nystagmograph' which was used to amplify the signal. The amplified signal was then sent to an *HVLab* data acquisition computer

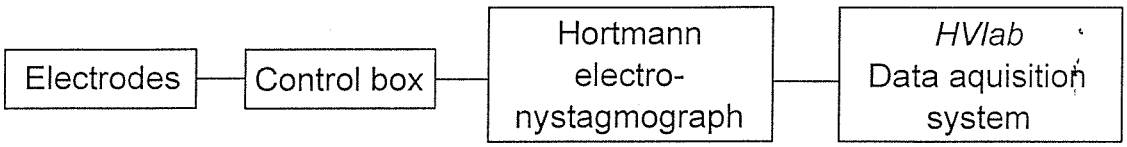

Figure 3.7. Diagram to show the connections of equipment for electro-oculography measurements. Eye displacement data was sampled at 30 samples per second, with a low pass filter at 10Hz.

(built at the Human Factors Research Unit at the University of Southampton) which digitally sampled the signal at a rate of 30 samples per second with a low pass filter at 10Hz. Each signal could be viewed and analysed using the *HVLab* software. The accuracy of electro-oculography recordings is in the region of 0.5-1.0 degree of visual angle (Hallett, 1976).

Eye movements were calibrated by asking subjects to look at 3 crosses marked horizontally on a wall in front of them. The first cross was directly in front of the subject (between the two eyes) and the other crosses were at 15° visual angle symmetrically either side. Subjects made eye movements between the crosses at the verbal request of the experimenter. The calibrations were also recorded to the *HVLab* data acquisition system.

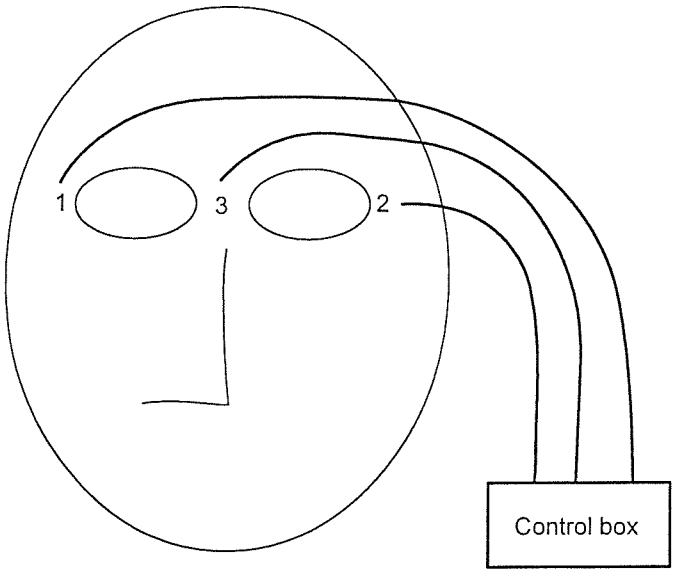

Figure 3.8. Shows the position of the electrodes on a subject for the electro-oculography measurements.

3.4.2 Infra-red light eye movement measurement (IRIS)

The final experiment of this thesis, presented in Chapter 9, required a more accurate measurement of eye movements than those which could be achieved with standard electro-oculography measurement techniques. The experiment used a system from the company 'Skalar Medical' called IRIS (infra-red light eye-movement measurement) which has a measurement range of 25° horizontally and 20° vertically, with an accuracy of 1 minute of visual arc (Reulen *et al.*, 1988). The system consisted of an emitter and sensor which are positioned in front of the eye (Figure 3.10 shows the sensor placement). The varying reflection of the eye, as it moves, is detected by the sensor and an output voltage proportional to displacement of the eye is generated. A subject wearing the measurement device is shown in Figure 3.13, front and rear panel controls are shown in Figure 3.11 and 3.12.

The output from the IRIS system was sent to an *HVLab* computer system and the displacement signals for the left and right eyes were sampled at a rate of 300 samples per second, with a low-pass filter cut-off at 100Hz. The equipment connections are shown in Figure 3.9.

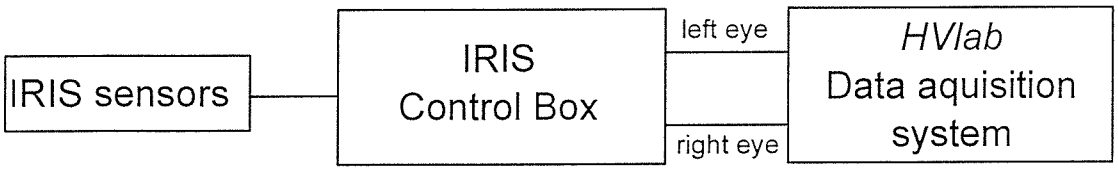

Figure 3.9. The equipment connections for the IRIS system. Eye displacement data was sampled at 300 samples per second, with a low pass filter at 100Hz.

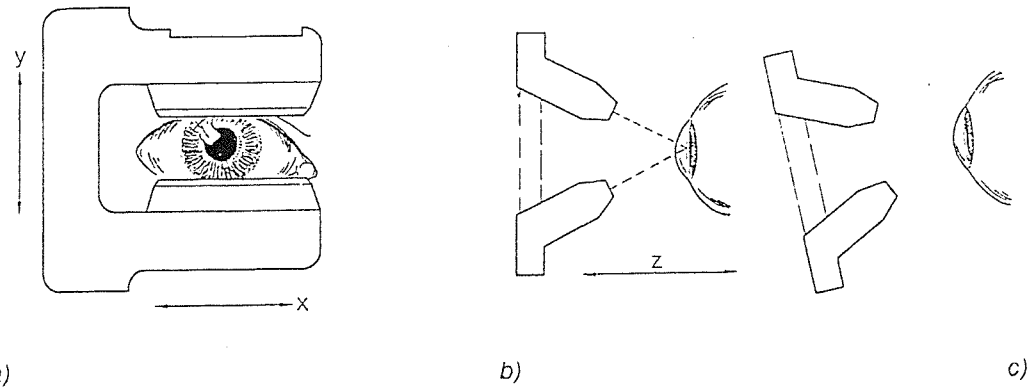

Figure 3.10. Horizontal sensor adjustment. (a) front view (b) side view (c) alternative adjustment.

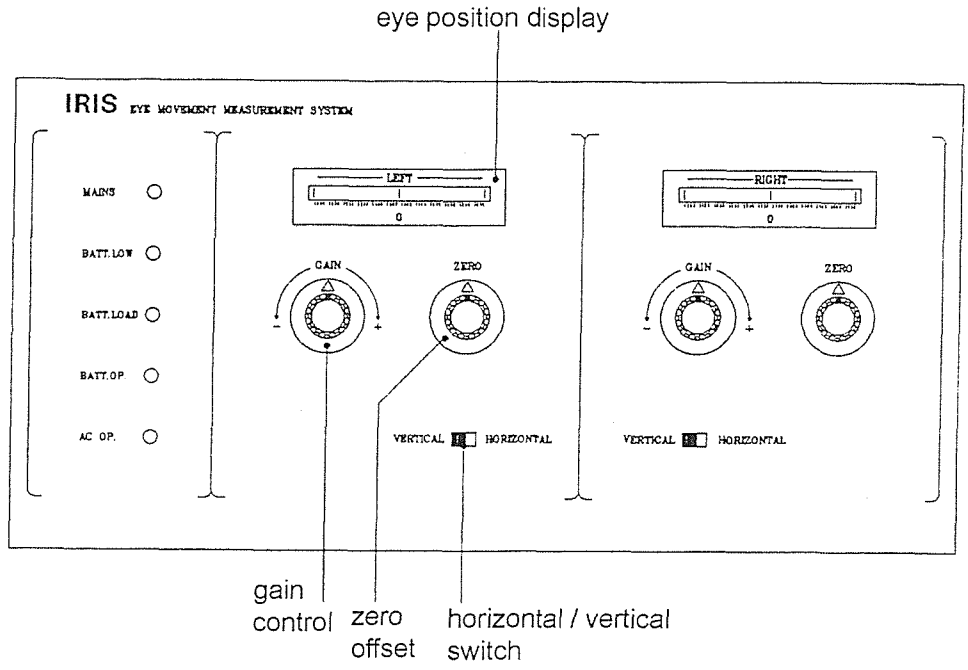

Figure 3.11. The front panel controls of the IRIS system.

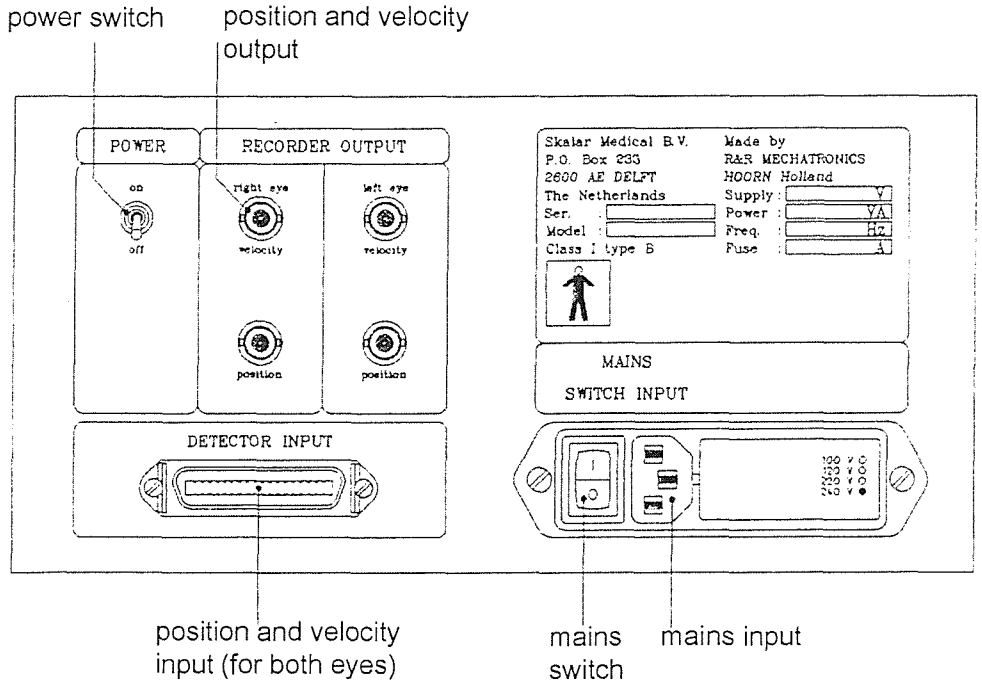

Figure 3.12. The rear panel controls of the IRIS system.

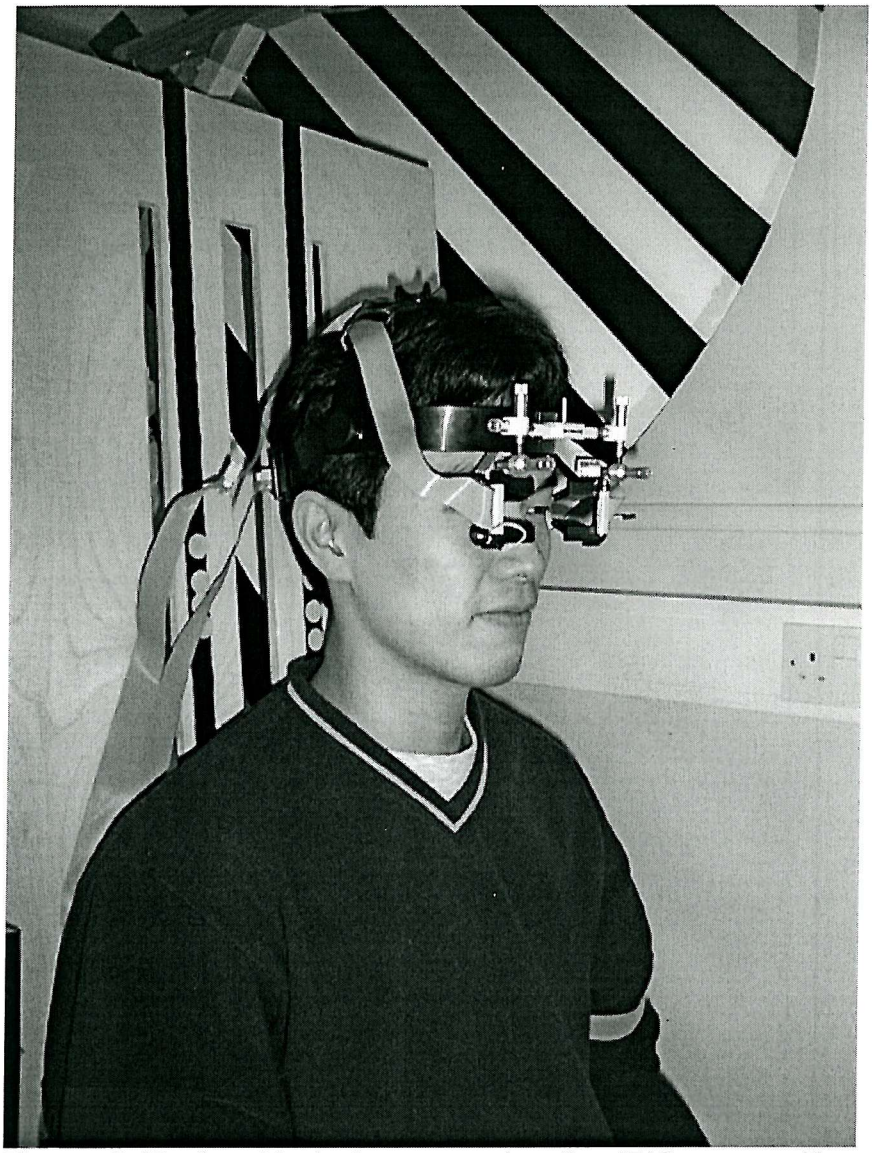

Figure 3.13. A subject shown wearing the IRIS eye position sensors.

3.5 Other experimental procedure

This section gives details on the subjective scales which were used in the experimental work for subjects to report their symptoms of motion sickness, their perception of self-motion (vection), the questionnaires used to rate their post-exposure symptoms and the questionnaire to measure their previous susceptibility to motion sickness in standard forms of transport (e.g. cars, buses, ships).

3.5.1 The subjective motion sickness rating scale

During each of the experiments presented in the later chapters of the thesis subjects reported a number from the subjective rating scale in Table 3.1 to indicate their subjective symptoms of motion sickness at that time. The scale is based on a scale by Golding and Kerguelen (1992). Since motion sickness symptoms do not necessarily occur in any particular order subjects were able to report any number on the scale at any time. Accumulated illness ratings were calculated, after exposure, by summing the motion sickness ratings reported each minute.

Table 3.1. The subjective motion sickness rating scale. (Golding and Kerguelen, 1992). Subjects report a number each minute for the duration of the exposure.

Subjective Response	Corresponding Feeling
0	No symptoms
1	Any symptom, however slight
2	Mild symptoms e.g. stomach awareness, but no nausea
3	Mild nausea
4	Mild to moderate nausea
5	Moderate nausea, but can continue
6	Moderate nausea, want to stop

3.5.2 Subjective vection rating scales

The scale shown in Table 3.2 was used to record subjective self-motion ratings each minute. The scale was designed to indicate common perceptions of self-motion, such as whether a subject felt like the optokinetic drum was the only thing moving, whether the subject felt like the drum was moving and also experienced self motion intermittently, continuously or whether the subject perceived continuous self-motion whilst perceiving a stationary optokinetic drum. Accumulated vection scores were calculated by assigning a value of 0 to 'Drum only', 1 to 'Drum and self, intermittent', 2 to 'Drum and self, continuous' and 3 to 'Self only'.

Table 3.2. Subjective vection rating scale. Subjects reported one of the following options each minute for the duration of the exposure.

Perception of what is moving	You report:
Drum Only	You perceive that the only thing moving is the drum (real or virtual).
Drum and Self (intermittent)	You perceive the drum to be moving but also experience periods of self motion.
Drum and Self (continuous)	You perceive the drum to be moving and simultaneously experience continuous self motion.
Self Only	You perceive the drum to be stationary and experience continuous self motion only.

The subjective vection rating scale was used in the first three experiments presented in this thesis (Chapters 4 to 6). It was used for both the real optokinetic drum and for simulated optokinetic drums presented on the VR4 virtual reality head-mounted display.

In the fourth experiment an optokinetic drum simulation was not used, so a different vection rating scale was created. Shown in Table 3.3, this scale was a percentage scale where 0% indicated no vection (i.e. only the visual stimulus was perceived to be moving). An increasing percentage score indicated increased vection, for example 50% indicated that the subject perceived the stimulus and themselves to be moving at approximately the same speed (in opposing directions). 100% indicated that the subject felt that they were moving and the visual stimulus was stationary. Subjects could report any number between 0 and 100% at each measurement, made each minute. An average percentage score was calculated for each subject from the individual percentage vection scores.

3.5.3 Motion sickness history questionnaire

Before commencing an experiment, subjects were asked to complete a motion sickness history questionnaire (Griffin and Howarth, 2000) to indicate their previous susceptibility to motion sickness caused by the common forms of transport. The

Table 3.3. Vection scale for experiments 4 to 6. Subjects report a percentage score between 0 and 100% each minute to indicate their perception of self motion.

Perception of motion (vection)	You report:
You feel like you are stationary and it is the dot(s) which appear to be moving only.	0%
You feel like you are moving a bit, but the dot(s) are moving more	1-49%
You feel like you are moving at the same speed as the dot(s)	50%
You feel like you are moving a lot and the dot(s) are moving a bit	51-99%
You feel like you are moving and the dot(s) appear stationary	100%

questionnaire allows values to be calculated for susceptibility in the previous year ($I_{susc.(yr.)}$), total susceptibility in all previous years (M_{total}) or, if necessary, susceptibility to land or non-land transport could be calculated separately. The full questionnaire is shown in an Appendix of this thesis.

3.5.4 Post-exposure rating scale

After exposure, subjects were asked to complete a post-exposure symptoms questionnaire to indicate the symptoms which they had experienced at any time during the exposure to the moving stimulus. This post-exposure scale was used in the first five experiments (Chapters 4 to 8). Subjects were asked to fill in the questionnaire by ticking a response for each symptom of 'none', 'slight', 'moderate' or 'severe'. The symptoms 'difficulty focusing' and 'blurred vision' were removed from the questionnaire in the third experiment (Chapter 6) where an artificially blurred stimulus was presented to subjects in one condition. A score for each symptom was calculated for each subject by allocating a score of 0 for 'no symptoms', 1 for 'slight symptoms', 2 for 'moderate symptoms' and 3 for 'severe symptoms'. The individual values for each symptom were summed to give a total post-exposure symptom score.

Table 3.4. The post-exposure symptom questionnaire.

Symptom	None	Slight	Moderate	Severe
General Discomfort				
Fatigue				
Headache				
Eye Strain				
Difficulty Focusing				
Increased Salivation				
Increased Sweating				
Nausea				
Difficulty Concentrating				
Blurred Vision				
Dizziness				
Stomach Awareness				
Burping				

Chapter 4. Experiment 1. Comparison of motion sickness and vection in a real and virtual reality optokinetic drum

4.1 Introduction

The purpose of the first experiment, presented in this chapter, was to record vection and motion sickness scores in a standard optokinetic drum and in a virtual reality simulation of an optokinetic drum and to investigate correlations among subjects between vection and motion sickness scores.

Another objective of this experiment was to investigate whether circular vection and motion sickness could be generated using the restricted field of view of a virtual reality head-mounted display, and whether motion sickness ratings in this 'virtual drum' were correlated with those obtained with the same field of view in a standard optokinetic drum. Virtual reality allows flexibility in varying the visual display and has the potential to replace traditional optokinetic drums, mirror systems and other optical display devices (e.g. film projectors) that have been used to investigate motion sickness and vection.

It was predicted that, for individual subjects, the two environments would produce similar sickness. Consequently, across the group of subjects, it was hypothesised that there would be a correlation between sickness ratings obtained in the two conditions. It was also hypothesised that, within conditions, ratings of motion sickness would be correlated with ratings of vection.

4.2 Method

In part of the experiment, subjects were seated inside the optokinetic drum (as described in Chapter 3). A strap connected to the backrest of a chair restrained the head of each seated subject. Subjects wore spectacles designed to restrict their field of view to 48° horizontally and 36° vertically, which matched the field of view of the virtual reality display.

In the other part of the experiment, an animation of the optokinetic drum was

presented on the head-mounted display (Virtual Research VR4). The same sequence of images was presented to both eyes simultaneously. The animation was programmed so as to give a similar visual experience to the viewer as being in the real drum. Each black and white stripe subtended approximately 8° visual angle and the stripes moved across the screen at 30° per second, equivalent to 5 r.p.m. of the optokinetic drum. Subjects did not wear vision correction in either the real or the virtual condition.

Whilst watching the animation, subjects sat inside the real drum (which was rotating) so that the environment (i.e. sound, temperature and enclosed feeling) was similar in both conditions. The same system was used to restrain the head.

Sixteen male subjects, aged 20 to 28 years (mean 22.9 years) participated in the experiment. Visual acuity without correction was measured using the Keystone visual skills profiles (see section 3.3.1) conducted at the near point (2.5 dioptres, 0.4 m) and far point (0.25 dioptres, 4m). Prior to experiencing the visual motion, all subjects completed a motion sickness history questionnaire providing details of travel history and previous motion sickness experience (Griffin and Howarth, 2000). The responses were used to derive motion sickness susceptibility ratings for each subject.

All subjects experienced both the real and the virtual optokinetic drum for up to 30 minutes. Eight subjects commenced with the real drum and eight commenced with the virtual drum. There was at least one week between exposures to reduce effects of habituation. Subjects experienced each condition at the same time of day. At half-minute intervals during each exposure, subjects provided ratings on the 7-point motion sickness scale (Table 3.1) and on a 4-point vection scale (Table 3.2). Following each exposure, subjects completed a symptom checklist (see section 3.5.3).

Subjects gave their informed consent to participate in the experiment that was approved by the Human Experimentation Safety and Ethics Committee of the Institute of Sound and Vibration Research.

4.3 Analysis

The motion sickness ratings were summed over the 30-minute exposure period to give an 'accumulated illness rating' for each subject. If a subject terminated the

session (i.e. reached a rating of 6 on the motion sickness scale), a rating of 6 was assigned for the remaining period. Vection ratings were allocated scores: 0 for 'drum only', 1 for 'drum and self intermittent', 2 for 'drum and self continuous' and 3 for 'self only' (see Table 3.2). The 'accumulated illness ratings' and the 'accumulated vection ratings' were compared across conditions (i.e. between the real and the virtual drums) using the Wilcoxon matched-pairs signed ranks test. Correlations for 'accumulated illness ratings' across conditions, correlations between 'accumulated vection ratings', 'total illness ratings' and 'past susceptibility' within conditions were determined using the Spearman's rank correlation.

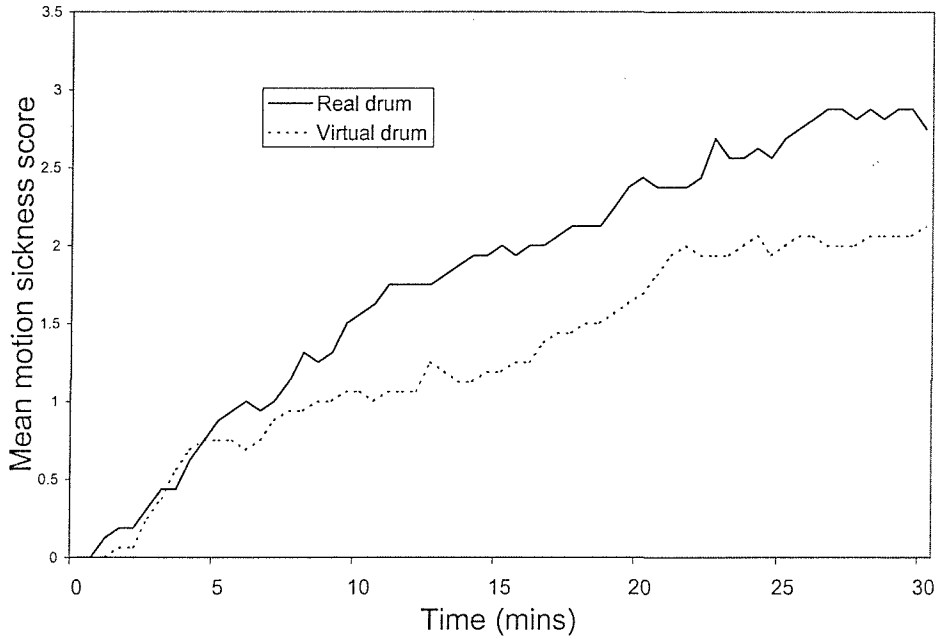

Figure 4.1. Mean motion sickness ratings for the real and virtual drum (motion sickness ratings in the real drum are greatest).

Additional analysis was carried out using subject survival times. The time taken for a subject to reach a rating of 2 ("mild symptoms e.g. stomach awareness but not nausea") on the motion sickness scale was used as the event of interest in this analysis. Initially, Spearman's rank correlation test was used to find significant interactions and then Cox regression analysis was used to determine more about the nature of the correlations found. Survival analysis using Cox regression was chosen as it allowed all data to be included in the analysis (e.g. a subject who withdraws from the experiment because of nausea could be included by analysing the time when a rating of 2 was reached), while taking into account the responses of subjects who did not reach a rating of 2. Subjects who withdrew from the experiment because

of nausea were included without having to make assumptions about sickness ratings at later times.

4.4 Results

There was no difference between the vection ratings in the two conditions (Wilcoxon, $p>0.10$). However, the accumulated illness ratings (summed over 30 minutes within the two conditions) differed significantly, with mean values of 38.9 in the virtual

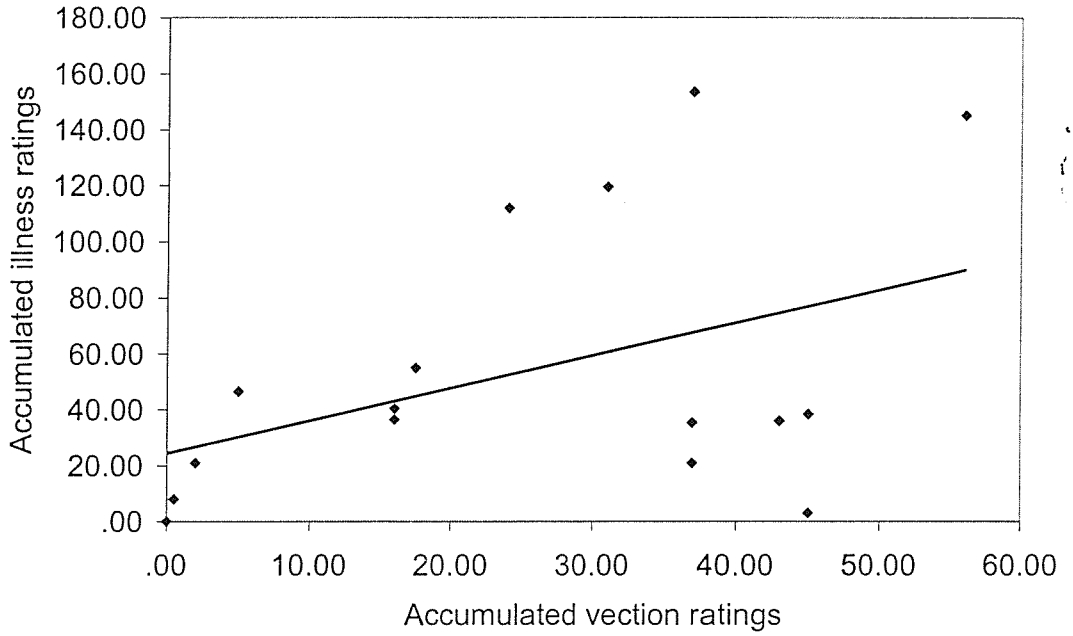

Figure 4.2 Accumulated vection and illness ratings – real drum

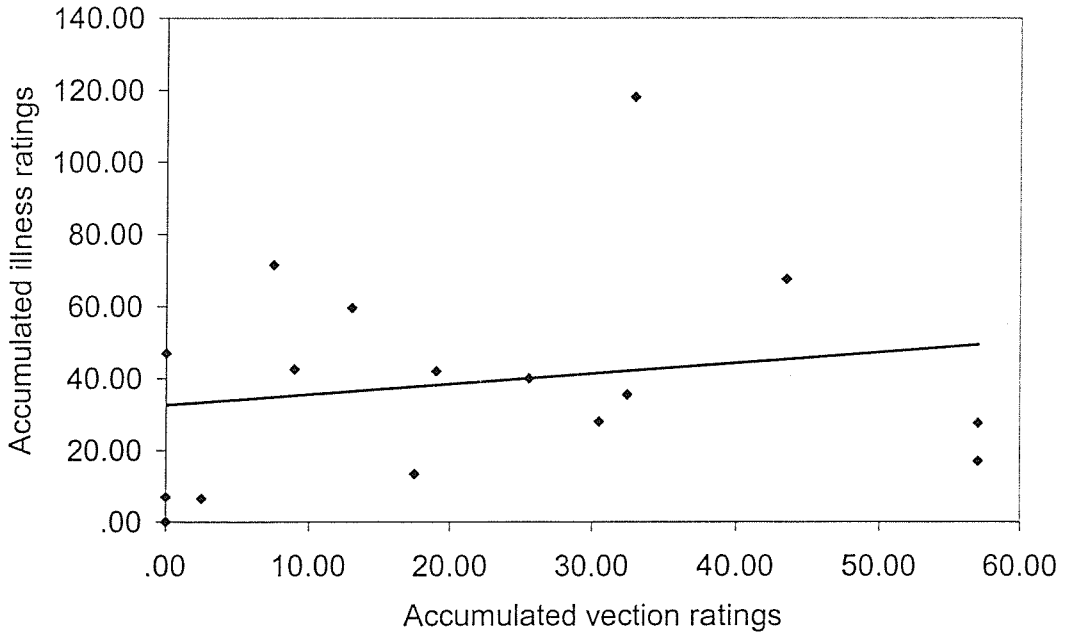

Figure 4.3 Accumulated vection and illness ratings – virtual drum.

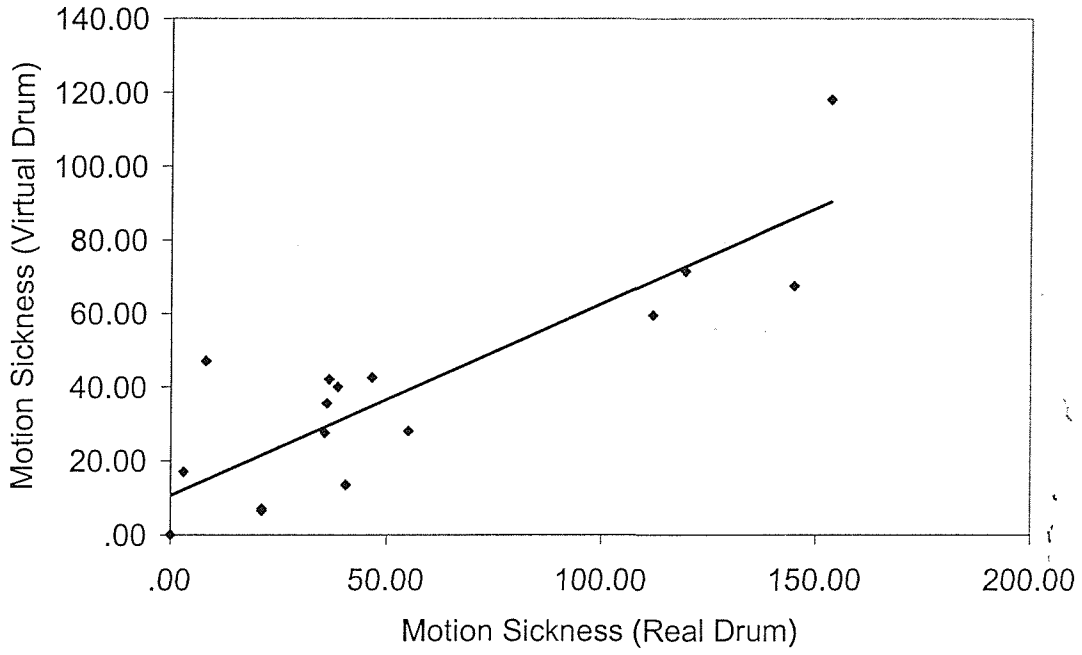

Figure 4.4. Motion sickness scores in the real and virtual drums

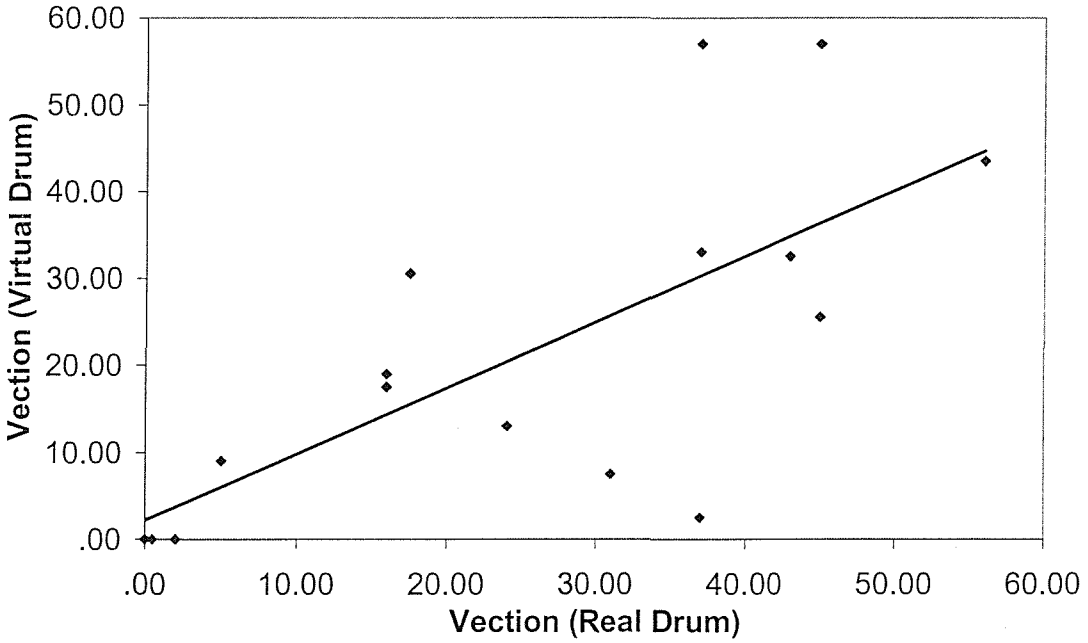

Figure 4.5. Vection scores in the real and virtual drums.

drum and 54.5 in the real drum (Wilcoxon, $p<0.05$). Figure 4.1 shows how the mean sickness ratings vary with time in both conditions. Post exposure symptoms were not significantly different in the two conditions (Wilcoxon, $p>0.10$). There was no correlation between the accumulated vection scores and the total illness scores in

either the real drum (ρ= 0.306, p>0.10) or the virtual drum (ρ= 0.223, p>0.10) - see Figures 4.2 and 4.3.

There was a significant correlation between the accumulated illness ratings of subjects in the two conditions – see Figure 4.4 (ρ= 0.755, p<0.001). There was also a significant correlation between the accumulated vection scores of individual subjects in the two conditions – see Figure 4.5 (ρ= 0.768, p<0.001). These results indicate that subjects who experienced motion sickness in one condition tended to experience motion sickness in the other condition and those who experienced vection in one condition also tended to experience vection in the other.

There was no apparent effect of order of presentation on the motion sickness ratings. This was tested by comparing the group of 8 subjects who experienced the real drum first and those who experienced the real drum second and likewise for those who experienced the virtual drum first and second. These comparisons showed that there was no significant difference between first or second groups in either case (Mann-Whitney U test, p>0.10).

4.4.1 Survival analysis – real drum

The time taken for a subject to reach '2' on the motion sickness scale 'mild symptoms e.g. stomach awareness but no nausea', the subject visual acuity at near (0.4m) and far points (2.5m) and the rating of past susceptibility derived from the motion sickness questionnaire ('total susceptibility to motion sickness', M_{total}, as per Griffin and Howarth, 2000) were tested with Spearman's rank correlation test. It was found that there was a significant influence of visual acuity at the near point on survival time (ρ= 0.678, p<0.01) poor acuity being associated with shorter survival times (i.e. earlier onset of symptoms). Visual acuity at the far point was not significantly correlated with survival time (ρ= -0.330, p>0.10). Past susceptibility to motion sickness was not significantly correlated with survival time (ρ= -0.039, p>0.10). Figure 4.6 shows the scatter plot of visual acuity and survival time for the real drum.

4.4.2 Survival analysis – virtual drum

In the virtual reality drum, survival time was again correlated with visual acuity at the near point (ρ= 0.577, p<0.05) but not at the far point (ρ= -0.067, p>0.10). Past

susceptibility was not significantly correlated with survival time but there was a trend towards significance (ρ= -0.437, p<0.10). Figure 4.7 shows the scatter plot of visual acuity and survival time for the virtual drum.

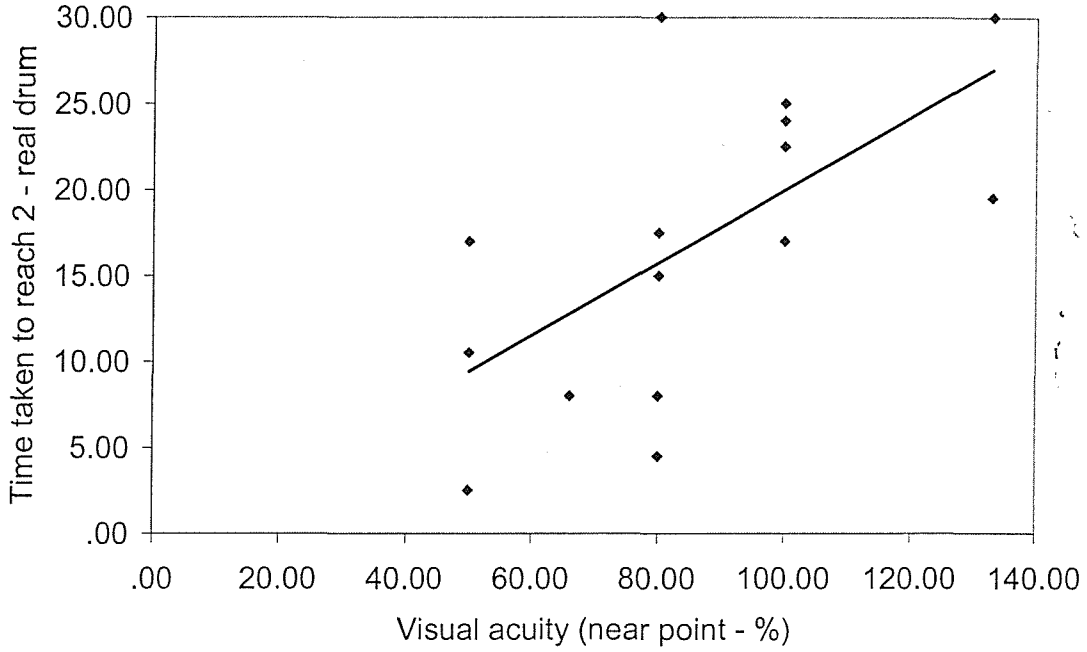

Figure 4.6. Visual acuity vs. survival time – real drum.

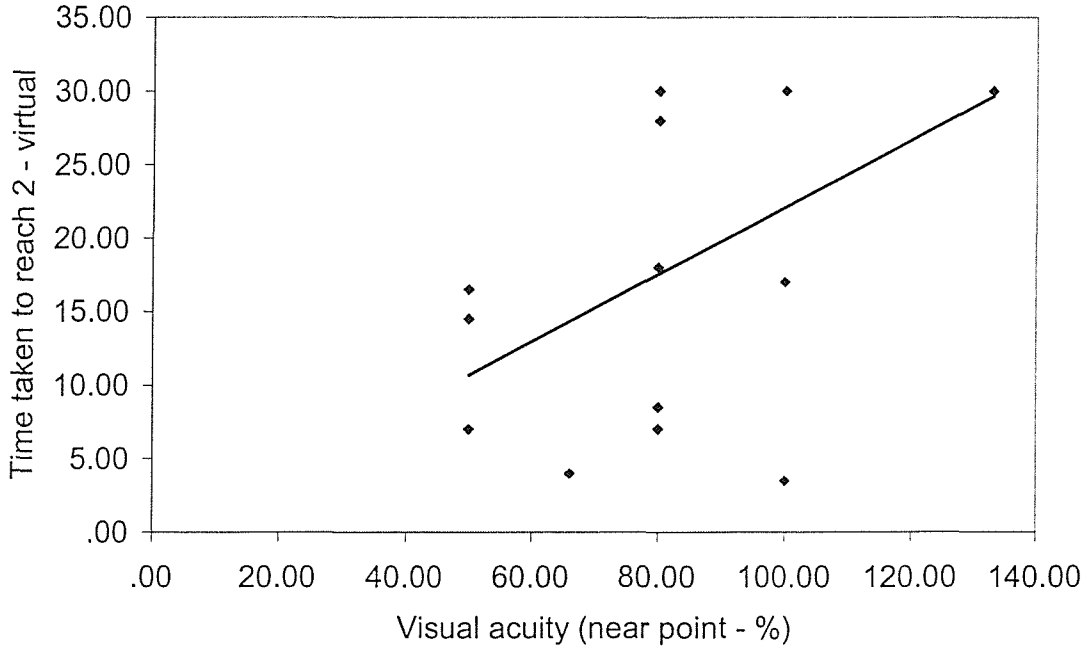

Figure 4.7. Visual acuity vs. survival time – virtual drum.

4.4.3 Cox's proportional hazards model

The factor found to significantly influence survival time in both the real and virtual reality drums was found to be visual acuity at the near point (0.4m) with lower survival times (more sickness) with poorer acuity. The visual acuity data were investigated with Cox regression to yield further information about the nature of the relationship. The visual acuity data was split into two groups for each of the conditions – low (lower than 20:20) and high (20:20 or higher). There were 9 subjects with low and 7 subjects with high acuity. A significant influence of visual acuity on survival time was found in the real drum (Cox regression, $p<0.05$) and in the virtual drum (Cox regression, $p<0.05$). Table 4.1 shows the Cox's proportional hazards model results for the real and virtual drums. The e^{β} values shows that a subject in the real drum was 3 times more likely to reach '2' on the motion sickness scale during the 30 minute exposure period if their visual acuity was less than 20:20. A subject in the virtual drum was nearly 5 times more likely to reach '2' on the motion sickness scale if they had lower than 20:20 vision.

Table 4.1. Cox proportional hazards model.

Condition	Independent variables	e^{β}	Sig (β)
Expt 1 – Virtual Drum	Visual acuity at the near point in two groups – high (>=20:20), low (<20:20)	4.9137	0.0476
Expt 1 - Real Drum	Visual acuity at the near point in two groups – high (>=20:20), low (<20:20)	3.0555	0.0436

4.4.4 Visual acuity and vection

Individual subject visual acuity scores were not correlated with individual accumulated vection ratings in either the real condition ($\rho=0.306$, $p>0.10$) or the virtual condition ($\rho= 0.223$, $p>0.10$). The relation between vection and acuity could not be investigated in the same way as the relation between sickness and acuity (with a Cox regression model) because vection comes and goes during optokinetic stimulation.

4.5 *Discussion and conclusions*

The results indicated that perceptions of vection did not significantly influence the motion sickness symptoms experienced in either the real or the virtual reality optokinetic drum. Further experiments will also separately measure motion sickness and vection, in order to understand more about the relationship between them and to test whether they can be independently manipulated.

Visual acuity was found to be significantly correlated with motion sickness survival time, with poorer acuity resulting in greater sickness. This is not something which has been previously been reported in the literature and occurred in the real and virtual reality versions of the optokinetic stimulus. The effect of visual acuity on motion sickness is investigated further in Experiment 2.

Motion sickness scores in the real and virtual drums differed significantly. However the motion sickness scores for individual subjects across the two conditions were correlated significantly as were the vection scores across the two conditions. The correlations indicate that the virtual reality display may be a useful tool for the study of motion sickness where it can present a large variety of different visual scenes which would be impossible or expensive with other traditional means such as optokinetic drums or projector systems.

The small difference in motion sickness scores between the two conditions may have been due to slight imperfections in the virtual model where there were occasional jumps in the playback and some stationary pixels which were visible in the background of the display. These minor deficiencies in the display were fixed in the second experiment presented in Chapter 5.

4.6 *Updated model*

The model has been updated to take into account the influence of visual acuity and the lack of a correlation between vection and motion sickness. The model is shown in Figure 4.8.

The tentative link between vection and motion sickness in the first model is broken so that vection and motion sickness appear in the model as separate outputs. The two remaining routes to 'motion sickness' in the model are via eye movements directly influencing motion sickness or via foveal image slip.

The finding that visual acuity was significantly correlated with motion sickness survival times, with poor acuity associated with increased motion sickness, is included in the model. Visual acuity as measured in this experiment, was a measure of the ability of the fovea to discriminate fine detail at high contrast, so this influence is included acting upon the foveal pursuit path of the model. Here it may act to decrease the influence of the foveal pursuit component on the slow phase of nystagmus.

This updated model predicts that visual acuity will only be significantly associated with motion sickness when the eyes are free to move or there is foveal image slip. In a condition with fixation (e.g. where the eyes are focusing on a stationary cross in front of moving stripes) it would be expected that the influence of visual acuity would not occur. This is investigated further in the next Chapter.

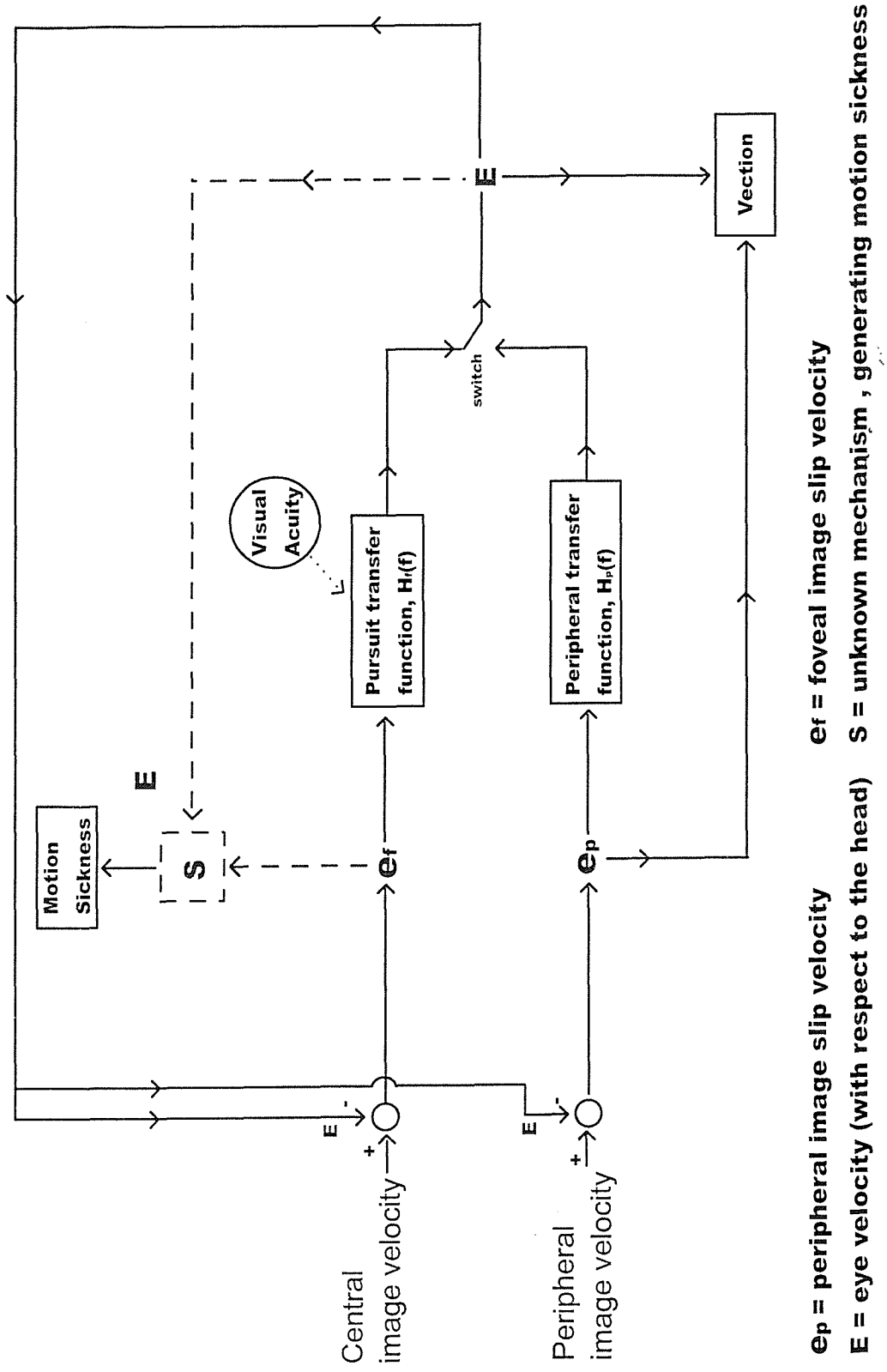

Figure 4.8. Model version 2. The proposed influence of visual acuity on the pursuit component of the slow phase velocity and the lack of a correlation between vection and motion sickness have been included.

Chapter 5. Experiment 2. Motion sickness and vection with and without visual fixation.

5.1 Introduction

In the first experiment, subjects with poor acuity gave higher illness ratings: an effect that does not appear to have been previously reported. No correlation between vection and motion sickness was found. The influence of visual acuity on motion sickness was investigated in this experiment. Previous research (Stern *et al.,* 1990) has shown a reduction in motion sickness when eye movements are suppressed by the act of fixation on a stationary object in front of an optokinetic background. It has been hypothesised that motion sickness is controlled partly by eye movements (Ebenholtz *et al.,* 1994) but that vection is mainly controlled by the peripheral vision (Brandt *et al.,* 1973).

The second experiment therefore suppressed eye movements in one condition by providing a stationary fixation point while the remaining visual scene moved as in the first experiment. By examining model 2 (Section 4.6) it was hypothesised that the presence of the fixation point would reduce eye movements because of the dominance of the fovea on the control of eye movements. It was also predicted that motion sickness would be reduced, because the two possible paths in the model to motion sickness are via eye movements or via foveal image slip, both of which are reduced by fixation. Vection was predicted to be the same in both conditions because of the suggested dominance of the peripheral visual receptors on vection and the predicted independence of vection and eye movements.

It was also predicted that, without the fixation point, motion sickness would be correlated with visual acuity, as in the first experiment, but with fixation there would be no correlation between visual acuity and motion sickness because of the reduction in eye movements or the reduction in foveal image slip.

5.2 Method

Subjects watched two conditions on the Virtual Research VR4 head-mounted display: the same optokinetic drum simulation as used in Experiment 1 and a similar

condition but with the addition of a stationary cross in front of the moving stripes. The two conditions are shown in Figure 5.1. Both conditions simulated optokinetic drum rotation of 30°/second (5 r.p.m.). The images were presented with an improved video interface which removed the occasional glitches and appearance of stationary pixels found in experiment 1. It was possible to ensure that the eyes of subjects were open by looking through a gap in the side of the display.

Subject visual acuity was measured as in the first experiment. Eye movements in the horizontal plane were continuously recorded throughout both conditions using electro-oculography and acquired to computer using an *HVLab* data acquisition system at 30 samples per second, with a low pass frequency cut-off at 10Hz (see Chapter 3 for further information).

The exposure duration for each condition was 30 minutes, with subjects reporting motion sickness symptoms and vection each minute as described in the first experiment. Eighteen subjects took part in the study, with each subject experiencing both conditions separated by an interval of at least 2 weeks. Subjects experienced each condition at the same time of day. Nine subjects experienced the 'fixation' condition first and the other 9 subjects experienced the 'non-fixation' condition first. The heads of subjects were restrained by the use of a strap attached to the display and to the backrest of the chair. Subjects sat in the chair of the optokinetic drum used in experiment 1, but with the drum in its raised position. Subjects heard white noise through headphones during the presentation, and were spoken to through a microphone each minute. Motion sickness ratings and vection ratings were reported each minute as in Experiment 1 (see Tables 3.1 and 3.2).

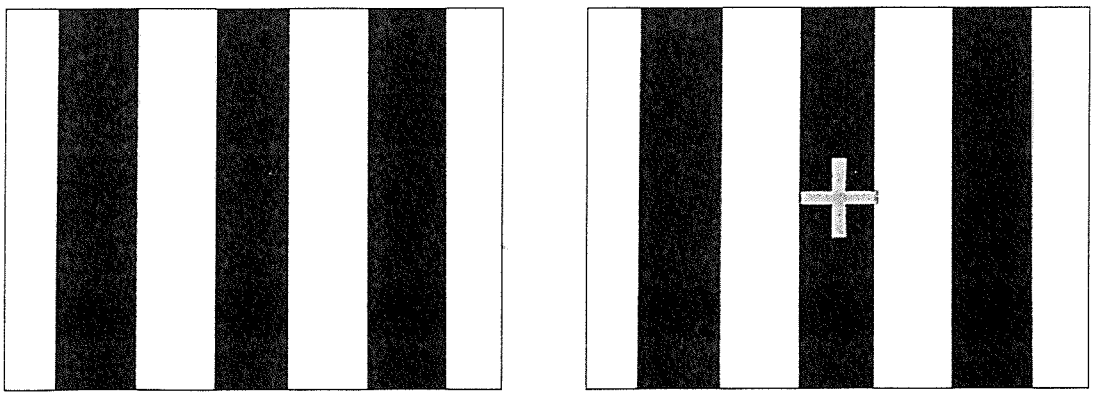

Figure 5.1. The normal condition and the fixation condition. In the fixation condition subjects focused on the stationary cross while the stripes moved behind it.

5.3 *Analysis*

5.3.1 Eye movements

The eye movement data were visually inspected. No repetitive eye movements occurred during the fixation condition, indicating that nystagmus was completely suppressed. In the condition without the fixation cross, a variability in eye movements was observed between subjects, with high variation in the duration for which nystagmus occurred. Some subjects had periods with no eye movements and other periods when eye movements were typical of tracking the black and white stripes (i.e. nystagmus: smooth pursuit followed by a rapid return saccade). Nystagmus generally occurred for between 30% and 100% of the exposure when there was no fixation. An approximate percentage time in which nystagmus occurred and an approximate nystagmus frequency was found for each subject in the non-fixation condition. The average frequency was determined only from the periods in which nystagmus occurred. The inspection of eye movements was performed without knowing which subject was being analysed.

5.3.2 Statistics

Motion sickness and vection scores were analysed using the Wilcoxon matched-pairs signed ranks test. Spearman's rank correlation test was used to test the relationships

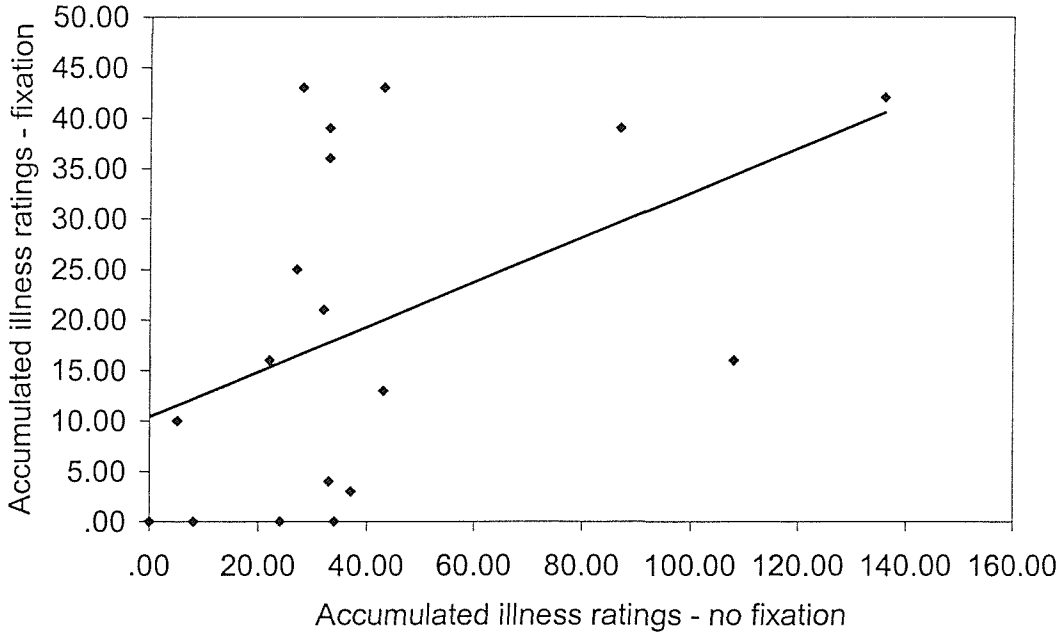

Figure 5.2. Accumulated illness ratings in the two conditions.

between vection, motion sickness and past susceptibility. Survival analysis was performed as in experiment one.

5.4 Results

5.4.1 Motion Sickness

The mean accumulated illness rating over 30 minutes was significantly less in the fixation condition: 19.4 with fixation compared to 40.7 without fixation (Wilcoxon, $p<0.01$). Post exposure symptoms as measured by the questionnaire were also lower with fixation (Wilcoxon, $p<0.05$). Mean motion sickness scores against time are shown in Figure 5.3. Total illness ratings for individual subjects in the two conditions were marginally significantly correlated ($\rho=0.445$, $p<0.10$) and are shown in Figure 5.2.

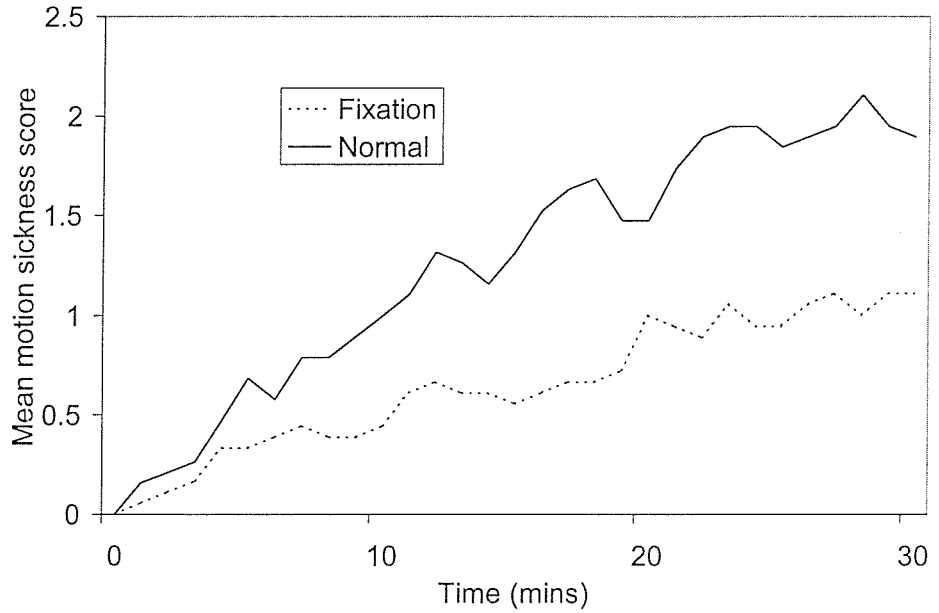

Figure 5.3. Mean motion sickness ratings against time for the two conditions.

5.4.1.1 Survival analysis - normal condition

A marginally significant correlation was found between visual acuity at the near point and survival time ($\rho= 0.432$, $p<0.10$) with poor acuity being associated with shorter survival times (i.e. earlier onset of symptoms). Figure 4.4 shows survival time for varying visual acuity. Visual acuity at the far point was not significantly correlated with survival time ($\rho= 0.186$, $p>0.10$). Past susceptibility to motion sickness was not

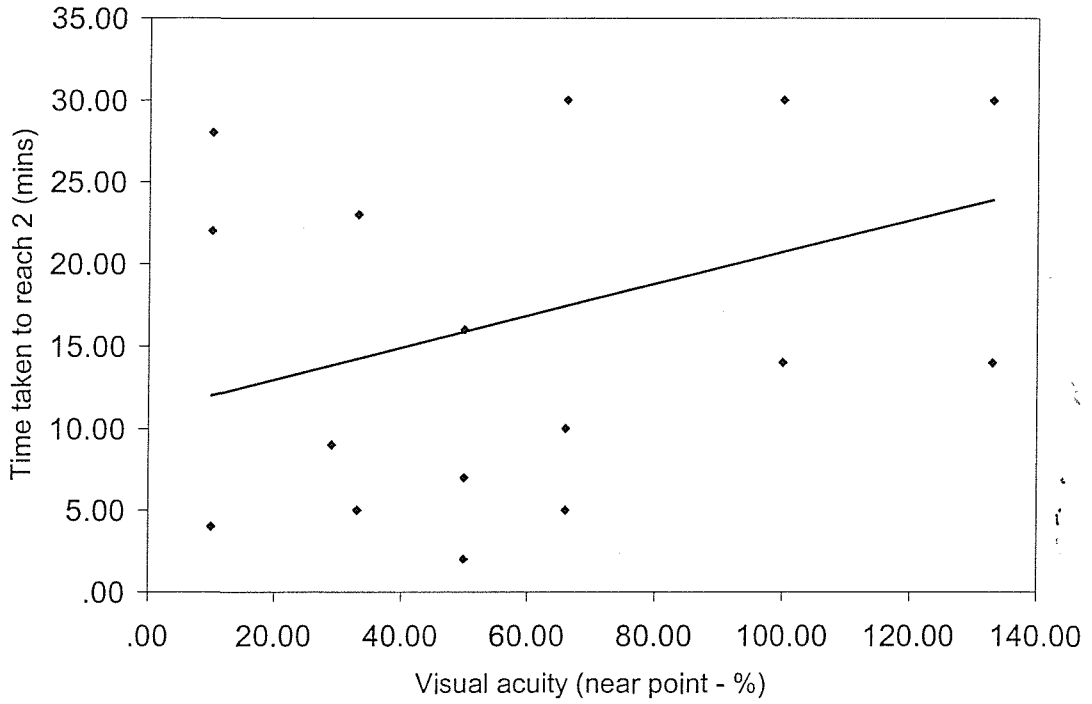

Figure 5.4. Variation of survival time with visual acuity for the non-fixation condition.

significantly correlated with survival time (ρ= -0.044, p>0.10). There was an effect of the percentage time of eye movements on survival time (Spearman rho = -0.574, p<0.05): an increase in nystagmus was associated with a reduced survival time. There was no significant correlation between survival time and average nystagmus frequency (ρ = -0.158, p>0.10).

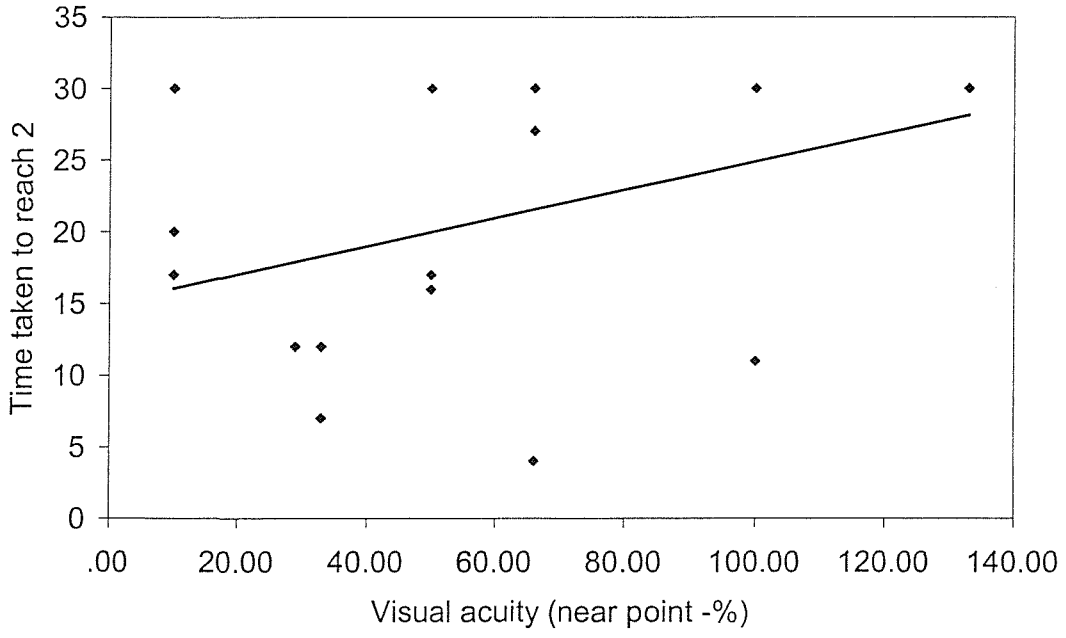

Figure 5.5. Variation of survival time with visual acuity for the fixation condition.

5.4.1.2 *Survival analysis – fixation condition*

In the fixation condition it was found that visual acuity at the near point was not correlated with survival time (ρ= 0.389, p>0.10) nor at the far point (ρ= -0.067, p>0.10). There was a marginally significant correlation between survival time and past susceptibility to motion sickness (ρ= -0.437, p<0.10). Figure 4.5 shows survival time for varying visual acuity in the fixation condition.

5.4.1.3 *Cox's proportional hazards model*

In the normal condition the percentage time in which nystagmus was occurring was found to have a significant influence on survival time and the visual acuity data recorded at the near point showed a marginally significant association with survival time. A Cox regression analysis was performed to find out more about the associations, with the visual acuity data at the near point split into high (20:20 or greater) and low (less than 20:20) and the nystagmus time variable. There were 12 subjects with low acuity and 6 subjects with high acuity. It was found that visual acuity had a significant effect on survival time (Cox regression, p<0.05) but the nystagmus time variable was not found to be significant when included with visual acuity (Cox regression, p>0.10).

In the fixation condition visual acuity was not found to be significant but the past susceptibility ratings showed a strong trend towards a significant influence. The effect of past susceptibility was investigated in a Cox regression model and was found to be significant (p<0.01). Table 5.1 shows the Cox's proportional hazards model for the significant variables in both conditions.

Table 5.1. Cox proportional hazards model.

Condition	**Independent variables**	e^{β}	**Sig (β)**
Expt 2 – Normal Cond.	Visual acuity at the near point in two groups – high (>=20:20), low (<20:20)	5.1058	0.0358
Expt 2 – Fixation Cond.	Past susceptibility	1.0624	0.0098

5.4.2 Vection

Individual subject accumulated vection scores did not correlate with accumulated illness ratings in either the normal condition (ρ=0.178, p>0.10) or in the fixation condition (ρ= 0.086, p>0.10). There was no significant difference in the accumulated vection ratings with or without fixation (Wilcoxon, p>0.10) or the time taken to first experience vection (Wilcoxon, p>0.10). Inspection of the raw results showed that nine subjects reported greater vection with fixation while nine subjects reported greater vection without fixation. Eye movements during the condition without fixation were compared with vection ratings. There was no apparent difference in vection ratings when the eyes were moving or stationary: vection was reported when the eyes were moving and when the eyes were stationary.

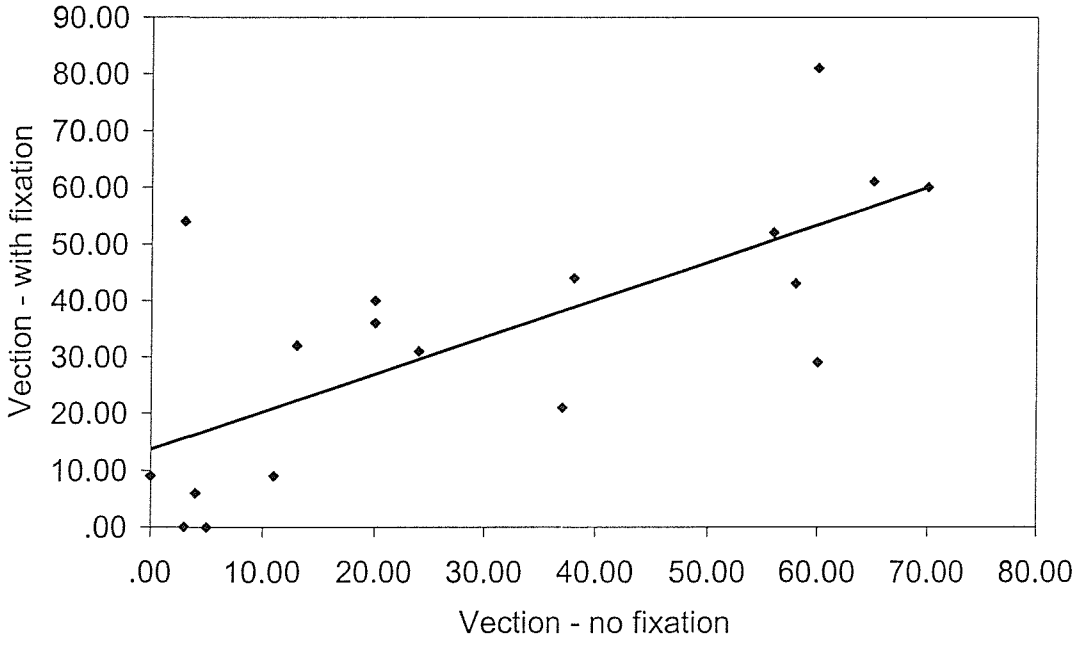

Figure 5.6. Vection scores in the two conditions.

There was a significant correlation between subject accumulated vection ratings in the two conditions (ρ= 0.674, p< 0.01) indicating that those subjects who experienced vection in one condition also experienced vection in the other, despite eye movements occurring during the normal condition but not during fixation (see Figure 5.6).

5.5 *Discussion and conclusions*

The reduction in sickness with fixation is consistent with reductions in eye movements or a reduction in motion on the fovea reducing motion sickness. Although visual fixation reduced motion sickness it did not affect vection. This suggests that vection does not have a large influence on motion sickness with this type of moving visual scene. It also suggests that vection was not greatly influenced by eye movements. This is consistent with vection being mainly determined by motion in the periphery of the visual field and being independent of eye movements as predicted by the model.

Vection ratings were similar in both conditions despite the difference in motion sickness. The ratings of vection in both experiments were uncorrelated with ratings of motion sickness. This is consistent with the findings of experiment one and this suggests that 'sensory conflict' brought about by the illusion of motion was not the cause of sickness. The results show that vection and sickness are not simply related: they appear to be distinct phenomena that can occur together but may also occur independently, depending on the properties of the display and the nature of the task.

There was a correlation between accumulated vection ratings in the two conditions but there was only a marginal correlation of accumulated illness ratings in the two conditions. This indicated that subjects who experienced motion sickness in one condition did not necessarily experience motion sickness in the other condition, but those experiencing vection in one condition were likely to experience vection in the other. This, again, is consistent with motion sickness being influenced by foveal vision or eye movements (which differed between conditions) and vection being influenced by peripheral vision (which was similar in the two conditions) and independent of eye movements.

The association of visual acuity with motion sickness has occurred so far in both conditions of experiment one (real and virtual reality) and in the non-fixation condition of this experiment. The association was not found in the fixation condition. Two things are different during fixation: (i) there are no eye movements (ii) there is no motion of images on the fovea. This suggests that visual acuity may possibly influence eye movements which are in turn influencing motion sickness in an unknown way, or that image slip detected on the fovea is influencing motion

sickness. However, the difference between the correlation coefficients found between visual acuity and motion sickness without fixation (ρ=0.432, p<0.05) and between visual acuity and motion sickness with fixation (ρ=0.389, p>0.10) is not large. Caution in the interpretation of the results is necessary, as the correlation between visual acuity and motion sickness may possibly be found to be significant in a further experiment.

Ratings of past susceptibility were not found to be a significant influence on motion sickness survival except during the fixation condition. This allows for the possibility that visual acuity is an influence on motion sickness when there is motion on the fovea or eye movements, and that past susceptibility to other forms of motion sickness may be important when this influence of acuity is diminished by the act of fixation.

5.6 Updated model

The model, presented in Figure 5.7, is identical to that shown in Chapter 4. The correlation between visual acuity and motion sickness, when the eyes are free to move, or when there is motion on the fovea may have been confirmed by this experiment. Further investigation will be required.

The finding that vection and motion sickness are distinct phenomena has been confirmed by the ability of motion sickness symptoms to be manipulated separately from vection perceptions, and by no correlations being found between vection and motion sickness in subjects.

The influence of visual acuity is investigated further in the third experiment presented in the next chapter.

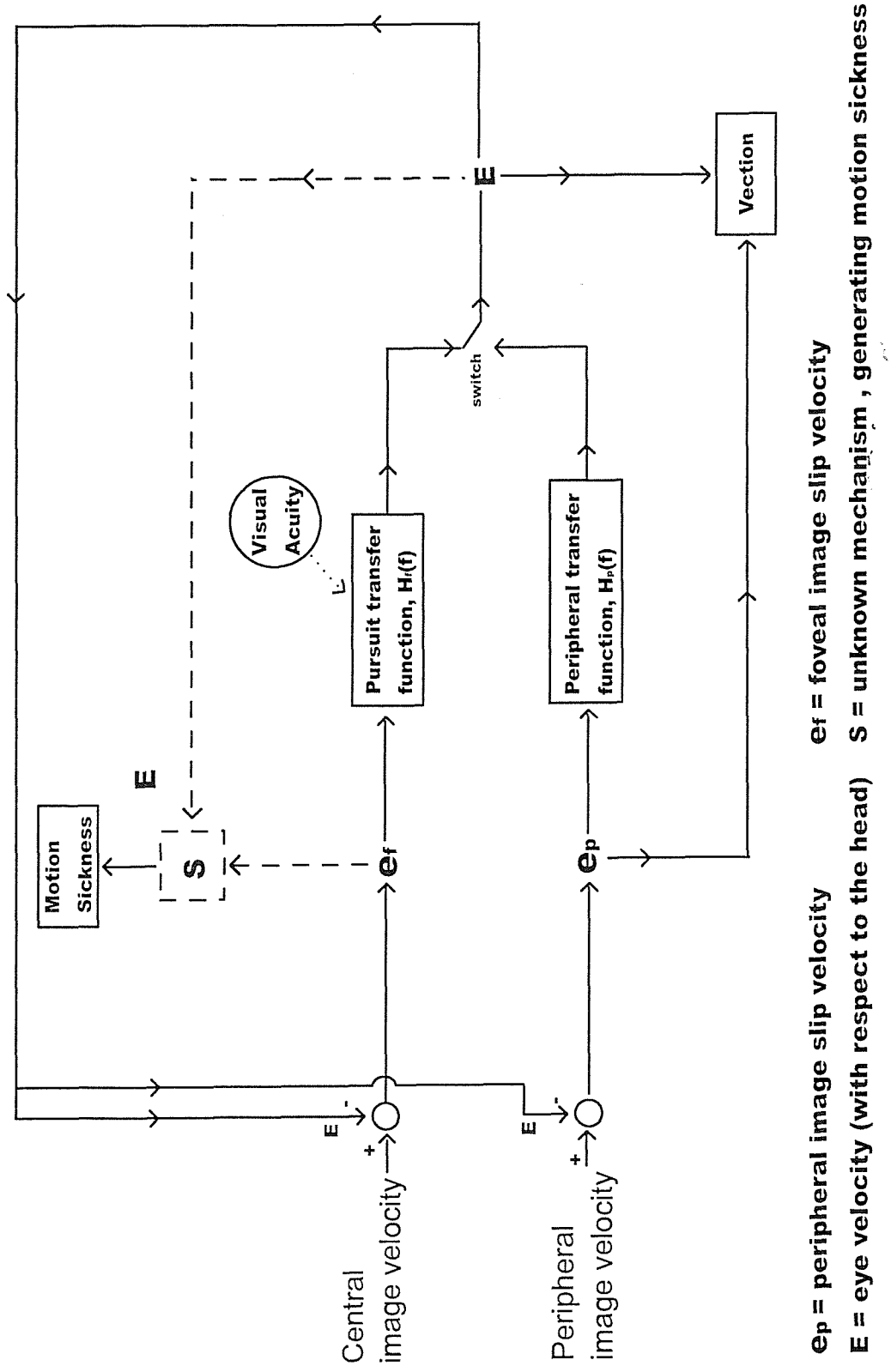

Figure 5.7. Model version 2. The model is identical to that shown in Chapter 4.

Chapter 6. Experiment 3. Motion sickness and vection with normal and blurred optokinetic stimuli

6.1 Introduction

Chapters 4 and 5 have shown that motion sickness and vection can be manipulated separately and may be distinct phenomena. Visual acuity has been shown to be correlated with motion sickness survival in all conditions, except during fixation. The association between visual acuity and survival time may possibly occur because it has an influence on eye movements which in turn influence motion sickness, or the detection of image slip on the fovea is somehow influencing motion sickness.

It was decided to test the possibility that artificial blurring of the stimulus viewed by subjects with good acuity could have the same effect as that of poor acuity. The experiment presented an artificially blurred optokinetic stimulus in one condition and compared the reports of motion sickness and vection with those arising from a normal optokinetic stimulus. It was hypothesised, with reference to model version 2 that vection would not differ between the two conditions because of the proposed peripheral dominance of vection. The removal of the high spatial frequency content of the stimulus, by artificial blurring, was predicted to increase motion sickness in the same way as poor acuity, the reasons for the effect still being unknown at this stage.

6.2 Method

Twenty subjects aged 18 - 28 years were selected for the experiment on the basis that they had good eyesight, which was defined for the purposes of this experiment as 20:20 vision or better, uncorrected, measured at the near point (0.4m) by the Landolt broken ring test, using the Keystone visual skills profiles.

The exposures consisted of moving visual stimuli presented on the Virtual Research VR4 head-mounted display (see Figure 6.1). The horizontal speed of the stripes was 30°/second as in all previous conditions. The blurring of the stripes in one condition was intended to reduce the resolution of the image presented by 50%. This was roughly estimated by using the blur parameter within the material editor of 3D Studio

MAX v1.2 and applying the blur to a bitmap image of text. The text in the image was of the same form as is commonly used in Snellen visual acuity tests, with increasingly small letters in horizontal lines. Eight lines of text were used and blur was applied until the bottom four lines of text were no longer readable, on the Virtual Research VR4 head-mounted display, with corrected vision. The resulting level of blur was noted and applied to the black and white striped image used in the optokinetic simulation. The blurring was applied to the source file in this way rather than by viewing the stimulus through blurring lenses which would have had a magnifying effect. The blurring had the effect of reducing the definition of the boundary between the black and the white stripes, so that there was a more gradual change from black to white, rather than a sharp edge (see Figure 6.1b).

Subjects were seated in the chair of the real optokinetic drum as used in all previous experimental conditions and the head of each subject was strapped to the back of the chair to prevent head movement. Each subject experienced both conditions, with 10 experiencing the normal condition first and 10 experiencing the blurred condition first. Exposure times were 30 minutes. There were two weeks or more between sessions to reduce effects of habituation and subjects experienced each condition at the same time of day. During the exposures, subjects rated their symptoms of motion sickness and vection as previously. After exposure, subjects completed the simulator sickness

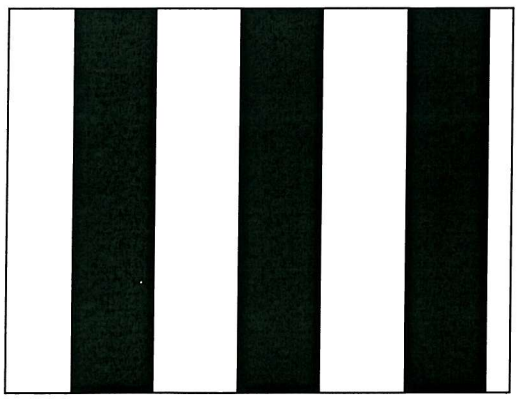

Figure 6.1a Normal stripes

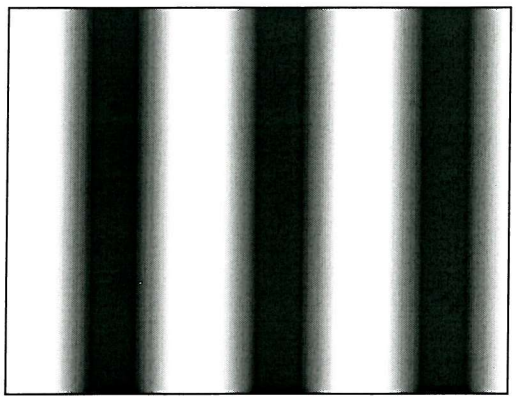

Figure 6.1b Blurred edges

questionnaire as previously used with the exception of the symptoms 'blurred vision' and 'difficulty focusing', which were removed!

6.3 Analysis

Motion sickness and vection scores across conditions were analysed using the Wilcoxon matched-pairs signed ranks test. Spearman's rank correlation test was used to test the relationships between vection and motion sickness in conditions. Survival analysis was performed as in Experiment 1 and Experiment 2.

6.4 Results

6.4.1 Motion Sickness

Accumulated illness ratings were calculated for each subject in both conditions. The mean accumulated illness rating for the normal condition was 39.5 and for the blurred condition was 40.8. There was no significant difference between the motion sickness ratings (Wilcoxon, $p>0.10$). The post exposure symptoms questionnaire showed there to be significantly more symptoms in the blurred condition (Wilcoxon, $p<0.05$). The mean illness ratings are shown against time in Figure 6.2.

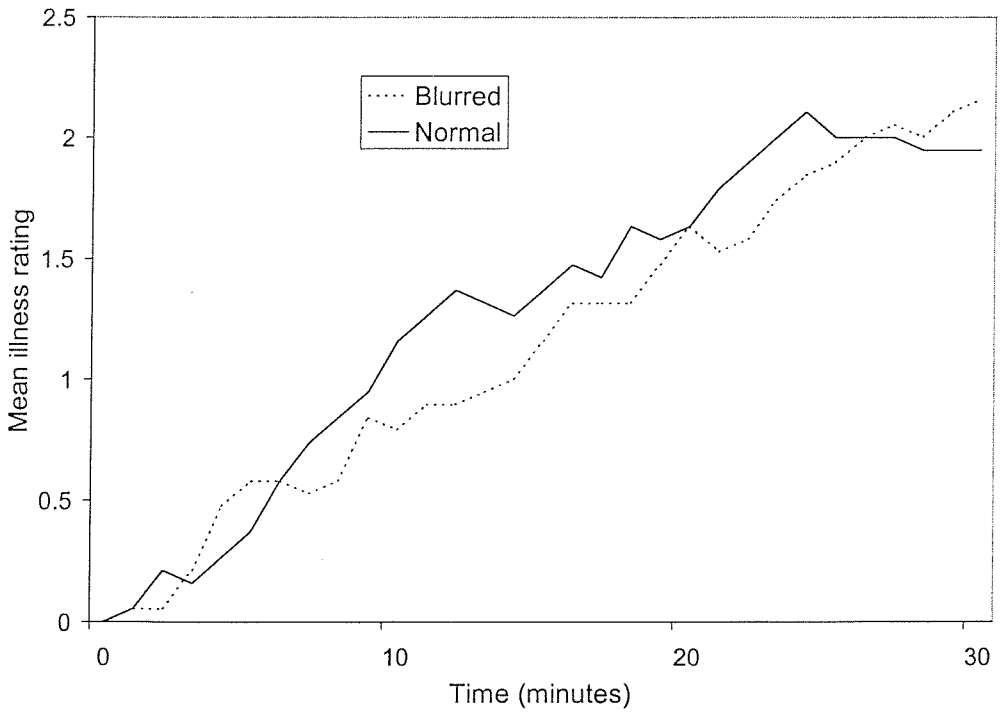

Figure 6.2. Mean illness ratings against time.

Subject motion sickness scores were correlated across the conditions ($\rho=0.620$, $p<0.01$) indicating that subjects who experienced motion sickness in one condition

also tended to experience motion sickness in the other. There were no correlations between accumulated vection scores and motion sickness scores in either the blurred (ρ=0.199, p>0.10) or the normal condition (ρ=0.130, p>0.10), see Figures 6.5 and 6.6.

6.4.2 Vection

There was no significant difference between the vection scores in the two conditions (Wilcoxon, p>0.10). There was a significant correlation between subject vection scores across conditions (Wilcoxon, p<0.001), indicating that subjects perceiving vection in one condition also tended to perceive vection in the other condition.

6.4.3 Survival analysis – normal condition

In the normal condition there was no correlation between survival time and subject visual acuity measured at the near point (ρ=-0.297, p>0.10) or measured at the far point (ρ=-0.215, p>0.10). Past susceptibility was not found to be correlated with survival time (ρ=-0.352, p>0.10) in this condition. Figure 6.3 shows the scatter plot of visual acuity at the near point and survival time for the normal condition.

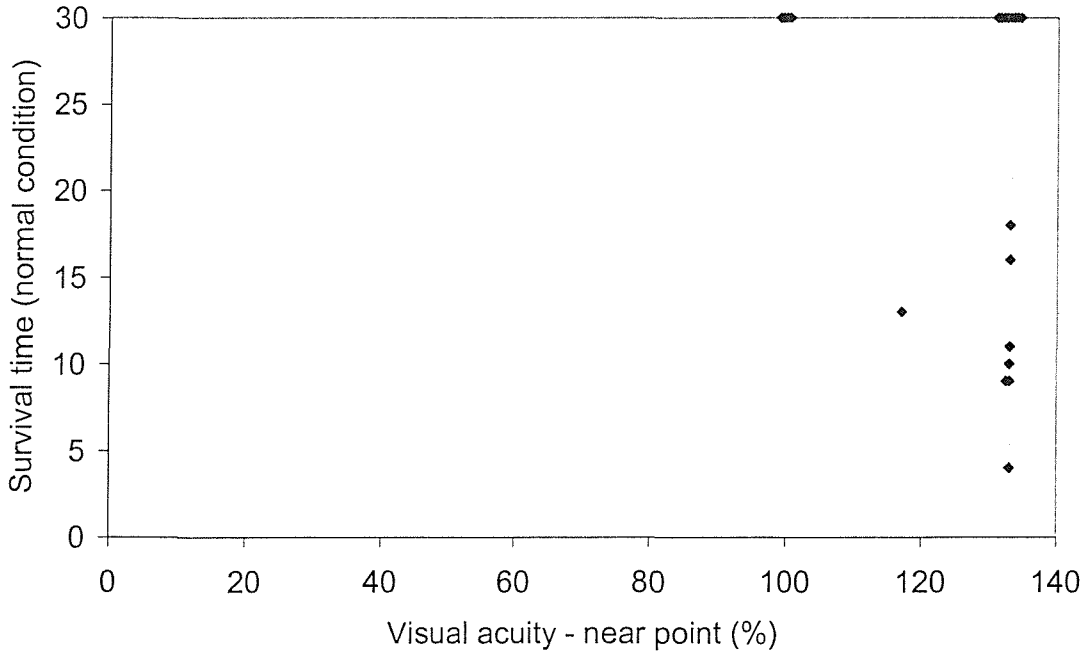

Figure 6.3. Variation of survival time with visual acuity for the normal condition.

6.4.4 Survival analysis – blurred condition

In the blurred condition there was no correlation between survival time and visual acuity at the near point (ρ=-0.204, p>0.10) or at the far point (ρ=-0.002, p>0.10). Past susceptibility was not correlated with survival time (ρ=-0.059, p>0.10). Figure 6.4 shows the scatter plot for visual acuity at the near point and survival time.

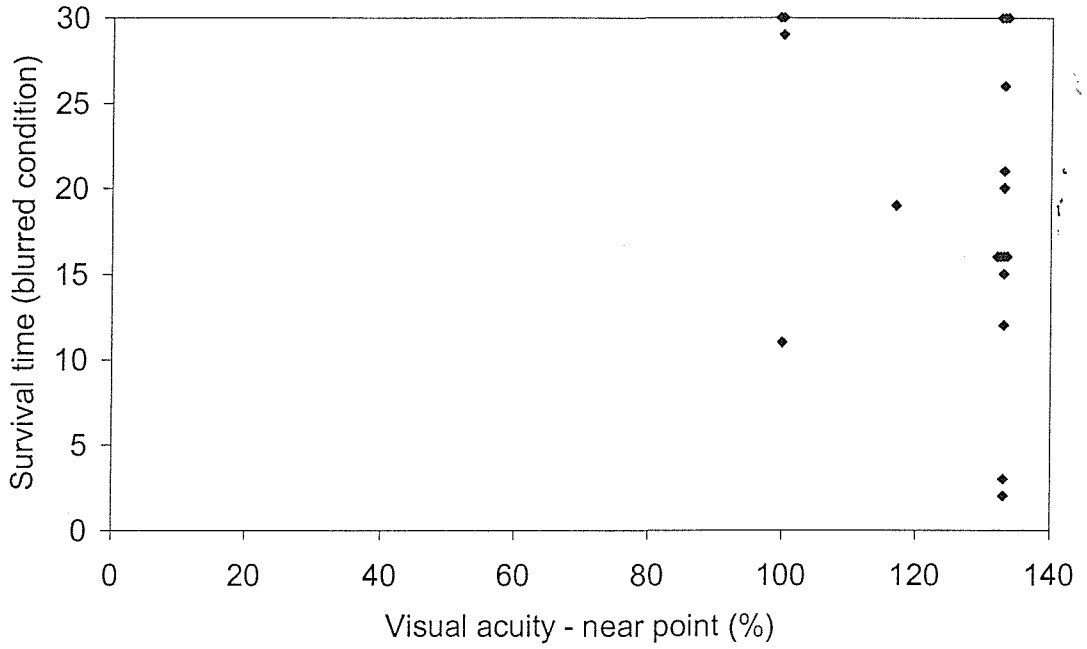

Figure 6.4. Variation of survival time with visual acuity for the blurred condition.

6.4.5 Cox's proportional hazards model

No significant correlations were found in the survival analysis, as shown above, therefore no Cox regression model was necessary.

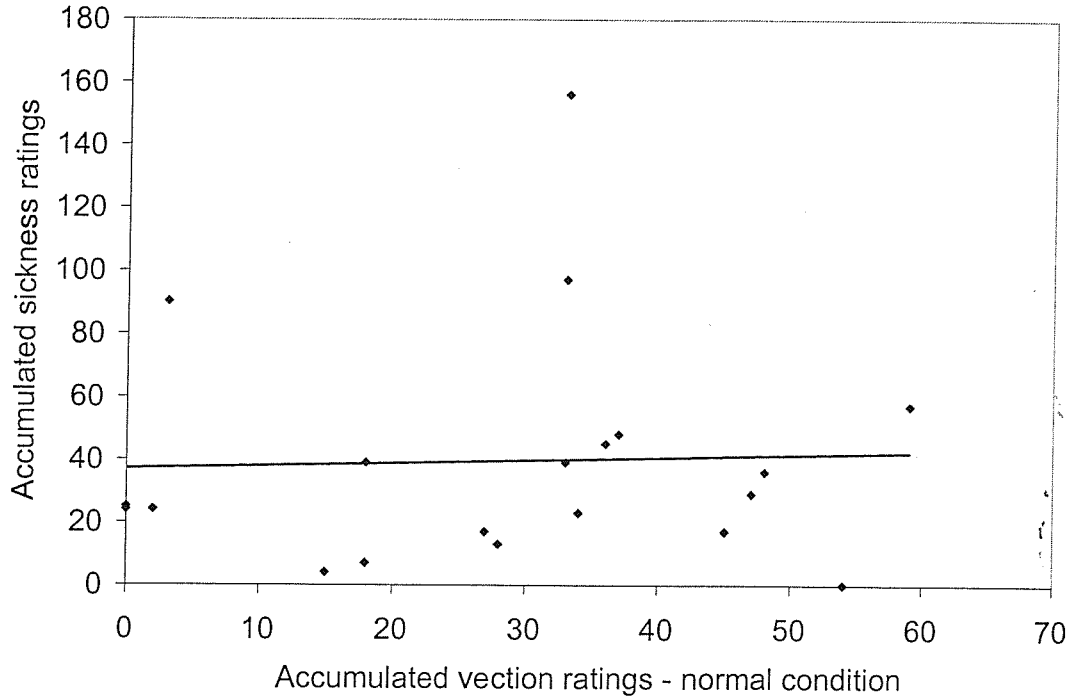

Figure 6.5. Vection and motion sickness scores in the normal condition.

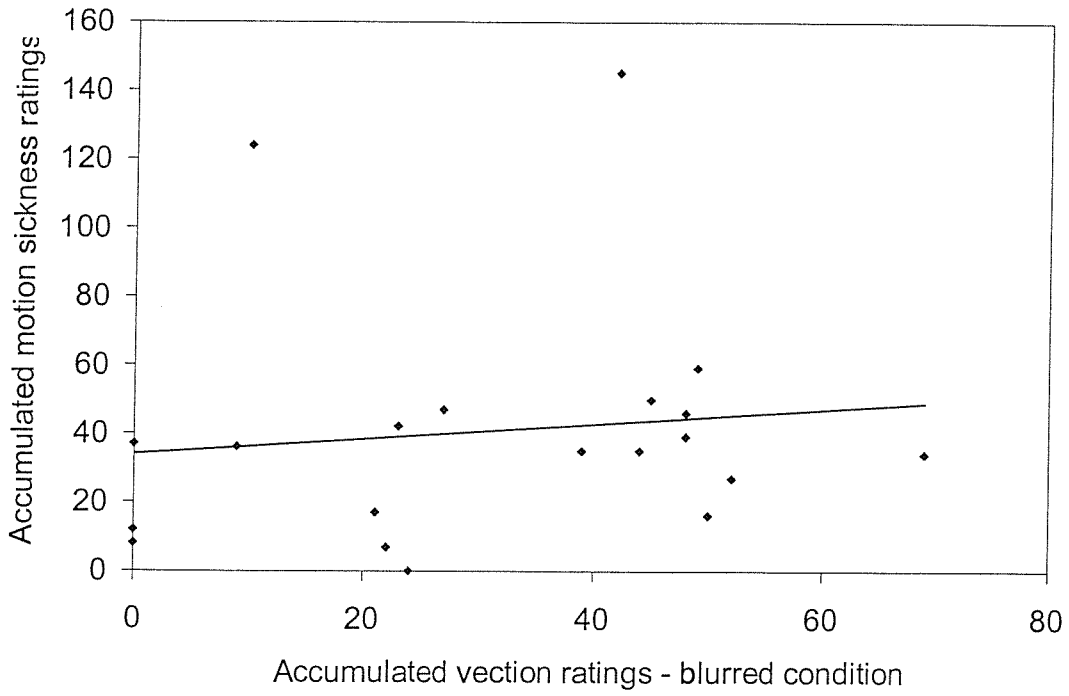

Figure 6.6. Vection and motion sickness scores in the blurred condition.

6.5 Discussion and conclusions

The conclusion drawn from Experiments 1 and 2, that vection and motion sickness are not related but are separate phenomena, is supported by this experiment. There were no correlations found between motion sickness and vection. Vection was also found to be similar in both conditions despite the artificial blur effect in one condition. Peripheral vision is not as sensitive to high spatial frequencies as the fovea. Removal of some of the high frequency content from the visual stimulus, by blurring, may not have changed the visibility of the stimulus in the periphery. This may explain why vection, which is probably controlled mainly by peripheral vision, did not vary between the normal and blurred conditions.

Accumulated illness ratings were not significantly different between conditions but post exposure symptoms were significantly different. This suggests that the artificial blur was only partially successful in the aim of increasing motion sickness.

It is possible that the artificial blurring of the stimulus may not have been completely successful in simulating poor visual acuity. The effect of the blur was to smear the boundary between the black and the white stripes. It may be that visual acuity has an effect on motion sickness which is not simply related to the amount of visual blur present. It may also be the case that there were some high frequency components left in the visual display, for example a straight edge can still be seen where the blurred boundary between the black and white stripes ends (Figure 6.1b). The increased symptoms reported post-exposure may suggest that the visual blur had some increased effect on motion sickness incidence. However, this result should be treated with caution at this stage.

Visual acuity was not significantly correlated with survival time in either condition. This is not surprising because all subjects had visual acuity as measured by the Landolt 'broken ring' test of 20:20 of greater. There was not enough variation in the visual acuity to see any significant correlations.

6.6 Updated Model

The possible effect of visual blur on motion sickness is added into the model (see

Figure 6.7). As with the visual acuity influence it is shown to act on the foveal pursuit component of the slow phase of nystagmus. The influence is shown with a dotted line to show that it is uncertain. The rest of the model remains unchanged. The peripheral influence on vection was confirmed, as were the distinct outputs for vection and motion sickness.

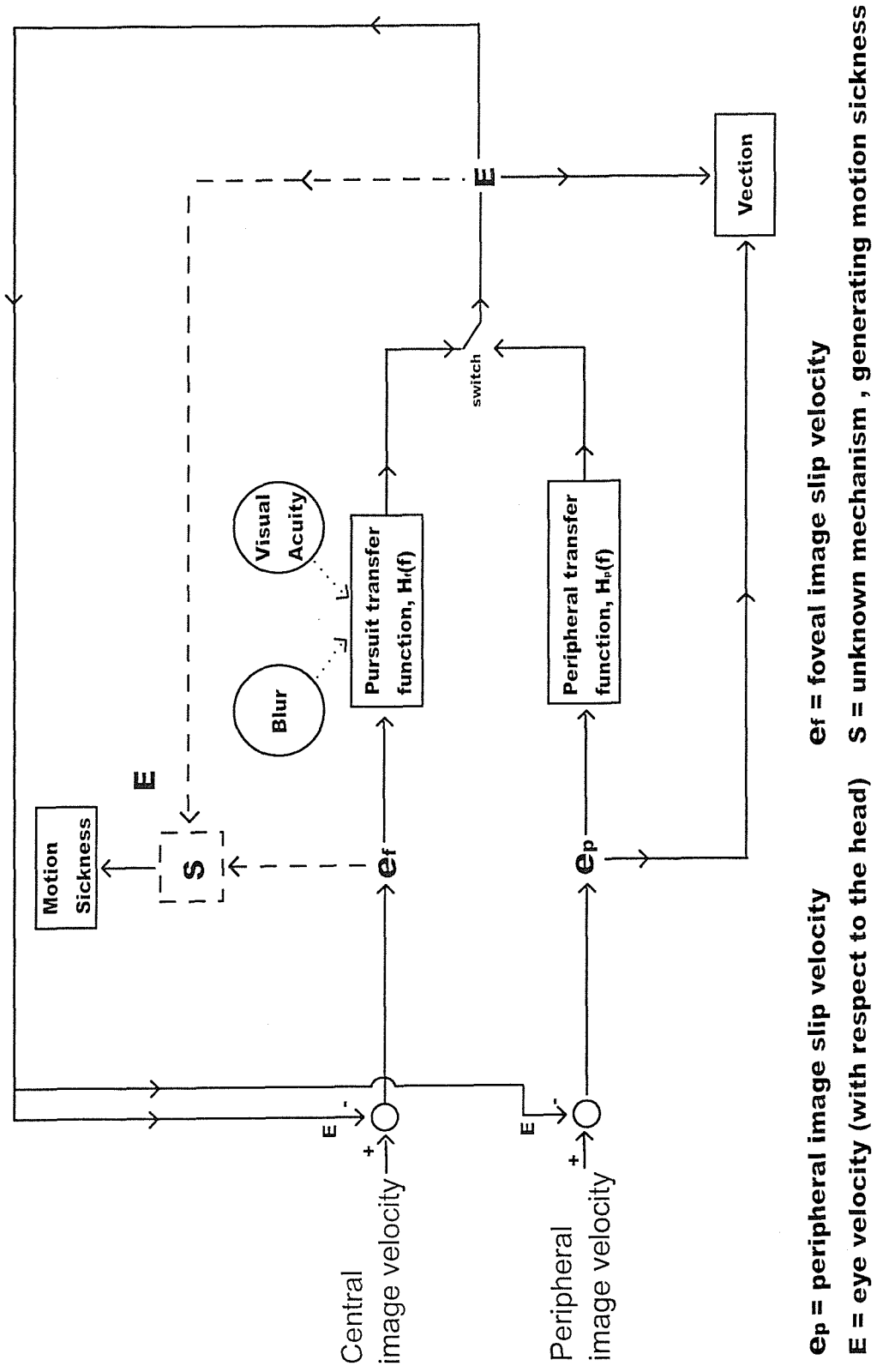

Figure 6.7. Model version 3. Updated model to show the possible influence of artificial blur.

Chapter 7. Experiment 4. Motion sickness and vection with a single and multiple dot display

7.1 Introduction

The model shown in the previous chapter has shown that vection and motion sickness are separate phenomena, modelled as separate outputs. This has been shown by the lack of correlations between vection and motion sickness scores and by the ability to manipulate motion sickness separately from vection, by the use of a fixation cross. The model predicts that not only can motion sickness be varied with no change in vection but also that vection can be varied independently of motion sickness. An experiment was devised to test this prediction. Subjects were presented with two conditions, one with a single moving dot which moved from left to right with a sudden jump back to its starting position, and one with multiple moving dots which moved continuously across the screen (see Figure 7.1). This method resulted in the same foveal stimulation in both conditions but different peripheral stimulation (see Section 7.2 for a full explanation).

Motion sickness was reduced with fixation in Experiment 2 (Chapter 5) and nystagmus has been shown in the literature review (Section 2.3.7.2) to be influenced dominantly by foveal vision. In the present experiment it was hypothesised that motion sickness would be similar in the two conditions because the foveal stimulus would be the same in each condition and eye movements would be the same. It was also hypothesised that vection would be higher in the full field condition because of the increased peripheral stimulation in this condition.

7.2 Method

Sixteen male subjects, aged 20-25 years participated in the experiment. Visual acuity was measured as in the previous experiments. Subjects viewed two conditions on the Virtual Research VR4 head-mounted display: (i) a single dot which moved from left to right over a distance of 18° at a rate of 27°/second before jumping back to its starting position and repeating on an infinite loop (ii) five horizontal rows of dots, with each dot 18° apart, moving continuously from left to right at a rate of 27°/second.

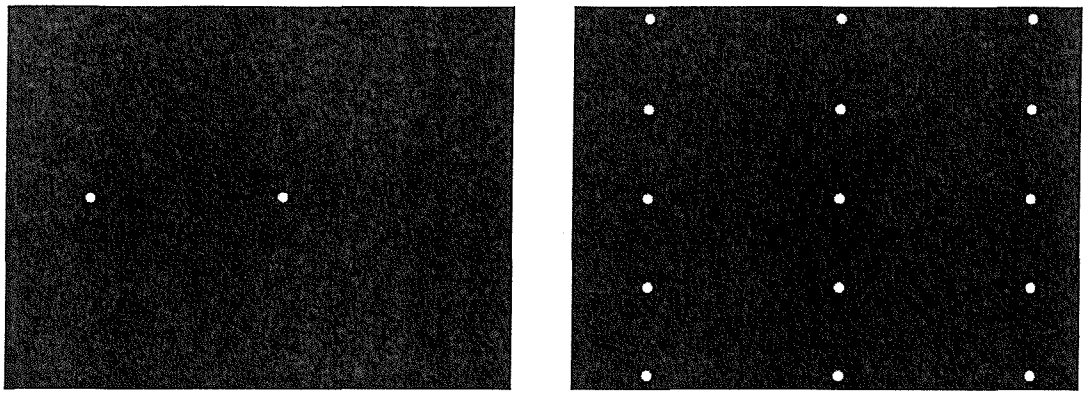

Figure 7.1. The two conditions. The picture on the left shows the start and end point for the single dot and the picture on the right shows the full field of dots.

Each subject experienced both conditions. Eight subjects commenced with the single dot and eight commenced with the multiple dot display. There were at least two weeks between exposure sessions to reduce any habituation effects and exposures were performed at the same time of day. During each exposure the following information was recorded at one minute intervals for a total of 30 minutes: the motion sickness rating on a 7 point scale (as used previously – Table 3.1) and a rating of the vection experienced, on a percentage scale (see Table 7.1). The vection scale had to be different to that used previously because there is no drum simulation in this experiment and a consistent scale was needed for the single and multiple dot conditions. At the end of each exposure, subjects filled out a post-exposure symptom checklist as before.

In condition one, subjects were asked to track the single dot continuously as it moved from left to right and then jumped back to its starting position. In condition 2, subjects were asked to track each dot in the middle row as it passed. In this way the foveal stimulus and eye movements were identical in the two conditions: a single dot moving from left to right at 27°/second for 18° followed by a rapid jump back of 18° to the next dot. The resulting eye movement was nystagmus with a smooth pursuit of 18° followed by a rapid return saccade, with a frequency of 1.5Hz, in each condition. During exposure, eye movements were recorded onto an *HVLab* data acquisition system at 30Hz sample rate using the Hortmann electro-nystagmograph described in Chapter 3.

Subjects gave their informed consent to participate in the experiment that was approved by the Human Experimentation Safety and Ethics Committee of the Institute of Sound and Vibration Research.

Table 7.1. New vection scale. Subjects report a percentage score between 0 and 100% each minute to indicate their perception of self motion.

Perception of motion (vection)	You report:
You feel like you are stationary and it is the dot(s) which appear to be moving only.	0%
You feel like you are moving a bit, but the dot(s) are moving more	1-49%
You feel like you are moving at the same speed as the dot(s)	50%
You feel like you are moving a lot and the dot(s) are moving a bit	51-99%
You feel like you are moving and the dot(s) appear stationary	100%

7.3 Analysis

Eye movements were studied by visual inspection and by time-frequency analysis to determine the dominant frequency component throughout the exposures. Average vection scores were calculated by taking the mean of the thirty responses, expressed as a percentage. Accumulated illness ratings were calculated as previously.

Motion sickness and vection scores across conditions were analysed using the Wilcoxon matched-pairs signed ranks test. Spearman's rank correlation test was used to test the relationships between vection and motion sickness in conditions. Survival analysis was performed as in experiments 1 to 3.

7.4 Results

7.4.1 Eye movements

The electro-oculography data was inspected by eye, for indications that the eye movements were similar in each of the conditions. Figure 7.2 shows sample eye movement data from each condition.

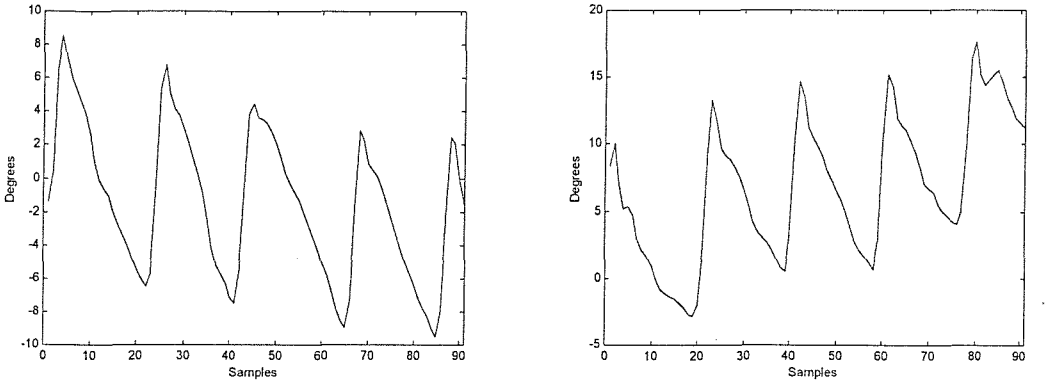

Figure 7.2. Sample eye movement data for one subject in the single (left) and full field (right) conditions. Sample rate is 30.3 samples per second, low pass filtered at 10Hz. Data above is the first 3 seconds of data in each condition.

Inspection of the eye movements for subjects revealed that eye movements were continuous throughout each exposure condition, indicating that tracking of the dots for long periods was possible. Time-frequency analysis revealed that the power in each set of eye movements was at around 1.5Hz in each condition throughout the exposures. This indicated that the experimental design was successful in generating eye movements that were similar in each condition. The foveal stimulation (of a single moving dot) was hence very similar in each condition, whilst the peripheral stimulus varied from nothing (single condition) to 14 continuously moving dots (full field condition).

7.4.2 Motion sickness

The mean accumulated illness ratings were 19.9 for the single dot and 22.8 for the full field of dots condition. There was no significant difference between the illness ratings in the two conditions (Wilcoxon, $p>0.10$). The post exposure symptoms questionnaire also showed no difference between the two conditions (Wilcoxon, $p>0.10$). Subject motion sickness scores were correlated between the two conditions ($\rho=0.516$, $p<0.05$). There were no correlations found between vection and motion sickness in the single dot condition ($\rho=0.191$, $p>0.10$) or the full field condition ($\rho=0.184$, $p>0.10$). Mean illness ratings against time for the two conditions are shown in Figure 7.3.

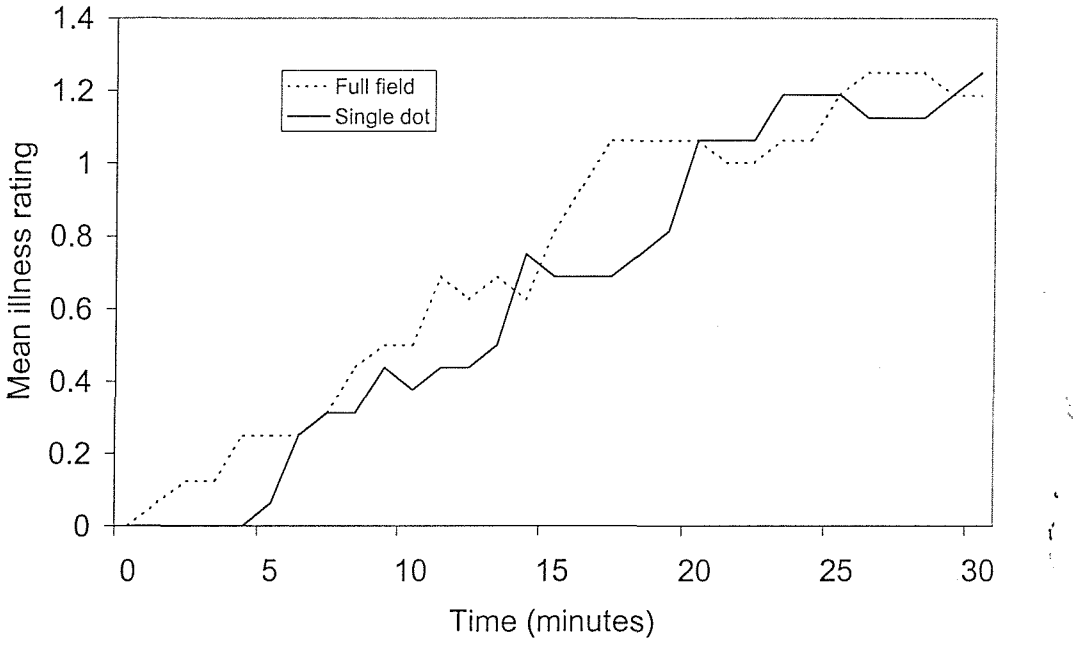

Figure 7.3. Mean illness ratings for the single and multiple dot conditions against time.

7.4.3 Vection

Mean vection scores were 12.6 (%) in the single dot condition and 27.4 (%) in the full field condition. The difference was significantly different (Wilcoxon, $p<0.05$). Vection scores for subjects across conditions were significantly correlated ($\rho=0.551$, $p<0.05$). This indicates that subjects reporting vection in one condition were likely to report vection in the other condition, but with generally higher vection in the full field condition. The small amount of vection in the single dot condition could possibly be due to the way in which the single dot was displayed. There were two frames each second ($1/30^{th}$ of one second) where the two dots shown in Figure 7.1 were simultaneously visible. This may have resulted in some stimulation of the peripheral retina.

7.4.4 Survival analysis – single dot condition

There was no correlation between survival time and visual acuity at the near point ($\rho=0.411$, $p>0.10$). There was no correlation between the visual acuity at the far point and survival time ($\rho=0.360$, $p>0.10$). Past susceptibility was not correlated with

survival time (ρ=-0.407, p>0.10). Visual acuity and survival time for the single dot condition are shown in Figure 7.4.

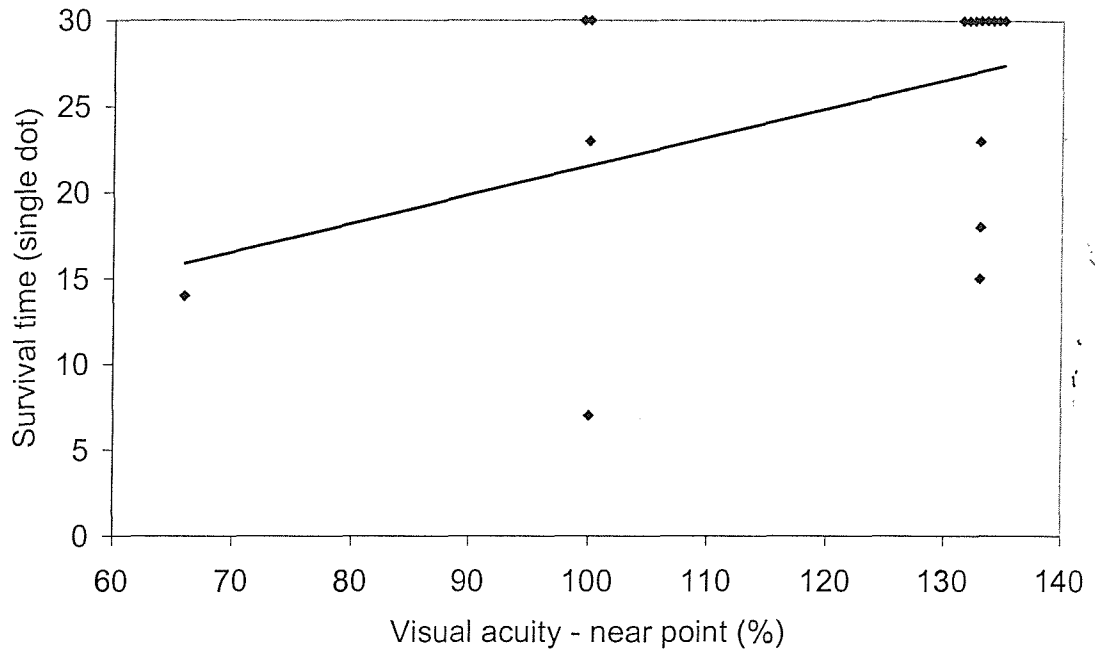

Figure 7.4. Survival time shown for varying visual acuity at the near point (single dot condition).

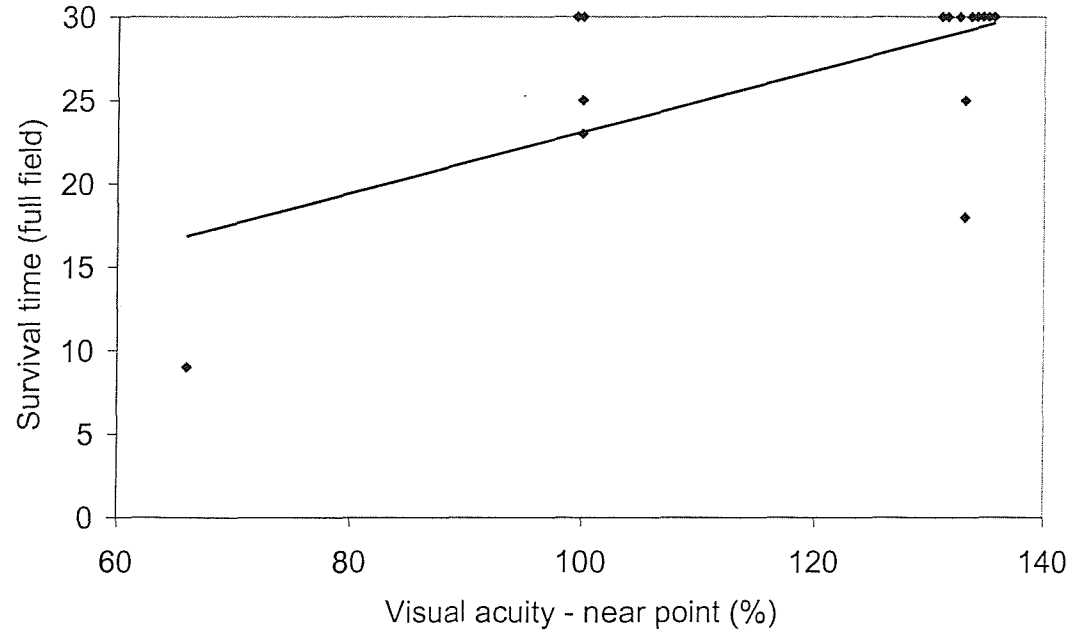

Figure 7.5. Survival time shown for varying visual acuity at the near point (full field condition).

7.4.5 Survival analysis – multiple dot condition

There was a marginally significant correlation between survival time and visual acuity in the full field condition (ρ=0.479, p<0.10) with subjects having shorter survival times if they had lower acuity. There was no correlation between the visual acuity at the far point and survival time (ρ=0.205, p>0.10) or past susceptibility and survival time (ρ=-0.388, p>0.10). Again, the visual acuity correlation should be treated with caution because of the limited range of visual acuity among the subjects and the influence of one outlier on the correlation. Figure 7.5 shows visual acuity at the near point and survival time.

7.4.6 Cox's proportional hazards model

A Cox regression model, with survival time defined as the time taken to reach 2 (mild symptoms, e.g. stomach awareness but not nausea), was formed for each of the two conditions, with visual acuity at the near point as a covariate. The data was not split into low and high categories because there was only 1 subject in the low category (less than 20:20 vision). The visual acuity data were entered into the model as the individual scores recorded for subjects. There was 1 subject with 20:30 vision, 4 subjects with 20:20 vision and 11 subjects with 20:15 vision.

In the full field of dots condition, a significant effect of acuity was found, with subjects with poor acuity surviving for a shorter period of time (Cox regression, p<0.05). The Cox's proportional hazards model for the full field condition is shown in Table 7.2. In the single dot condition, there was a marginally significant effect of visual acuity on survival time (Cox regression, p<0.10) with subjects with poor acuity surviving for a shorter period before reaching 2 on the motion sickness scale. The Cox's proportional hazards model for the single dot condition is also shown in Table 7.2.

Table 7.2. Cox's proportional hazards model.

Condition – expt 4	**Independent variables**	e^{β}	**Sig (β)**
Single dot	Visual acuity at the near point (%)	0.9648	0.0692
Multiple dots	Visual acuity at the near point (%)	0.9478	0.0332

7.5 *Discussion and conclusions*

In previous experiments, vection and motion sickness have been shown to be separate phenomena which were not correlated. Motion sickness was shown to be reduced with a fixation point, without varying the vection perceived. The model presented at the end of Chapter 6 predicted that vection could also be varied without varying the symptoms of motion sickness. This experiment confirmed that possibility. Vection was found to be significantly different between the two conditions, with more vection found in the full field condition where there was a greater stimulation of the periphery.

Motion sickness was not significantly different between the two conditions. The stimulation of the fovea and the eye movements made in response to the two conditions were similar. It is not possible to state on the basis of this experiment whether image slip on the fovea or eye movements themselves are responsible for the motion sickness.

A correlation between visual acuity and survival time was noted again in this experiment in the full field condition, with a marginally significant correlation found in the single dot condition. The finding, on the basis of this experiment alone, should be treated with extreme caution because of the influence of a single subject on the correlations. There was only one subject with visual acuity below 20:20. This subject had relatively low survival times in each of the two conditions, which resulted in the significant, or marginally significant correlations being found. Removing the subject from the correlations decreased the level of significance to levels of $p>0.10$.

7.5.1 Comparison of the accumulated illness ratings with previous experiments

The accumulated illness ratings in this experiment were quite low by comparison with the previous experiments. The mean illness ratings were 19.9 (single dot) and 22.8 (multiple dots) compared to 38.9 (virtual condition – experiment 1) and 40.7 (no fixation – experiment 2). The difference in motion sickness incidence cannot be easily explained. The subjects in each experiment were largely independent, the stimulus velocity was slightly lower in this experiment, the stimulus was easier to track because of the discrete dots supplying obvious fixation points and all but one subject had 20:20 vision or greater in this experiment. The reduction in peripheral

vision in both conditions (discrete dots as compared to the full screen of stripes used previously) should not be ruled out as a possible cause of the difference, although vection was significantly greater with the full dot condition, indicating that there was significantly greater stimulation of the peripheral vision.

7.6 Updated model

The model is unchanged from chapter 6. Two possible routes to motion sickness still exist, via eye movements or foveal image slip, and will need further experimentation to discover which of the two is the dominant route. Vection and motion sickness are confirmed as separate outputs which can be manipulated independently of each other. Vection is confirmed as a mainly peripheral phenomenon while motion sickness has been shown to be foveally influenced, either by foveal image slip or via nystagmus (which shows foveal dominance). The model is shown in Figure 7.6.

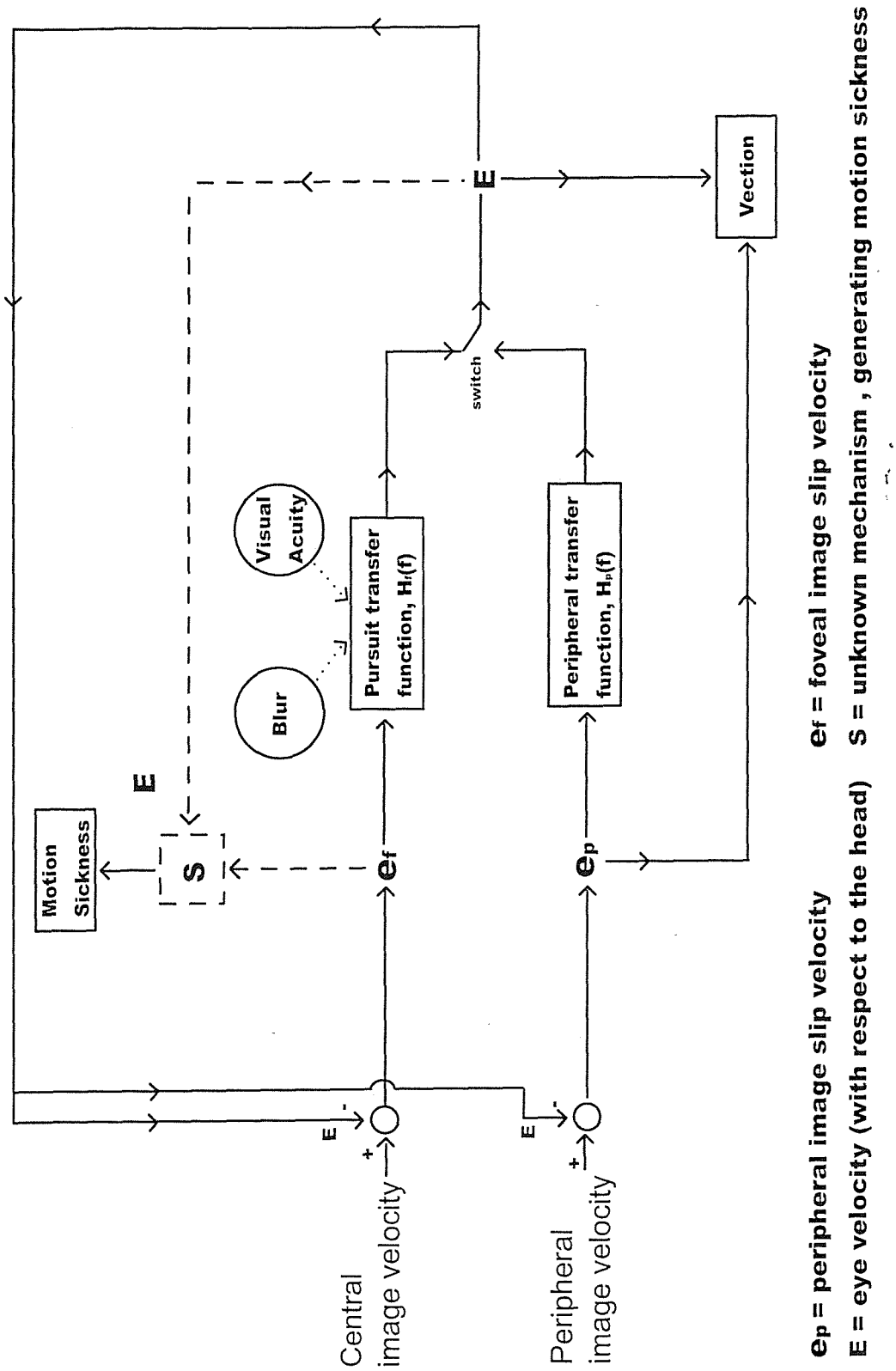

Figure 7.6. Model version 3. The model is unchanged from Chapter 6. The prediction that vection could be changed without changing motion sickness was confirmed.

Chapter 8. Comparison of motion sickness with and without vision correction

8.1 Introduction

The previous experiments have shown a possible influence of visual acuity on motion sickness survival time, with poorer acuity being associated with shorter survival times. The model shown at the end of Chapter 7 shows the hypothesis that visual acuity and possibly artificial blur, act to influence the pursuit component of the slow phase of nystagmus. It was necessary to understand more about the influence of visual acuity, for instance whether it is only poor sensitivity to high spatial frequencies at high contrast (as measured by the Landolt broken ring test) which influence motion sickness, or whether it is an effect which occurs across a broad range of spatial frequencies at varying contrast. Subjects were tested with and without their corrective spectacles or contact lenses and completed contrast sensitivity tests, to measure their contrast sensitivity at a range of spatial frequencies, in addition to the standard vision tests used previously.

By studying the model (Chapter 7), it was hypothesised that only sensitivity to high spatial frequencies would be correlated to motion sickness incidence because of the proposed influence of the fovea, which is responsible for detection of high spatial frequencies, on eye movements. It was also predicted that subjects without their vision correction would experience greater motion sickness symptoms. Vection was predicted to be similar in the two conditions.

8.2 Method

8.2.1 Pre – exposure tests

Twenty subjects aged 18 to 33 years were selected on the basis that they wore spectacles or contact lenses in everyday life. The visual tests were administered as in all the previous experiments. An additional test was performed: the 'Arden' test of contrast sensitivity (Skalka, 1981). The test was performed at a distance of 0.5m with each eye measured separately, at each frequency. The test consisted of cards with vertical bar gratings whose darkness varied sinusoidally from grey to darker grey (see Figure 8.1), with the contrast between the darkest and lightest areas increasing along the vertical length of the card. As the card was withdrawn from a holder, the

difference in contrast gradually became more discernible. The experimenter exposed the card and the subject indicated to the experimenter when it was possible to see the difference in contrast (i.e. it no longer looked all one shade of grey). A number was read from the card at that point to give a score. The maximum score was 20. If a subject did not see the contrast at 20 (with the full card exposed) then the arbitrary figure of 25 was assigned as the score for that card, as per the Arden test instructions. Each successive card had a higher spatial frequency. The six spatial frequencies tested were 0.3, 0.6, 1.25, 2.5, 5 and 10 cycles / degree.

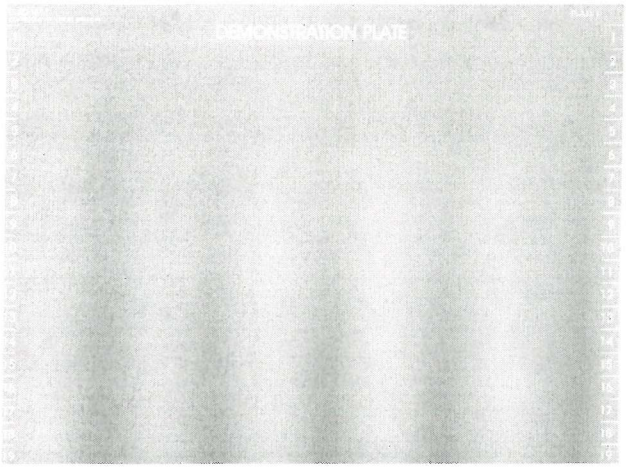

Figure 8.1. Arden test of contrast sensitivity.

All tests were performed with and without visual correction (i.e. spectacles or contact lenses). Subjects were asked to provide a copy of the prescription for their spectacles or contact lenses. They also completed the motion sickness history questionnaire.

8.2.2 Exposure sessions

Two exposure sessions consisted of 20 minutes in the optokinetic drum rotating clockwise at 5 r.p.m. Subjects viewed the drum with vision correction for one session and without vision correction for another session. The two sessions were at least two weeks apart to help minimise any habituation effects. Ten subjects commenced by viewing without vision correction and the other 10 commenced viewing with vision correction.

Subjects reported motion sickness scores as in all previous experiments and vection scores on the percentage scale as used in Experiment 4 (Table 7.1). During the exposure period, subjects were viewed on a video monitor to ensure that they had their eyes open and were looking straight ahead. Immediately after exposure, subjects completed a post exposure symptoms questionnaire to indicate symptoms experienced during exposure.

8.3 Analysis

Average vection and accumulated illness ratings were calculated as previously. Motion sickness, vection scores across conditions and comparisons of visual acuity across conditions were analysed using the Wilcoxon matched-pairs signed ranks test. Spearman's rank correlation test was used to test the relationships between vection and motion sickness in conditions. Survival analysis was performed as in previous experiments, with the addition of the contrast sensitivity scores.

8.4 Results

8.4.1 Contrast sensitivity vs. visual acuity

Subject visual acuity at the near point was significantly different with and without vision correction (Wilcoxon, $p<0.000$) (i.e. all subjects had poorer acuity at the near point without correction). Contrast sensitivity scores were significantly different with and without vision correction (Wilcoxon, $p<0.01$) with the exception of the lowest measured frequency of 0.3 cycles per degree, which was marginally significantly different (Wilcoxon, $p<0.10$).

Correlations between visual acuity and contrast sensitivity at the different spatial frequencies *without correction* were increasingly significant with increasing spatial frequency. The correlations are shown in Table 8.1. Correlations are negative because a high score on the Arden contrast sensitivity test corresponds to poor vision, whereas a high score on the acuity test corresponds to good vision.

The contrast sensitivity scores *with* correction did not correlate with visual acuity – possibly because there was very little variation in the visual acuity scores with correction. Only one subject had worse than 20:20 vision with correction.

Table 8.1. Correlations between Landolt acuity and contrast sensitivity scores at varying spatial frequency, without vision correction.

Spatial frequency (cycles per degree)	Correlation with visual acuity (as measured by the Landolt broken ring test)
0.30	ρ= -0.172, p = 0.468
0.60	ρ= -0.573, p = 0.008
1.25	ρ= -0.703, p = 0.001
2.50	ρ= -0.672, p = 0.001
5.00	ρ= -0.692, p = 0.001
10.0	ρ= -0.766, p = 0.000

8.4.2 Motion sickness

The accumulated illness ratings were significantly higher when subjects did not wear their spectacles – a mean of 35.1 without correction and 21.5 with correction

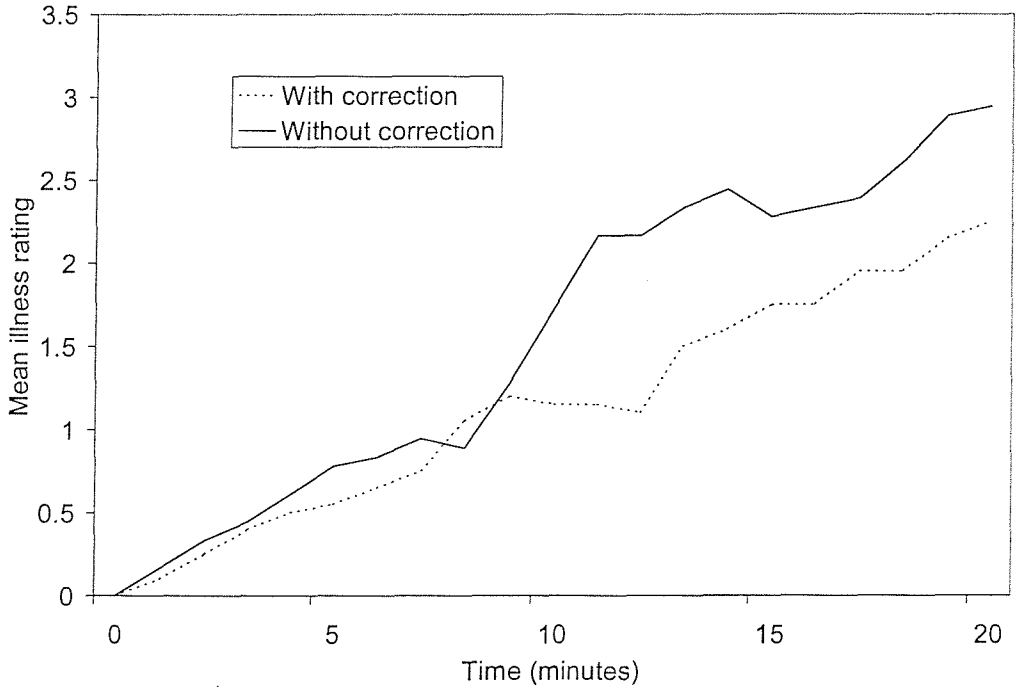

Figure 8.2. Mean illness ratings against time for both conditions.

(Wilcoxon, p<0.05). Post exposure symptoms were significantly higher when subjects did not wear spectacles (Wilcoxon, p<0.05). Motion sickness scores across the two conditions were significantly correlated (ρ=0.650, p<0.01). Motion sickness scores

were not correlated with vection scores in either the corrected vision (ρ=-0.114, p>0.10) or in the uncorrected vision condition (ρ=-0.004, p>0.10). Figure 8.2 shows the mean illness ratings against time for the two conditions.

8.4.3 Vection

There was no difference in the vection scores for the two conditions (Wilcoxon, p>0.10). Vection scores across the two conditions were significantly correlated (ρ= 0.623, p<0.01).

8.4.4 Survival analysis – uncorrected vision

In the uncorrected vision condition, subject visual acuity scores at the near point were correlated with survival time for the uncorrected vision condition (ρ= 0.480, p<0.05). As found previously, subjects had lower survival times if they had lower acuity. Subject scores for the two lowest frequencies of contrast sensitivity (0.3 and

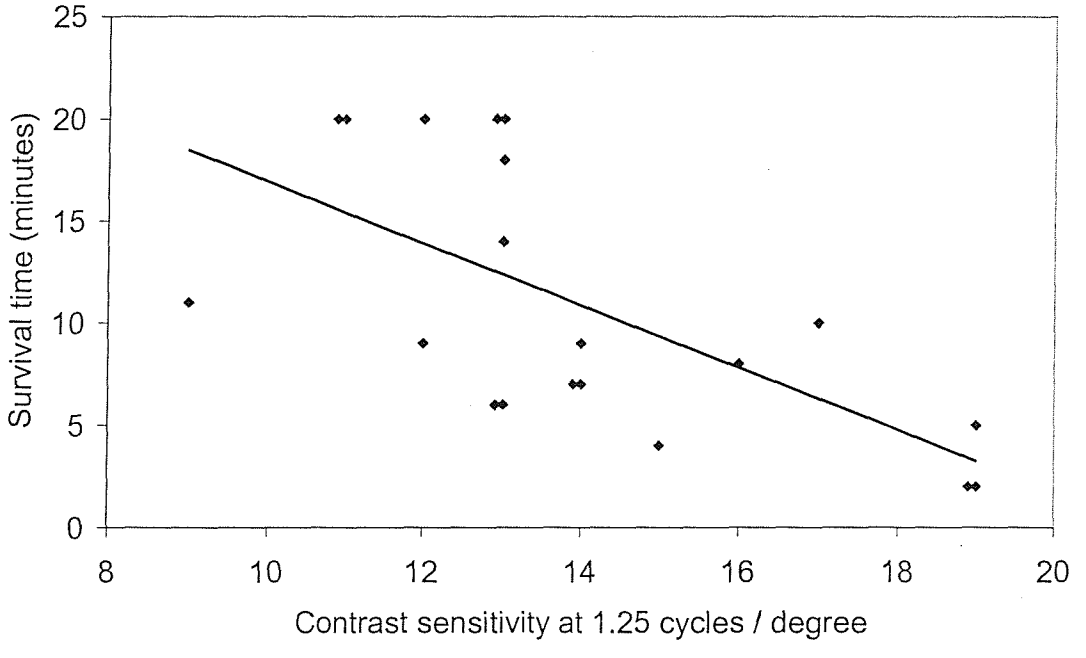

Figure 8.3. Survival times for various subject acuity scores at the 1.25 cycles per degree spatial frequency. Uncorrected vision condition.

0.6 cycles/°) were not correlated with motion sickness but the scores at the four highest spatial frequencies were either significantly correlated or there was a marginally significant correlation (the correlations are shown in Table 8.2.).

With each of the spatial frequencies those subjects with poorer contrast sensitivity had lower survival times. With the exception of the 1.25 cycles/° spatial frequency, the correlations become stronger with increasing spatial frequency. The contrast sensitivity score at the spatial frequency of 1.25 cycles/° was correlated very strongly with motion sickness survival time, see Figure 8.3.

Table 8.2. Correlations between contrast sensitivity scores and time taken to reach number 2 on the motion sickness scale for the uncorrected vision condition.

Spatial frequency (cycles per degree)	Correlation
0.30	ρ = -0.158 p = 0.507
0.60	ρ = -0.324 p = 0.164
1.25	ρ = -0.726 p = 0.000
2.50	ρ = -0.418 p = 0.067
5.00	ρ = -0.423 p = 0.063
10.0	ρ = -0.560 p = 0.010

8.4.5 Survival analysis – corrected vision

In the corrected condition, visual acuity at the near point was not correlated with motion sickness (ρ=0.298, p>0.10), however there was a much smaller range of visual acuity scores with corrected vision (only one subject had a score of lower than 20:20 with correction). There were no significant correlations between survival times and contrast sensitivity scores (the statistics are shown in Table 8.3). Motion sickness susceptibility ratings derived from the history questionnaire were marginally significantly correlated with motion sickness survival times (ρ=-0.381 p<0.10).

Table 8.3. Correlations between contrast sensitivity scores and time taken to reach number 2 on the motion sickness scale for the corrected vision condition.

Spatial frequency (cycles per degree)	Correlation with survival time (corrected vision condition)
0.30	ρ = -0.187 p = 0.430
0.60	ρ = -0.133 p = 0.576
1.25	ρ = -0.021 p = 0.929
2.50	ρ = -0.043 p = 0.858
5.00	ρ = -0.002 p = 0.994
10.0	ρ = -0.094 p = 0.693

8.4.6 Cox's proportional hazards model

For the uncorrected vision condition, the visual acuity data and contrast sensitivity variables, at 1.25 and 10 cycles per degree, were added into a Cox regression model. It was found that the contrast sensitivity score recorded at 1.25 was significantly influencing survival time, with poorer vision resulting in a decreased survival time, as expected. The visual acuity at the near point and the contrast sensitivity at 10 cycles / degree were not found to be significant influences in this Cox regression model when included with the contrast sensitivity data at 1.25 cycles / degree, although were significant when included individually. This indicates that, of the three variables, the contrast sensitivity score at 1.25 cycles per degree was the most significant influence on survival time. The Cox's proportional hazards model is shown in Table 8.4.

In the corrected vision condition, the marginally significant correlation between past susceptibility and survival time, was investigated with a Cox regression model. No influence of past susceptibility was found on survival time by the Cox regression model. The data are shown in Table 8.4.

Table 8.4. Cox's proportional hazards model for both conditions.

Condition	Independent variables	e^{β}	Sig (β)
Uncorrected vision	Contrast sensitivity at 2.5 cycles per degree	1.518	0.0008
Corrected vision	Past susceptibility	1.0275	0.1775

8.5 Discussion

8.5.1 Corrected vs. uncorrected vision

Motion sickness was significantly higher in the condition without vision correction as expected from the previous experiments and model. Vection was no different between conditions and the vection scores were uncorrelated with motion sickness scores, again as expected from the model. The influence of visual acuity on motion sickness was found in the uncorrected vision condition where there was a wide range of acuity scores. It was not found in the corrected vision condition, probably due to the small variation in acuity (all the subjects, with the exception of one, had better than 20:20 vision).

8.5.2 Contrast sensitivity vs. visual acuity

There were correlations found between the Landolt measure of visual acuity and contrast sensitivity at all but the lowest spatial frequency. This was only the case in the uncorrected vision condition, where the majority of subjects had visual acuity scores in the range of 20:200 (low) to 20:30 (high), which correspond to spatial frequency limits of 6 cycles per degree to 40 cycles per degree. The increasingly significant correlations between visual acuity and contrast sensitivity at the higher spatial frequencies measured (i.e. 5-10 cycles per degree) may possibly occur because these higher frequencies fall within the range of 6-40 cycles per degree, i.e. the upper limit of visual acuity measured for these particular subjects.

In the case of visual acuity measured with corrected vision, the high scores in the visual acuity test, where 20:20 vision corresponds to a spatial frequency of 60 cycles per degree (i.e. 1 minute of visual arc) may not have been expected to correlate with the low and medium spatial frequency scores.

8.5.3 Contrast sensitivity and motion sickness survival.

Generally the higher contrast sensitivity scores were more highly correlated with motion sickness survival time than the low frequency scores. The two lowest frequencies (0.3 and 0.6 cycles per degree) were not significantly correlated. The higher frequencies were all correlated or marginally correlated with survival time. Correlation coefficients increased with spatial frequency, with the exception of the 1.25 cycles per degree spatial frequency where a highly significant correlation was found between survival time and visual acuity at that frequency (ρ=0.726, p<0.000). Whether this is a chance result, or a more significant finding is not known at this stage.

8.5.4 The possible effect of spectacle magnification on motion sickness

There were 17 subjects (out of the total of 20) who wore spectacles in this experiment, whilst the remaining 3 wore contact lenses. Spectacles have the effect of either minimising or magnifying the image seen through them. This does not occur with contact lenses because they fit directly onto the eye. A possible reason for the difference in motion sickness between the two conditions could be the difference in image magnification or minification. However, in the uncorrected vision condition, all subjects viewed the optokinetic drum without vision correction. The heads of subjects were restrained in all conditions, so the vestibulo-ocular reflex response was not activated. In this condition, visual acuity and contrast sensitivity scores were correlated with motion sickness survival time. This suggests that the effect of visual acuity and contrast sensitivity to the higher spatial frequencies occur independently of a possible separate effect of image magnification.

8.6 Conclusion and updated model

It may be concluded that the influence of vision on motion sickness is based mainly on lack of sensitivity to medium to high spatial frequencies (i.e. poor resolution of fine images on the fovea). The result from Experiment 3 (Chapter 6) where an increase in post exposure symptoms were found with artificial blurring of the stimulus, is consistent with the result from this experiment (i.e. that motion sickness survival is mostly correlated with lack of sensitivity to higher spatial frequencies, which are missing with poor acuity, and were artificially removed by blurring).

8.6.1 Updated model

The model has been updated in a simple way by simply adding 'low pass filter' symbols to the 'acuity' and 'blur' inputs to indicate that these are both methods of reducing the high frequency information available on the fovea, which in turn may be responsible for the influence on motion sickness. The model is presented in Figure 8.4.

The reason for the increase in motion sickness with poorer contrast sensitivity to high spatial frequencies is not known, although the model suggests a hypothesis: that reduced sensitivity to high spatial frequencies may reduce the influence of the fovea on the control of the slow phase of nystagmus. Nystagmus gain has been shown to be lower with reduced input from the fovea (see Section 2.3.7.2 for a full review), so in the case of poor contrast sensitivity to high spatial frequencies, the gain of the slow phase of eye movements may be lower. If this is the case then the velocity of image slip on the fovea (and peripheral retina) will be greater with poorer visual acuity. In the model, two inputs to motion sickness still remain: (i) via foveal image slip (ii) via eye movements. The 6th and final experiment, presented in the next chapter addresses the possibility that eye movements may vary with visual acuity and contrast sensitivity to high spatial frequencies.

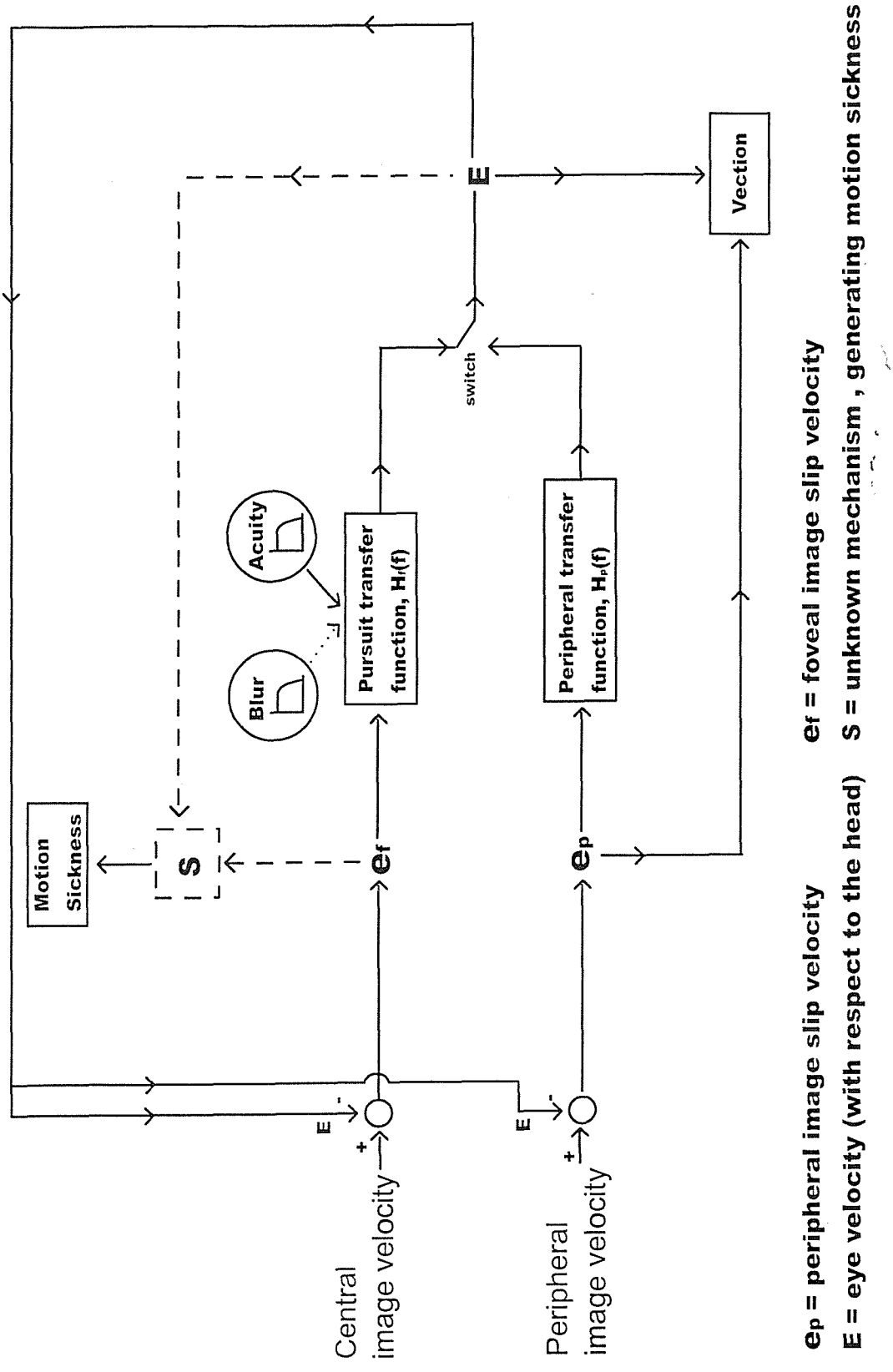

Figure 8.4. Model version 4. Taking into account the correlation between contrast sensitivity to high spatial frequencies and motion sickness.

Chapter 9. Experiment 6. Comparison of the slow phase velocity of nystagmus with and without vision correction

9.1 Introduction

The model presented in Chapter 8 (Figure 8.4) suggested a possible route for the influence of visual acuity, or contrast sensitivity to high spatial frequencies, on the slow phase of nystagmus. The model predicted that contrast sensitivity to high spatial frequencies may influence the velocity of the slow phase of nystagmus. It leads to the hypothesis that the gain of the slow phase (velocity of the slow phase divided by the velocity of the optokinetic drum) may be lower with decreased contrast sensitivity to high spatial frequencies (i.e. reduced foveal acuity).

The final experiment of this thesis aimed to test the hypothesis that visual acuity or contrast sensitivity can influence the slow phase velocity of nystagmus in response to motion of an optokinetic drum. Eye movements were recorded with an accuracy of 1 minute of visual angle (Reulen *et al.,* 1988), using an infra-red corneal reflection system (IRIS), with and without vision correction.

9.2 Method

9.2.1 Pre–exposure tests

Thirteen male subjects, aged 18-25, were selected on the basis that they wore spectacles or contact lenses in everyday life. They completed the visual acuity and Arden contrast sensitivity tests as performed in the previous experiment.

9.2.2 Exposure sessions

Two exposure sessions consisted of 5 minutes in the optokinetic drum rotating clockwise at 35 degrees per second (slightly greater than the 5 r.p.m. used in previous experiments). Subjects viewed the drum with their spectacles or contact lenses on for one session, followed by a 20 minute rest period, then viewed the same optokinetic stimulation without their spectacles or contact lenses. Six subjects commenced viewing with their vision corrected, and seven subjects commenced viewing with their vision uncorrected.

Subjects reported motion sickness scores each minute on the 7 point scale used previously and vection scores on the percentage scale as used in Experiments 4 and 5. During the exposure period, subjects were viewed on a video monitor to ensure that they had their eyes open and were looking straight ahead. Subjects did not complete motion sickness history questionnaires or post exposure symptom questionnaires. Symptoms were recorded during the five minute exposures merely to ensure that susceptible subjects did not reach excessive nausea or vomiting.

During exposures, eye movements were recorded using an IRIS (Skalar Medical Company) infra-red corneal reflection system, as described in Chapter 3. This allowed a resolution of 1 minute of visual angle of eye movement to be recorded, without the drift problems commonly associated with electro-oculography systems. The eye movements for each eye were recorded using an *HVLab* Data acquisition system at a sample rate of 300 samples per second, with a low pass filter cut off at 100Hz. Eye movements were calibrated for each eye separately before and after exposure by asking subjects to look at 3 crosses marked horizontally on a wall in front of them. The first cross was directly in front of the subject (between the two eyes) and the other crosses were at 15° visual angle symmetrically either side. Subjects made eye movements between the crosses at the verbal request of the experimenter. The calibrations were also recorded to the *HVLab* system at 300 samples per second.

The drum velocity was 35°/second, slightly higher than previous experiments where it was 30°/second. This higher speed was initially used in error for the first subject, in place of the 30°/second speed previously used, and then maintained for the remaining subjects.

9.3 Analysis

9.3.1 Eye movements

Only the data from the left eye were analysed for each subject because the infra-red sensor on the right eye had a tendency to move during the exposure. This was apparent by looking at the position of the sensors and confirmed by studying the calibration data before and after exposure. The left eye calibrations were consistent and were hence used for the analysis. Eye movements were analysed manually by

inspection of the data files. A system was devised to ensure the values found were free from bias:

- Eye movement recordings were modified with reference to the calibration data corresponding to each file in order to make each file displayed as visual angle against time.
- The first 10 slow phase eye movements each minute, for each subject, were analysed.
- The slope of the slow phase was calculated by taking a point 0.02 seconds from the start and 0.02 seconds from the end of the slow phase, finding the difference in visual angle (in degrees).
- The difference in angle was divided by the time between the two points to give a slow phase eye velocity in degrees / second.
- The measurements were performed without any reference to individual visual acuity data for the subjects.

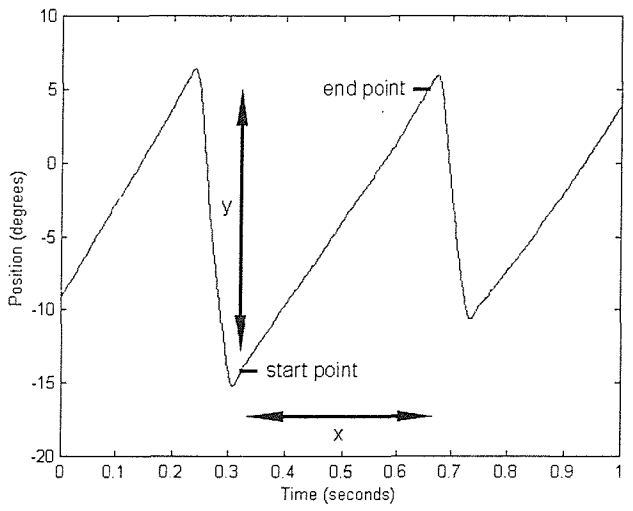

Figure 9.1. Calculation of slow phase velocity. Velocity = y divided by x. For illustration only – start and end points are not exact. Data shown is the first second for one subject.

Figure 9.1 illustrates the process used to calculate the velocity of the slow phases. The first 10 slow phases of each minute were taken for a total of five minutes per subject giving a total of 50 measurements in each of the corrected and uncorrected conditions. A mean nystagmus frequency for each minute was also calculated by counting the number of slow phases which occurred in the first 10 seconds of each minute and dividing by 10 to calculate number of eye movements per second.

9.3.2 Statistics

Friedman tests were used to test whether there was any significant difference between the slow phase velocities recorded each minute, whether there was any

significant difference between nystagmus frequencies recorded each minute and whether there was any significant difference between the subjective vection scores reported by subjects each minute.

An overall mean velocity was calculated for each subject from the 50 slow phase velocities found from the above procedure. The mean velocities for each subject for the two conditions were compared using the Wilcoxon matched-pairs signed ranks test. Nystagmus frequencies and motion sickness scores for the two conditions were also compared using the Wilcoxon matched-pairs signed ranks test.

Spearman's rank correlation test was used to study correlations between visual acuity, contrast sensitivity scores, mean nystagmus frequency and mean slow phase velocity.

9.4 Results

9.4.1 Motion sickness

There was no significant difference found in the accumulated illness ratings between conditions. This was not surprising given the very short exposure durations (Wilcoxon, $p>0.10$).

9.4.2 Eye movements

The mean slow phase velocity was 30.8°/second in the corrected vision condition and 29.26°/second in the uncorrected condition. The mean velocity of each subject (calculated from the 50 measurements of slow phase velocity per subject, in each of the conditions) was not significantly different between the uncorrected vision and the corrected vision condition (Wilcoxon, $p>0.10$). The frequency of nystagmus was not significantly different between the two conditions (Wilcoxon, $p>0.10$).

9.4.3 Friedman test

In order to test whether the slow phase velocities, nystagmus frequencies and subjective vection scores varied during the short exposure time, Friedman tests were performed. The results of the Friedman tests showed that there was no change in the

slow phase velocity of nystagmus over the five minute period with correction (Friedman, $p>0.10$) or without correction (Friedman, $p>0.10$). There was no change in the frequency of nystagmus measured over the 5 minute period with correction (Friedman, $p>0.10$) or without correction (Friedman, $p>0.10$). There was a significant difference in the subjective vection scores recorded each minute with correction (Friedman, $p<0.000$) and without correction (Friedman, $p<0.000$). Study of the vection scores indicated that vection increased during the five minute exposure periods.

9.4.4 Spearman's rank correlation test

Visual acuity, contrast sensitivity scores, the mean nystagmus frequency and the mean slow phase velocity were tested for significant correlations, using Spearman's rank correlation.

9.4.4.1 Slow phase velocity - uncorrected vision condition

In the uncorrected vision condition, correlations were found between slow phase velocity and visual acuity measured at the near point ($\rho=0.728$, $p<0.01$), between slow phase velocity and contrast sensitivity at 1.25 cycles/° ($\rho=-0.649$, $p<0.05$) and between slow phase velocity and contrast sensitivity at 10 cycles/° ($\rho=-0.554$, $p<0.05$). There was a marginally significant correlation between slow phase velocity and contrast sensitivity at 2.5 cycles/° ($\rho=-0.491$, $p=0.088$). No significant correlations were found between slow phase velocity and visual acuity at the far point or between slow phase velocity and contrast sensitivity at 0.3,0.6,1.25 or 5 cycles/°. The correlations are shown in Table 9.1. A plot of slow phase velocity against visual acuity at the near point is shown in Figure 9.2 and a plot of slow phase velocity against contrast sensitivity at 1.25 cycles/° is shown in Figure 9.3.

Table 9.1. Correlations between slow phase velocity, visual acuity and contrast sensitivity (uncorrected vision).

Vision measurement, without correction	Correlation with slow phase velocity
Visual acuity at the near point	ρ=0.728 , *p*=0.005
Visual acuity at the far point	ρ=0.375 , *p*=0.206
Contrast sensitivity at 0.3 cycles/°	ρ=-0.088, *p*=0.775
Contrast sensitivity at 0.6 cycles/°	ρ=-0.455, *p*=0.118
Contrast sensitivity at 1.25 cycles/°	ρ=-0.649 , *p*=0.016
Contrast sensitivity at 2.5 cycles/°	ρ=-0.491 , *p*=0.088
Contrast sensitivity at 5.0 cycles/°	ρ=-0.397 , *p*=0.179
Contrast sensitivity at 10 cycles/°	ρ=-0.554 , *p*=0.050

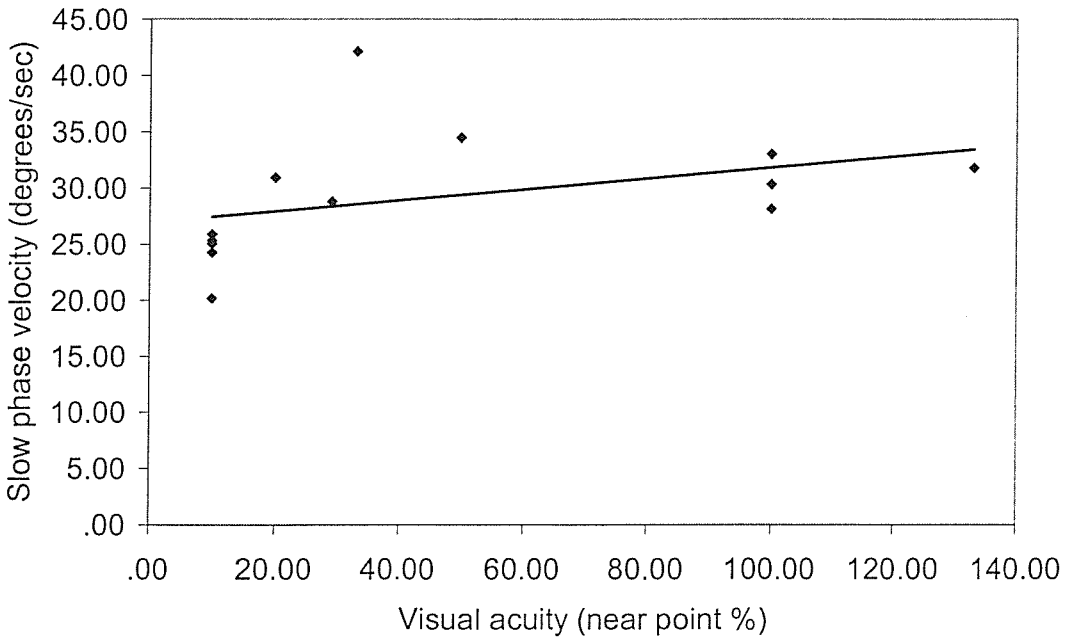

Figure 9.2. Variation of slow phase velocity for varying visual acuity, measured at the near point.

The significant correlation found between slow phase velocity and visual acuity at the near point was positive, indicating better visual acuity was associated with greater slow phase velocity. The correlations between slow phase velocity and contrast sensitivity scores were negative, also indicating that better contrast sensitivity (a lower score) was associated with a greater slow phase velocity.

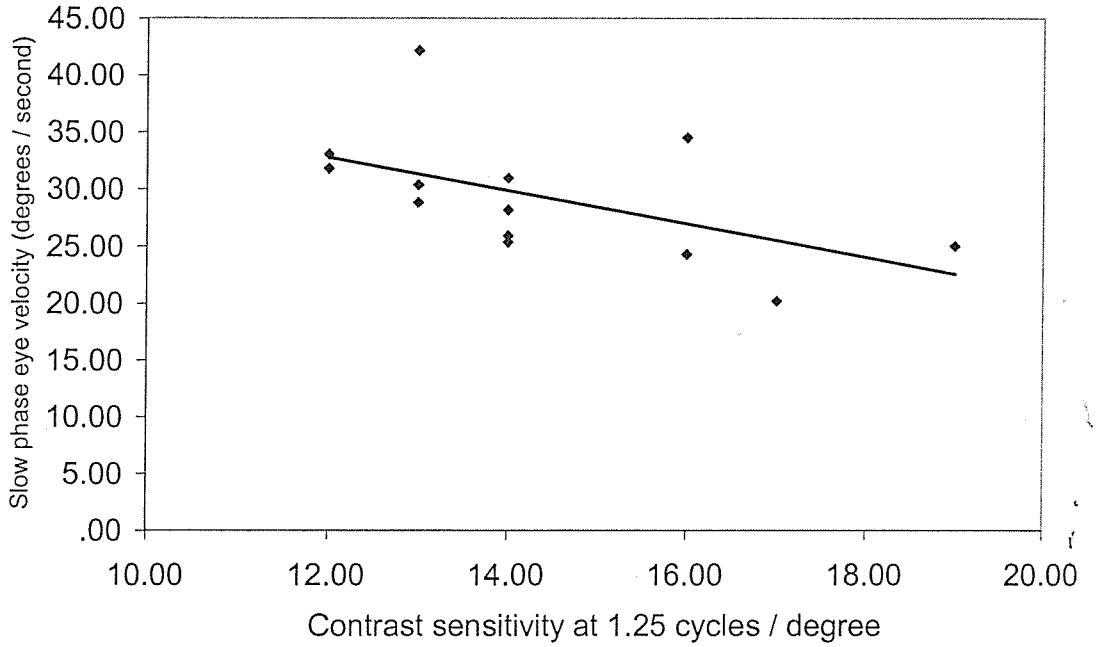

Figure 9.3. Variation of slow phase velocity with varying contrast sensitivity to 1.25 cycles per degree spatial frequency.

9.4.4.2 Slow phase velocity - corrected vision condition

In the corrected vision condition, no significant correlation was found between slow phase velocity and visual acuity at the near point (ρ=-0.231, p>0.10), nor between slow phase velocity and visual acuity at the far point (ρ=-0.231, p>0.10) (each subject had the same visual acuity at the near and at the far point, with correction, hence the correlations were the same). Significant correlations were found between slow phase velocity and contrast sensitivity at 0.3 cycles/degree (ρ=-0.609, p<0.05 – see Figure 9.4), between slow phase velocity and contrast sensitivity at 2.5 cycles/° (ρ=-0.598, p<0.05) and between slow phase velocity and contrast sensitivity at 5 cycles/° (ρ=-0.598, p<0.05 – see Figure 9.5). There was a marginally significant correlation between slow phase velocity and contrast sensitivity at 10 cycles/° (ρ=-0.549, p=0.052). No significant correlations were found between slow phase velocity and contrast sensitivity at 0.6 and 1.25 cycles/°. The correlations are shown in Table 9.2.

Table 9.2. Correlations between slow phase velocity visual acuity and contrast sensitivity scores (corrected vision condition).

Vision measurement, with correction	Correlation with slow phase velocity
Visual acuity at the near point	ρ=-0.231, *p*=0.447
Visual acuity at the far point	ρ=-0.231, *p*=0.447
Contrast sensitivity at 0.3 cycles/°	ρ=-0.609, *p*=0.027
Contrast sensitivity at 0.6 cycles/°	ρ=-0.469, *p*=0.106
Contrast sensitivity at 1.25 cycles/°	ρ=-0.527, *p*=0.064
Contrast sensitivity at 2.5 cycles/°	ρ=-0.575, *p*=0.040
Contrast sensitivity at 5.0 cycles/°	ρ=-0.598, *p*=0.031
Contrast sensitivity at 10 cycles/°	ρ=-0.549, *p*=0.052

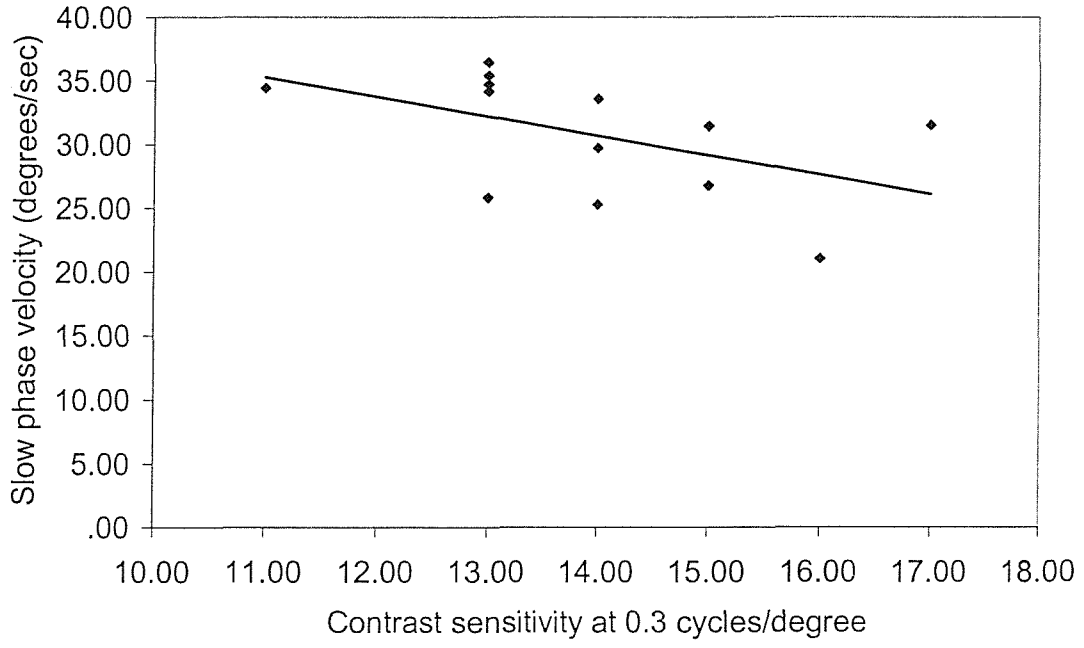

Figure 9.4. Variation of slow phase velocity with contrast sensitivity at 0.3 cycles/° in the corrected vision condition.

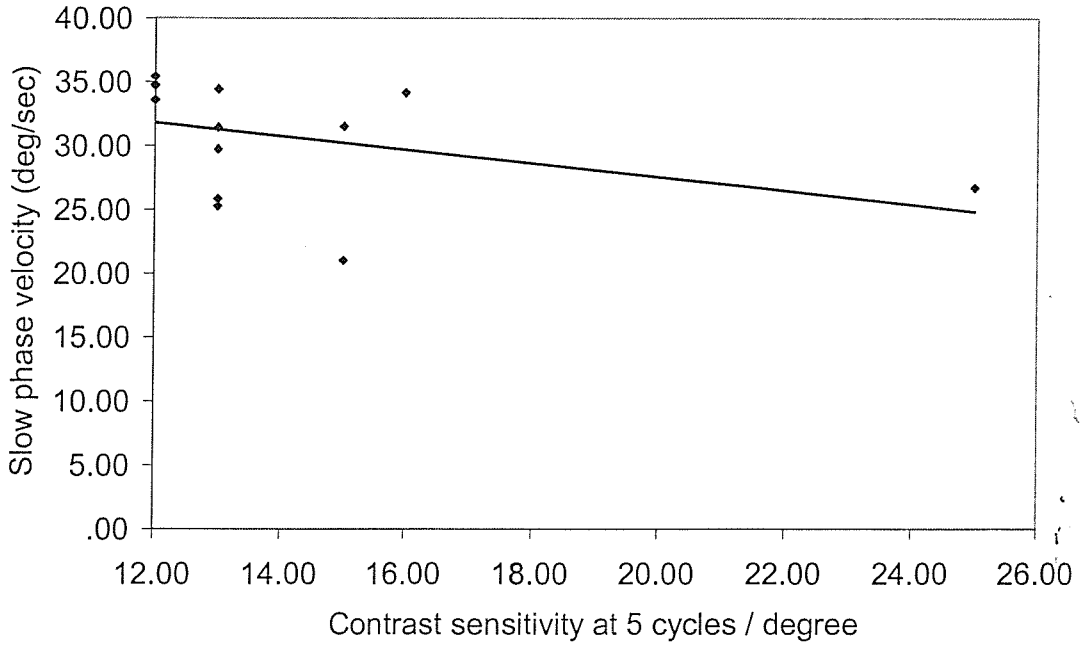

Figure 9.5. Variation of slow phase velocity with contrast sensitivity at 5 cycles/°, in the corrected vision condition.

9.4.4.3 Nystagmus frequency – uncorrected condition

In the uncorrected condition, there were no significant correlations between nystagmus frequency and visual acuity or contrast sensitivity scores. There was a marginally significant correlation between nystagmus frequency and visual acuity at the near point (ρ=0.512, p<0.10). There was no significant correlation found between nystagmus frequency and slow phase velocity (ρ=0.432, p>0.10) or between nystagmus and vection (ρ= -0.008, p>0.10). The correlations are shown in Table 9.3.

9.4.4.4 Nystagmus frequency – corrected vision condition

In the corrected vision condition, there were no significant correlations found between nystagmus frequency and visual acuity or contrast sensitivity scores. There was no significant correlation between nystagmus frequency and slow phase velocity, nor between nystagmus frequency and vection (ρ= 0.004, p>0.10). The correlations are shown in Table 9.4.

Table 9.3. Correlations between nystagmus frequency and vision measurements, without vision correction.

Variable (without correction)	Correlation with nystagmus frequency
Visual acuity at the near point	ρ=0.512, *p*=0.073
Visual acuity at the far point	ρ=0.132, *p*=0.667
Contrast sensitivity at 0.3 cycles/°	ρ=0.068, *p*=0.825
Contrast sensitivity at 0.6 cycles/°	ρ=-0.326, *p*=0.276
Contrast sensitivity at 1.25 cycles/°	ρ=-0.250, *p*=0.409
Contrast sensitivity at 2.5 cycles/°	ρ=-0.282, *p*=0.351
Contrast sensitivity at 5.0 cycles/°	ρ=-0.082, *p*=0.789
Contrast sensitivity at 10 cycles/°	ρ=-0.331, *p*=0.269
Slow phase velocity	ρ=-0.473, *p*=0.102
Vection	ρ=-0.004, *p*=0.979

Table 9.4. Correlations between nystagmus frequency and vision measurements, with vision correction.

Variable (with correction)	Correlation with nystagmus frequency
Visual acuity at the near point	ρ=-0.270, *p*=0.372
Visual acuity at the far point	ρ=-0.270, *p*=0.372
Contrast sensitivity at 0.3 cycles/°	ρ=0.097, *p*=0.753
Contrast sensitivity at 0.6 cycles/°	ρ=0.103, *p*=0.738
Contrast sensitivity at 1.25 cycles/°	ρ=-0.473, *p*=0.102
Contrast sensitivity at 2.5 cycles/°	ρ=-0.416, *p*=0.158
Contrast sensitivity at 5.0 cycles/°	ρ=-0.182, *p*=0.551
Contrast sensitivity at 10 cycles/°	ρ=-0.144, *p*=0.639
Slow phase velocity	ρ=-0.432, *p*=0.141
Vection	ρ= 0.004, *p*=0.989

9.5 Discussion

9.5.1 Slow phase velocity

The model predicted that slow phase eye velocity may be influenced by sensitivity to higher spatial frequencies and predicted that the velocity would be lower with lower sensitivity. The simple comparison of eye velocities recorded during the corrected and uncorrected conditions showed that the velocity of eye movements were slower in the uncorrected vision condition but the difference was not significant, as measured by the Wilcoxon test.

In the uncorrected vision condition there was a wide range of both visual acuity and contrast sensitivity at all of the spatial frequencies measured. The Spearman's rank correlation test showed that there was a correlation between visual acuity at the near point and slow phase velocity, of contrast sensitivity to the highest spatial frequency (10 cycles/°) and of contrast sensitivity to the 1.25 cycles/° spatial frequency. The correlations indicated that increased contrast sensitivity or increased visual acuity resulted in an increase in the slow phase velocity of the eyes in response to the optokinetic drum. The correlation between slow phase velocity and visual acuity was the most significant of the correlations measured, followed by contrast sensitivity at 1.25 cycles/° and then by contrast sensitivity at 10 cycles/°. The trend from these results is consistent with sensitivity to high spatial frequencies influencing the slow phase velocity of nystagmus. The correlation at 1.25 cycles/° shows that there may also be an influence of medium spatial frequencies on the slow phase velocity.

In the corrected vision condition, significant correlations with slow phase velocity were found at 0.3 cycles/°, 2.5 cycles/° and 5 cycles/°, with a marginally significant correlation found at 10 cycles/°. The variation in contrast sensitivity was less with vision correction, but there was still greater variation among the contrast sensitivity scores with vision correction than among the visual acuity scores (where all but one subject had greater than 20:20 vision). The correlations also showed that better contrast sensitivity (a lower score) was correlated with increased slow phase velocity. The correlations found with vision correction were unexpected. They occurred mainly at the higher spatial frequencies measured, with the exception of the 0.3 cycles/° frequency.

9.5.2 Nystagmus frequency

There was no significant difference between the frequency of nystagmus found with or without vision correction or any influence of visual acuity or contrast sensitivity on nystagmus frequency found. This may indicate that nystagmus frequency is determined mainly by the spatial frequency of the drum (i.e. the spacing between black and white stripes) rather than visual acuity or contrast sensitivity. Hu *et al.* (1997) found that varying the number of black and white stripes painted in an optokinetic drum could alter the average nystagmus frequency generated when subjects viewed the drum rotating at a constant velocity.

9.5.3 Possible effect of spectacle magnification on slow phase velocity

As mentioned in Section 8.5.4, there is a magnification or minification of the image viewed through spectacles. In the current experiment the main conclusions are drawn from the correlations between slow phase velocity and visual acuity and between slow phase velocity and contrast sensitivity scores, both found in the uncorrected vision condition. In the uncorrected vision condition there was no effect of magnification or minification because the subjects did not wear their spectacles or contact lenses in this condition. A difference in slow phase velocity would not be expected to occur due to the previous experience of the subjects, because head movements were restricted to prevent activation of the vestibulo-ocular reflex. The optokinetic drum activates the optokinetic and pursuit reflexes which are dependent on visual feedback, not past experience of magnification, in order to operate. It is concluded that the correlation between slow phase velocity and visual acuity is independent of the effect of magnification or minification of spectacles.

9.6 Conclusions

The reduction in velocity of the slow phase with decreased contrast sensitivity to higher spatial frequencies, means that subjects with poorer contrast sensitivity were less likely to make eye movements matching the speed of the stimulus which they were attempting to track, in this case the optokinetic drum. The model predicted two possible inputs to motion sickness: (i) via foveal image slip (ii) via eye movements directly, as hypothesised by Ebenholtz *et al.* (1994). This experiment confirmed that

foveal image slip increased with reduced contrast sensitivity to high spatial frequencies, because of the inability of subjects with low contrast sensitivity to match the speed of the stimulus as effectively as those with high sensitivity to high spatial frequencies.

Since foveal slip velocity was correlated mainly with contrast sensitivity to high spatial frequencies, and motion sickness has been found to be influenced by visual acuity and contrast sensitivity, as discovered in the previous chapters, it is possible that a correlation would be found between motion sickness and foveal image slip velocity if the two were measured over a longer period than used in this experiment.

The hypothesis of Ebenholtz *et al.* (1994) that eye movements themselves are a cause of motion sickness, is less likely to be the route of the motion sickness effect. There were large differences between the motion sickness survival times of subjects with low and high acuity in previous experiments. However, the eye movements themselves were similar with and without vision correction. Variation in slow phase velocity of only a few degrees per second would result in a large increase in foveal image slip velocity, and perhaps a concomitant increase in motion sickness. Further discussion, and the final model, are presented in the next chapter.

The finding that contrast sensitivity was correlated with slow phase velocity in the corrected vision condition may indicate that the extra variation which occurred in the contrast sensitivity tests, compared with the visual acuity tests, could be used as a means of predicting the velocity of eye movements in response to a certain stimulus velocity , even when vision is corrected.

The slow phase velocity and nystagmus frequency did not change significantly during the five minute measurement periods in either the uncorrected or the corrected vision conditions. Vection did change during the same periods, in both conditions. This probably indicates that slow phase velocity, nystagmus frequency and vection are not related, as predicted from the initial model (Figure 2.22) and subsequent models. A final model is presented in the next chapter.

Chapter 10. Discussion and final model

10.1 Discussion

The aims of the thesis were to investigate the relationship between vection and motion sickness with optokinetic stimuli, to investigate the possible influence of eye movements on motion sickness, the potential influence of visual acuity and other visual characteristics on motion sickness, and additionally to investigate the possibility of using virtual reality as a tool for studying motion sickness.

10.1.1 Vection and motion sickness

The experimental work failed to show any significant correlations between the vection scores of subjects and the motion sickness scores, in any of the conditions. Motion sickness was significantly reduced with fixation (Chapter 5) but vection was unchanged. Eye movements did not occur during the fixation condition but did occur in the normal condition, as expected. With the single and multiple dot displays (Chapter 7), it was found that vection was significantly higher with multiple dots but motion sickness was not significantly different. In this experiment, eye movements and the foveal stimulus was the same in both conditions (i.e. a single dot) but the peripheral stimuli varied between the two conditions, with increased peripheral stimulation in the full field of dots condition. The results from the above experiments showed that not only were there no correlations between vection and motion sickness, but that vection and motion sickness can be independently manipulated.

Vection appears to be controlled mainly by detection of motion in peripheral vision, which increased in the multiple dot condition but was similar with or without fixation. This is in agreement with the literature, for example Brandt *et al.* (1973) found that presenting an optokinetic stimulus in peripheral vision resulted in greater vection than when the same stimulus was presented in central (foveal) vision (further information is available in Section 2.3.7.2).

Eye movements do not appear to significantly influence vection. Vection was unchanged with or without fixation, despite no eye movements occurring during fixation, and was increased in the multiple dot condition compared to the single dot condition, despite similar eye movements in each condition. This is in agreement with

the finding of Brandt *et al.* (1973) that subjects who tracked a central optokinetic stimulus, which moved in the opposite direction to the peripheral stimulus, experienced vection in the direction which was expected from the peripheral stimulation, despite eye movements which occurred in the opposite direction. Vection was not found to significantly vary depending on the frequency of nystagmus recorded (Chapters 4 and 9). This result differs from that found by Hu *et al.* (1998), who found that vection increased with increasing frequency of nystagmus.

An increase of vection with time was found for the first five minutes of exposure to the optokinetic drum (Chapter 9). Despite the change in vection found during the first five minutes, no significant change in the frequency of nystagmus or of the slow phase nystagmus velocity were found. This again indicates that vection was probably not influenced by nystagmus frequency or slow phase velocity.

Previous studies have not shown any direct correlation between motion sickness and vection although it is often implied or stated that they are correlated. The phrase 'vection-induced motion sickness' is often used in the literature without any direct evidence of a causal connection (for example Hu *et al.,* 1997). It is evident, from the results of the experimental work in this thesis, that vection and motion sickness are distinct phenomena. The assumption that motion sickness is caused by vection cannot be made and vection should not be studied as a substitute for studying motion sickness. The results from this study apply to optokinetic stimuli generating circular-vection. Vection generated during a simulation of forward motion in a car (known as linear vection) may possibly be correlated with motion sickness. Further work on linear vection may be interesting.

10.1.2 Visual acuity and eye movements

The final experiment, presented in Chapter 9, showed that visual acuity and contrast sensitivity to high spatial frequencies had an influence on the slow phase velocity of nystagmus. It was found that the slow phase velocity, in response to a constant speed of optokinetic drum, was lower when subjects had poorer sensitivity to high spatial frequencies (i.e. poorer acuity). This finding was predicted from previous studies, for example Van Die *et al.* (1986) found that the velocity of the slow phase was lower when the influence of the fovea was reduced. This was the case when the fovea was blocked with a moving mask, when viewing the drum in low level light to

stimulate only the peripheral vision, or by allowing subjects who had a central retinal scotoma in one eye to view the drum with their normal and their affected eye separately. Cheng *et al.* (1975) also found a decrease in slow phase velocity when a stimulus was moved an increasing distance from the fovea. Howard *et al.* (1984) found that the velocity of the slow phase was reduced when a central band was deleted from an optokinetic display.

These studies showed that optokinetic nystagmus may have a dual response. A response which is driven by peripheral vision with a lower gain, and a response driven by the fovea, which dominates, and which enables the eye to track at a velocity nearer to that of the stimulus (i.e. a higher gain). This idea is supported by Robinson (1981), who found that animals without foveas (such as rabbits) take a longer time to build up eye velocity in response to an optokinetic drum and generally make eye movements at a lower velocity than animals with foveas, such as chimps and humans. Visual acuity and contrast sensitivity to high spatial frequencies are measures of the resolution of the fovea, hence reduced visual acuity was expected to reduce the influence of the fovea on the velocity of the slow phase of nystagmus (i.e. to decrease the velocity). The findings of the final experiment confirmed this hypothesis, for the particular speed of drum motion employed (35°/second). Post *et al.* (1979) attempted to measure an effect of visual acuity on slow phase velocity, but used blurring lenses. They did not account for the magnifying effect of the lenses on the slow phase velocity, so may have been unable to discover any effect of visual acuity on slow phase velocity if it occurred. Other studies of eye movements, in response to optokinetic stimuli, have not measured visual acuity or the possible effect it may have on the slow phase velocity, despite it being a possibility from other studies (e.g. Van Die *et al.*, 1986). Visual acuity should be measured when making measurements of eye movements in response to optokinetic stimuli.

It was found that the frequency of optokinetic nystagmus did not vary significantly with time and was not influenced by the visual acuity of subjects. This may possibly indicate that the frequency of eye movements is not dependent on the slow phase velocity, which varied with visual acuity. This is in agreement with Pyykko *et al.* (1985) who found that different anti-motion sickness drugs influenced the slow phase velocity of nystagmus in response to caloric irrigation, but found that nystagmus frequency did not vary significantly between the different drug conditions. Further research into the relationships between visual acuity, slow phase velocity and the frequency of nystagmus may be interesting.

10.1.3 Visual acuity and motion sickness

A decrease in slow phase velocity, found when subjects with poor visual acuity view an optokinetic drum, leads to an increase in the rate of which images slip on the retina during the slow phase. The image slip velocity is the difference in the velocity of the stimulus (e.g. motion of the optokinetic drum) and the slow phase eye velocity. Motion sickness was found to reduce in a fixation condition (Chapter 5) where subjects focused on a stationary cross. In this condition foveal image slip was reduced to nothing but there was still peripheral image slip. The single and multiple dot experiment (Chapter 7) found that motion sickness was not significantly different between the two conditions. The foveal stimulus was the same in both conditions (i.e. a single moving dot), but there was additional peripheral stimulation in the full field of dots condition. The results from the above experiments suggest that foveal image slip, rather than peripheral image slip, may be responsible for motion sickness, via an unknown mechanism.

Eye movements themselves, as a possible cause for motion sickness (Ebenholtz *et al.,* 1994) cannot be ruled out completely. However, the results from the experimental work suggest that they are less likely to be an influence on motion sickness symptoms directly, because there were large variations in symptoms depending on the visual acuity of subjects, but relatively small variations in the eye movements recorded with varying visual acuity. Small variations in the slow phase velocity of nystagmus can, however, result in a large increase in foveal slip velocity. For example if, in response to drum velocity of 35°/second, eye velocity changes from 34°/second with 20:20 vision to 33°/second with 20:40 vision, then foveal image slip has increased from 1°/second to 2°/second. Foveal slip may be an error signal which, via an unknown mechanism, is associated with motion sickness in response to optokinetic stimuli.

The idea that foveal slip is an important error signal used in the control of eye movements can be found in previous studies. For example, Muratore *et al.* (1979) found that after-nystagmus was observed after exposure to a single point of light moving in a sawtooth fashion (similar to the single dot condition in Chapter 7). The after-nystagmus had similar characteristics to that observed when subjects had been exposed to a full optokinetic drum. Shelhamer *et al.* (1994) found that vestibulo-ocular reflex gain adaptation occurred to the same extent when a subject viewed a

single moving dot stimulus, as occurred when a full field optokinetic drum was viewed. Vestibulo-ocular reflex adaptation occurred even when there was no motion of the subject. During a fixation condition the vestibulo-ocular reflex adaptation was reduced. They concluded that vestibulo-ocular reflex adaptation is based mainly on image slip detected on the fovea, with a smaller contribution from peripheral image slip.

The use of foveal slip as an error signal may extend to motion sickness. Foveal slip could possibly be used as a quantifiable variable in 'sensory conflict' theory. Foveal slip usually occurs only when there is a mis-match between the vestibular and visual systems. An example is found when magnifying glasses are used (e.g. Demer *et al.*, 1989). Foveal slip occurs with magnifying glasses, which drives the vestibulo-ocular reflex to adapt its gain, in order to reduce foveal slip and to restore acuity. In this case, motion sickness and dizziness tend to occur up until the point at which the vestibulo-ocular reflex has adapted fully to the level of magnification of the glasses, at which point users typically report a reduction in symptoms (Melvill Jones *et al.*, 1975). In optokinetic drums, foveal slip occurs because the velocity of the eye rarely matches that of the drum. As the speed increases, the gain of eye movements recorded drops (Howard *et al.,* 1984), hence foveal slip velocity increases with increasing drum speed. As shown in Chapter 9, it also increased with decreased acuity and sensitivity to high spatial frequencies. Shelhamer *et al.* (1994) found that vestibulo-ocular reflex gain adaptation still occurred in response to motion of an optokinetic drum without any motion of the subject, indicating that foveal slip is occurring in optokinetic drums. The brain may be perceiving a need for calibration of the eye movement response because of the foveal slip experienced. The precise physiological mechanism by which foveal slip may lead to motion sickness is unknown and is beyond the scope of this thesis.

10.1.4 Review of literature and experimental results

10.1.4.1 Restricted field of view

The reduction in motion sickness with a restricted field of view, found by Stern *et al.* (1990) could be explained by stationary edges suppressing nystagmus. Murasugi *et al.* (1986) found that stationary edges, used to restrict the field of view of an optokinetic display, could suppress nystagmus. The stationary edges acted as a form

of fixation, which was shown in Chapter 5 and in the literature (Stern *et al.,* 1990) to reduce motion sickness, possibly because of the reduction in foveal image slip. Howard *et al.* (1984) found that blurring the edges used to restrict the visual field reduced their effect. This blurring may reduce the influence of the fovea on the control of eye movements (in a similar way to poor acuity), which may have reduced the ability of the fovea to fixate on the stationary edges.

10.1.4.2 Speed of rotation of the drum

Hu *et al.* (1989) found that motion sickness increased, with increasing speed or rotation of an optokinetic drum. They attributed the increased symptoms of sickness to increased experiences of vection as the speed increased. No data for correlations between individual vection and sickness scores were shown.

The gain of nystagmus has been shown to decrease with an increase in the speed of an optokinetic drum (e.g. Van Die *et al.* 1986, Cheng *et al.* 1975). As discussed above, reduced gain means that the velocity of foveal slip increases with increasing drum speed. The above hypothesis, that increasing foveal image slip is associated with increased motion sickness symptoms, may explain a possible reason why motion sickness increased with higher drum speeds. At high drum velocities subjects reported a severe blurring of the stripes, presumably because at these velocities the gain of the slow phase of nystagmus would be approximately 0.5-0.6 (Howard, 1984) resulting in foveal image slip of the order of 36-45°/second.

Increasing visual flow rates in a military flight simulator were shown to increase motion sickness (Sharkey *et al.*, 1991). This finding may also indicate that minimising the visual flow rate helps to minimise the velocity of image slip on the fovea, perhaps reducing motion sickness. It may also suggest that fixation could possibly reduce motion sickness in simulators.

10.1.4.3 Strobed lighting

Melvill-Jones *et al.* (1979) made a discovery that motion sickness symptoms were absent in a group of subjects who viewed a room for several hours with left-right reversing prism spectacles, in strobed light. All subjects viewing the same room in

normal light experienced some symptoms of motion sickness or dizziness. It is a possibility that the use of strobed light, which reduced foveal image slip because of the short duration of the light flashes (4μsec), reduced motion sickness because of this decrease in foveal image slip. The authors found that the gain of the vestibulo-ocular reflex did not change significantly at high frequencies, indicating that foveal slip, as an error signal, was reduced or absent during the strobed light condition (foveal slip was shown to be a dominant influence on the vestibulo-ocular reflex, e.g. Shelhamer *et al.*,1994). The possibility that strobed light reduced motion sickness because of a reduction in foveal image slip should be treated with caution because the visual stimuli in the condition (strobed and normal) were obviously quite different. The complete absence of motion sickness, even in previously susceptible subjects, may make this an interesting area for future research.

10.1.4.4 Frequency of nystagmus and motion sickness.

Hu *et al.* (1998) found that the frequency of nystagmus in response to an optokinetic drum, spinning at 60°/second, was significantly correlated with the symptoms of motion sickness experienced. They found that increasing frequency of nystagmus was associated with increased vection and motion sickness. The second experiment presented in this thesis (Chapter 5) did not find any similar correlations between either vection or motion sickness. The final experiment (Chapter 9) did not find any significant variation in nystagmus frequency with time or depending on the visual acuity of subjects.

As discussed in Section 2.5.6, it is not really possible to comment on the results from Hu *et al.* (1998) because it is not clear how the electro-oculography data was analysed, for example whether the periods in which eye movements did not occur (as mentioned by the authors) were taken into account in the frequency calculation, or how common these periods were among subjects. Periods where subjects were not focusing, and eye movements were suppressed, could be similar to fixation and may have reduced motion sickness symptoms. If average frequency was calculated by summing the total number of saccades and dividing by time, then subjects who had the greater number of periods where they were not focusing will also have been found to have the lowest frequencies.

The results from this thesis suggest that nystagmus frequency may depend on the speed of the drum and positioning of the stripes, rather than visual acuity or slow phase velocity.

10.1.4.5 Image magnification errors in virtual reality

Draper (1998) showed that motion sickness occurred in virtual reality systems when there were image magnification problems. These occurred when the image presented to a subject moved at a different velocity than the head velocity of the subject (similar to magnifying glasses). Image slip occurred in this situation, at a velocity which was the difference between the head and image velocities. Draper (1998) showed that vestibulo-ocular reflex gain adaptation occurred when image magnification errors occurred in virtual reality, in order to reduce the slipping of images on the retina. An influence of visual acuity may possibly be occurring during this kind of experiment. Further research into this area in which eye movements and visual acuity are measured may be necessary. Investigating the possibility of introducing fixation into virtual reality, in order to reduce foveal slip and motion sickness, may also be an interesting area for study.

10.1.5 Virtual reality as a tool for motion sickness study

The virtual reality system employed in the experimental work of this thesis proved to be an effective way to study visually-induced motion sickness. In the comparison of motion sickness and vection in Chapter 4, motion sickness scores across conditions were highly correlated, as were the vection scores across conditions. This suggests that the virtual reality simulation was able to cause motion sickness and vection by the same mechanisms with which they occurred in the traditional optokinetic drum. Further experiments revealed additional uses of the virtual reality display, for example the ability to add a fixation cross in front of the moving stripes in a matter of minutes, with no physical changes to the hardware. The single and multiple dot experiment (Chapter 7) was also simple to create on the virtual reality display, but would have been difficult to achieve by more traditional means (e.g. a film projector system).

10.2 Final model

The final model, presented in Figure 10.1, is based on the results from all six experimental chapters and previous studies. The final version of the model differs from the previous model (chapter 8) by the removal of the route from eye movements directly to motion sickness. This is for the reason discussed above (Section 10.1.3), because there were large variations in motion sickness symptoms depending on the visual acuity of subjects, but relatively small variations in the actual eye movements recorded with varying visual acuity (in Chapter 9). The direct route from eye movements to motion sickness has not been ruled out altogether, but for the purposes of this model the foveal slip input to motion sickness is favoured as the most likely.

Head movements have been reintroduced into the model (from Robinson, 1981) because although no head movements occurred in any of the experimental work conducted for this thesis, it may be useful to include head movements when using the model to generate hypotheses for future experimental work. In the model 'H' is the head velocity and 'G' is gaze velocity, the velocity of the eye with respect to space. Gaze velocity in an optokinetic drum might be used to calculate the velocity of image slip on the retina when head movements and eye movements are made simultaneously. Gaze velocity 'G' replaces eye velocity 'E' on the left hand side of the model where summation of the drum velocity 'W' and gaze velocity 'G' occurs to calculate foveal slip velocity 'e_f' and peripheral slip velocity 'e_p'.

Head velocity is converted to an eye movement signal via the semi-circular canals, which are modelled using only the cupula time constant (T_c), as in the original model of Robinson (1981). The output from the vestibular system is added to the signal from the peripheral optokinetic system, as the two are complementary systems under normal circumstances, and both the vestibulo-ocular reflex and optokinetic nystagmus can be cancelled by the pursuit reflex (Robinson, 1965., Robinson, 1981). This is modelled by the switch in the model (Figure 10.1).

Artificial blur and visual acuity are shown to act on the foveal pursuit component of the slow phase velocity. Increasing visual blur or decreasing visual acuity are modelled to decrease the influence of the fovea, which reduces the velocity of the eye movement, and hence increases foveal slip via the feedback of 'gaze velocity' to the summation point on the left hand side (where 'gaze velocity' and 'world velocity'

are compared). Vection is shown to be dependent on peripheral image motion and to be independent of motion sickness.

Fixation, which can be introduced deliberately or accidentally by stationary edges near the fovea, can be seen to reduce motion sickness by reducing the foveal image slip velocity occurring.

10.2.1 Explanation of the complete model in detail

In order to provide a complete explanation of the model, this section looks at the model, with reference to the experimental findings and previously published studies.

10.2.1.1 Comparison of motion sickness and vection in a real and virtual reality optokinetic drum

The first experiment conducted found that motion sickness was slightly lower in the virtual reality condition, compared to the real optokinetic drum condition with the same field of view. Vection was not significantly different. The model above predicts that motion sickness would be the same in each of the conditions because they were expected to have equal foveal stimulation. The slight problem with the virtual reality display in the first condition, where some stationary pixels were visible in the background of the display (which were removed in subsequent experiments), may explain the slight decrease in the motion sickness symptoms in the virtual reality condition. The stationary pixels may have acted as fixation points, by which subjects could stop eye movements. The model shows that the fixation route reduces motion sickness by reducing foveal image slip. The amount of fixation occurring on stationary pixels may have not been high, i.e. it was probably intermittent, because the motion sickness symptoms were highly correlated between the two conditions. When a full fixation condition was used in Chapter 5, it was found that the motion sickness scores in the two conditions were not significantly correlated.

Vection is also be predicted to be the same in each condition from the model, which was found to be the case in this experiment

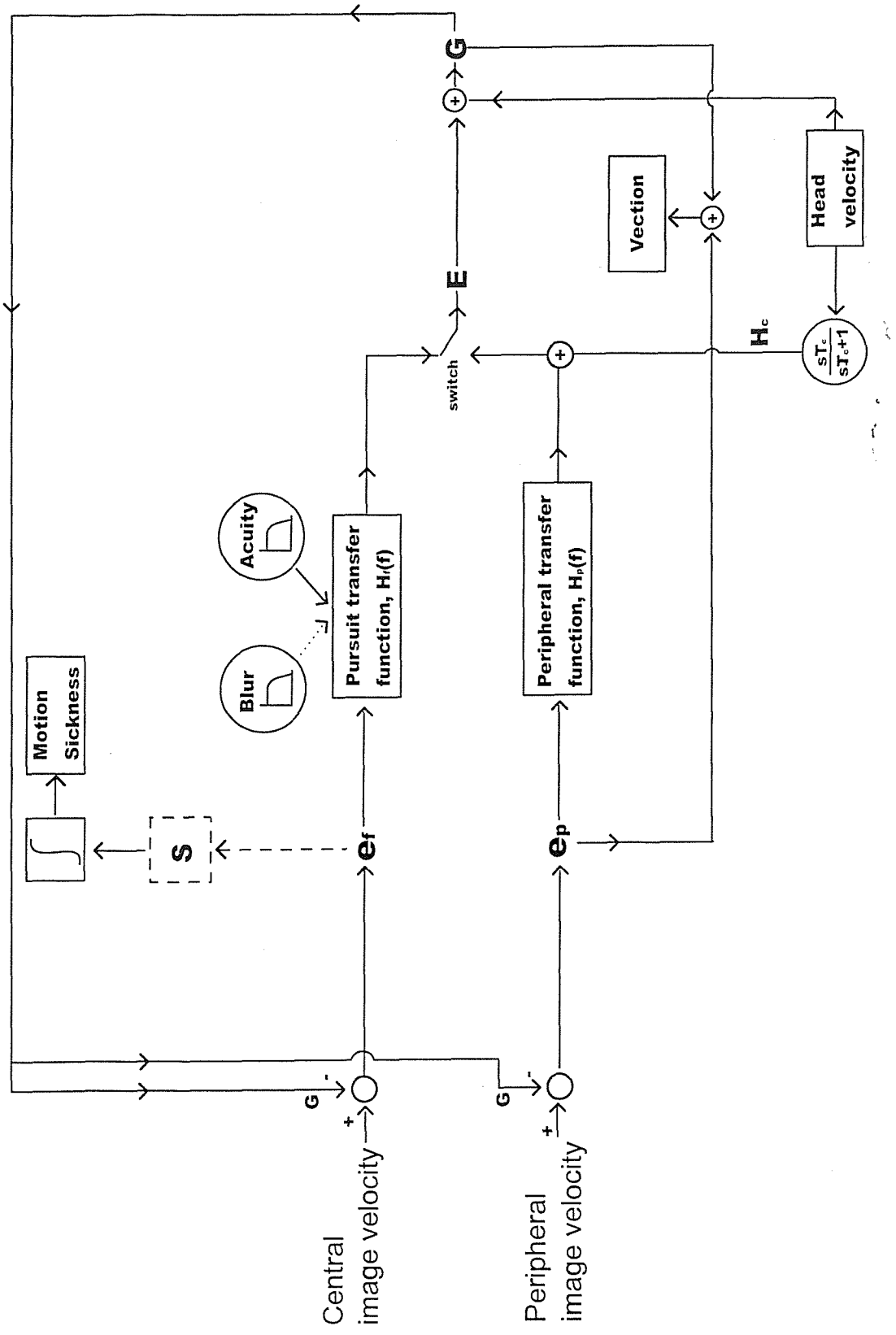

Figure 10.1. The final model, version 5. Head movements have been re-introduced from the original model of Robinson, 1981.

10.2.1.2 Experiment 2. Motion sickness and vection with and without fixation

The model predicts that motion sickness will be reduced by fixation, but that vection will be the same because of the peripheral domination of vection and the independence of vection and eye movements. The results showed that motion sickness was significantly reduced and that vection was not significantly different in the two conditions. The model also predicts that visual acuity will only influence motion sickness in the normal condition where foveal slip can occur. The results showed that motion sickness was only influenced by visual acuity in the normal condition and was not a significant influence in the fixation condition, where no foveal slip occurred.

10.2.1.3 Experiment 3. Motion sickness with and without artificial blurring

The model predicts that motion sickness will be increased by the introduction of artificial blurring, because the removal of the high spatial frequency content of the visual stimulus will have a similar effect on eye movements as poor acuity. The removal of the high spatial frequencies may reduce the influence of the fovea on the slow phase of nystagmus, which will act to reduce the gain, hence increasing foveal slip. The experiment only found a small increase in symptoms, as measured on the post exposure symptom questionnaire. Further experiments to investigate artificial blur may be necessary. Vection is predicted from the model to be similar with or without artificial blur. The experimental results found that vection was not significantly different between the two conditions.

10.2.1.4 Experiment 4. Comparison of vection and motion sickness with a single or multiple dot display

The model predicts that motion sickness will not be significantly different with a single moving dot or multiple dots, because motion sickness is proposed to be influenced by foveal image slip, which was identical in both conditions. The experimental results found that motion sickness was not significantly different. Vection was predicted to be significantly higher in the full field condition because there was significantly more peripheral stimulation. The results showed that this was the case. The model predicts

that visual acuity will be correlated with motion sickness in both of the conditions. There were marginal correlations found, but there was only a small variation of visual acuity among subjects.

10.2.1.5 Experiment 5. Comparison of motion sickness with and without corrected vision

The model predicts that motion sickness will be higher when subjects do not use visual correction (e.g. spectacles or contact lenses) compared to when they do, because they have reduced visual acuity without vision correction. The model predicts that vection will not differ. The results showed that motion sickness was higher without vision correction and that vection was not significantly different. The contrast sensitivity information showed that sensitivity to high spatial frequencies was associated with motion sickness, rather than at a range of low and high spatial frequencies.

10.2.1.6 Experiment 6. Comparison of the slow phase velocity of nystagmus with and without vision correction

The final experiment confirmed the model prediction that the slow phase velocity during exposure to optokinetic stimulation was dependent on visual acuity or contrast sensitivity to high spatial frequencies. Visual acuity is shown to act on the 'foveal pursuit transfer function'. Reducing foveal slip, via fixation or other means, such as removing a central band in an optokinetic drum or by increasing visual acuity, is modelled to reduce motion sickness. This could be investigated in further experimental studies.

Chapter 11. Conclusions and recommendations

11.1 Conclusions

This thesis has shown that motion sickness and vection are experienced during visual stimulation from an optokinetic drum and during virtual reality simulations of an optokinetic drum. It was shown that vection and motion sickness are probably separate phenomena. Subject motion sickness scores were not correlated with vection in any of the experiments conducted. Motion sickness could be reduced without significantly changing the amount of vection reported by subjects. Vection could also be reduced without significantly changing the motion sickness symptoms experienced. Vection was also found to vary independently of the frequency of nystagmus or of the slow phase velocity of nystagmus. Vection was found to be controlled mainly by motion in the periphery of vision.

The velocity of the slow phase of nystagmus was found to vary depending on the visual acuity, and the contrast sensitivity to higher spatial frequencies, of subjects. Those subjects who had poorer visual acuity, or lower sensitivity to high spatial frequencies, were found to have slower velocity eye movements, compared to subjects who had good visual acuity. Retinal slip velocity (the difference between the stimulus velocity and slow phase velocity) was higher for subjects with poor visual acuity, or low contrast sensitivity to high spatial frequencies, because these subjects had lower slow phase velocity of nystagmus.

Image slip on the fovea, rather than on the retina as a whole, was found to be a possible error signal in the development of motion sickness. Motion sickness was reduced when subjects focused on a stationary cross in front of an optokinetic simulation (reducing foveal image slip but not peripheral image slip). Motion sickness was not changed when subjects viewed a single dot (only foveal stimulation) or multiple dots (foveal and peripheral stimulation).

Visual acuity was found to be significantly correlated with motion sickness in a standard optokinetic drum and in the virtual reality simulation of an optokinetic drum. Subjects with poorer visual acuity experienced significantly more symptoms of motion sickness. Motion sickness was increased when subjects viewed an optokinetic drum

without their vision correction (spectacles or contact lenses) compared to when they viewed it with vision correction.

As mentioned above, foveal image slip was found to increase with poorer visual acuity, hence the hypothesis was developed that motion sickness during optokinetic simulation may be related to foveal slip, with an increase in motion sickness with increased foveal slip (associated with lower visual acuity and lower contrast sensitivity to high spatial frequencies).

Contrast sensitivity as a measurement of visual performance was found to have a much greater variation among subjects with vision correction than the variation of visual acuity among the same subjects. With vision correction, correlations were found between the slow phase velocity and contrast sensitivity at the high spatial frequencies and one of the low spatial frequencies. It may be possible to use contrast sensitivity as a more sensitive indicator of visual function, in order to predict the velocity of eye movements, even in subjects who have good visual acuity.

11.2 Recommendations for future work

The work in this thesis has concentrated on the fundamental understanding of interactions between visual acuity, contrast sensitivity, eye movements, motion sickness and vection. Further research could be conducted upon these lines, in order to further improve the model (Figure 10.1). Work could also investigate means by which motion sickness could be reduced in practical situations, by employing ideas generated by the model.

11.2.1 Fundamental work

Further study of the model could be performed in order to verify the foveal slip idea. An experiment in which the fovea was blocked at all times by a moving blinker system (similar to that used by Van Die *et al.,* 1986) would be expected to reduce motion sickness, compared to a condition without the fovea blocked, because of the reduction in foveal slip. This experiment would help to confirm whether eye movements themselves are a cause of motion sickness because eye movements would still occur, despite the elimination of the foveal input.

Further research to improve the model could look at understanding the interaction of eye movements and the vestibular system. Subjects were not moved in any of the experiments in this thesis, but in normal circumstances eye movements occur as a result of motion of the person and motion of the environment. The vestibulo-ocular reflex serves to stabilise vision during head movements. Optokinetic nystagmus occurs as a complementary system to stabilise vision under conditions of low frequency head movements and constant velocity rotation.

The model could be improved by a greater understanding of the interactions of the two systems. It may be possible to predict eye movements that might occur under combinations of motion of the subject and the environment. It may be able to take visual acuity and the excitation of the fovea and peripheral vision into account. An understanding of the interactions of the vestibulo-ocular reflex, optokinetic nystagmus and other eye movements may enable eye movements to be included in a wider 'sensory conflict' theory, which could be applied to motion sickness occurring in virtual reality, simulators and possibly even in ships, trains, cars and other modes of transport.

11.2.2 Practical applications

Applications to reduce motion sickness in virtual reality environments could be developed by the model. For example, in a perfect virtual reality simulation each head movement would be accompanied by the exact counter movement of the visual world, without any error or delay. This would not be expected to cause motion sickness because it would be indistinguishable from the real world condition.

Virtual reality departs from reality because of errors made either during the real time measurement of head movements or due to inefficient processing and delays in the generation of the computer imagery. Errors will still exist even in an optimally calibrated system because of the inherent delays in updating computer graphics in response to head movements and imperfect head velocity measurement techniques.

One of the results of the above errors is to create a slipping of the images on the retina. For example the user may make a head movement of 10°/s angular velocity. The virtual reality system must move the visual scene by 10°/s in the opposite direction to head motion in order to maintain a space stabilised image (i.e. the world

must appear stationary whilst the subject navigates through it). If the head tracking system incorrectly measures the head movement as 8°/s and only moves the visual scene by this velocity, there will be a slipping of the image of 2°/s on the retina. The world will appear to move independently of the subject. The same phenomenon can be created if the computer is slow to update the images so that the world is still moving after the head has stopped moving.

The above problems can be thought of in terms of 'image magnification errors' (see Section 2.6.2 for more details). This is similar to a subject wearing magnifying spectacles. The world in this case moves at a different velocity to the head, say 1.2 times as fast. There will be periods during exposure to virtual reality where the image is slipping during and after head movements, representing an optokinetic stimulus.

The effect of visual acuity and contrast sensitivity to high spatial frequencies on motion sickness survival time would be expected to be observed in virtual reality applications. Experiments which look at this possibility could be devised. Additional experiments which use fixation (as discussed in Chapter 5) could help to verify whether this can reduce motion sickness with this type of image motion, in the same way that it reduced motion sickness in the optokinetic drum.

Practical applications of the fixation idea could be generated in a head-coupled virtual reality simulation. The fixation system could be implemented with a fine mesh which could be updated more quickly than the normal content in the virtual reality system. This mesh could appear only during head movements where there is likely to be a slipping of the virtual reality content on the retina. The mesh should be placed so that foveal fixation can occur on the mesh during head movements. The background will still be visible so that performance is not impaired. The hypothesis is that fixation in virtual reality, created by means of a visible mesh, could reduce motion sickness by reducing foveal image slip.

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Appendix A. Sample power calculations

In each of the experiments conducted in this thesis, sample power calculations were conducted in order to find an appropriate sample size for the particular experiment.

Sample power tests involved the use of paired-samples t test (parametric statistics). The sample power and sample sizes generated will only be an approximation, because non-parametric tests were used in the actual analyses.

Values for standard deviations were chosen on the basis of the previous literature and studies conducted in the Human Factors Research Unit at the University of Southampton. A standard deviation of 15 was used for the accumulated motion sickness scores in experiments 1, 2, 3 and 5. Experiment 4 used a standard deviation of 7, while experiment 6 used a standard deviation of 7°/second for slow phase eye velocity.

A high correlation coefficient was used of 0.75 for all the sample power calculations because the same subjects were used in each group, in Wilcoxon matched-pairs signed-ranks tests.

Experiment 1

The sample size of 16 subjects was sufficient to show a significant difference ($p<0.05$) between the mean accumulated illness rating values on a paired-samples t test amounting to 7.9 with a power of 0.8.

Experiment 2

The sample size of 18 subjects was sufficient to show a significant difference ($p<0.05$) between the mean accumulated illness rating values on a paired-samples t test amounting to 7.4 with a power of 0.8.

Appendix B. Motion sickness susceptibility questionnaire.

INSTRUCTIONS

This questionnaire is primarily concerned with: (i) your susceptibility to motion sickness and, (ii) what types of motion are most effective in causing this sickness.

Please read the questions carefully and answer them **ALL** by either TICKING or FILLING IN the boxes which most closely correspond to you as an individual.

All the information you give is CONFIDENTIAL and will be used for research purposes only.

Thank you very much for your co-operation.

NAME _____ AGE _____ SEAT NUMBER _____

APPROXIMATE BODY WEIGHT _____ HEIGHT _____

1. In the past **YEAR**, how many times have you travelled **AS A PASSENGER** in the following types of transport?

	NEVER	1	2-3	4-15	16-63	64-255	256+
CARS							
BUSES							
COACHES							
SMALL BOATS							
SHIPS							
AEROPLANES							
TRAINS							

2. In the past **YEAR**, how many times have you felt ill, whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	1	2	3	4-7	8-15	16+
CARS							
BUSES							
COACHES							
SMALL BOATS							
SHIPS							
AEROPLANES							
TRAINS							

3. In the past **YEAR**, how many times have you VOMITED whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	1	2	3	4-7	8-15	16+
CARS							
BUSES							
COACHES							
SMALL BOATS							
SHIPS							
AEROPLANES							
TRAINS							

4. Do you **EVER** feel HOT or SWEAT whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

5. Do you **EVER** suffer from HEADACHES whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

6. Do you **EVER** suffer from LOSS/CHANGE OF SKIN COLOUR (go pale) whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

7. Do you **EVER** suffer from MOUTH WATERING whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

8. Do you **EVER** feel DROWSY whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

9. Do you **EVER** feel DIZZY whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

10. Do you **EVER** suffer from NAUSEA (stomach discomfort, feeling sick) whilst travelling **AS A PASSENGER** in the following types of transport?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

11. Have you **EVER** VOMITED whilst travelling **AS A PASSENGER** in the following types of transport?

	YES	NO	DON'T KNOW
CARS			
BUSES			
COACHES			
SMALL BOATS			
SHIPS			
AEROPLANES			
TRAINS			

12. Would you avoid any of the following types of transport because of motion sickness?

	NEVER	OCCASIONALLY	OFTEN	ALWAYS
CARS				
BUSES				
COACHES				
SMALL BOATS				
SHIPS				
AEROPLANES				
TRAINS				

13. Which of the following best describes your SUSCEPTIBILITY to motion sickness?

MUCH LESS THAN AVERAGE	☐
LESS THAN AVERAGE	☐
AVERAGE	☐
MORE THAN AVERAGE	☐
MUCH MORE THAN AVERAGE	☐

14. Have you ever suffered from any serious illness or injury?

YES ☐ NO ☐

15. Are you under medical treatment or suffering a disability affecting daily life?

YES ☐ NO ☐

Appendix C

Subject Instructions for experiments 1 to 6

Experiment 1 (real and virtual reality drum comparison)

Drum condition

The purpose of this experiment is to measure the illusion of motion and motion sickness, experienced during exposure to moving black and white stripes (optokinetic drum). You will be seated in the chair under the drum. A strap will be placed around your head and attached to the chair in order to keep your head still and you will be asked to wear special glasses to reduce your visual field. The drum will be lowered over you and rotated around you.

Please look straight ahead at the stripes at all times.

Do not deliberately follow the stripes. You should allow your eyes to naturally follow them.

You may stop the drum at any time, for any reason, by pressing your emergency stop button, or by requesting that it should be stopped.

Each minute you will be asked **'how do you feel?'** to which you should reply from the following Table:

Subjective Response	Corresponding Feeling
0	No symptoms
1	Any symptom, however slight
2	Mild symptoms e.g. stomach awareness, but no nausea
3	Mild nausea
4	Mild to moderate nausea
5	Moderate nausea, but can continue
6	Moderate nausea, want to stop

Each minute you will also be asked **'what is moving?'** to which you will reply from the following table to indicate your perception of motion:

Perception of what is moving	You report:
Drum Only	You perceive that the only thing moving is the drum (real or virtual).
Drum and Self (intermittent)	You perceive the drum to be moving but also experience periods of self motion.
Drum and Self (continuous)	You perceive the drum to be moving and simultaneously experience continuous self motion.
Self Only	You perceive the drum to be stationary and experience continuous self motion only.

The experiment will end after 30 minutes, when you reach 6 on the motion sickness scale, or if you wish to stop for any other reason.

Virtual reality condition

The purpose of this experiment is to measure the illusion of motion and motion sickness, experienced during exposure to moving black and white stripes (shown on a virtual reality display). You will be seated in the chair under the drum. A strap will be placed around your head and attached to the chair in order to keep your head still. The drum will be lowered over you and rotated around you whilst you watch the stripes moving on the virtual reality display.

Please look straight ahead at the stripes at all times.

Do not deliberately follow the stripes. You should allow your eyes to naturally follow them.

You may stop the drum at any time, for any reason, by pressing your emergency stop button, or by requesting that it should be stopped.

Each minute you will be asked **'how do you feel?'** to which you should reply from the following Table:

Subjective Response	Corresponding Feeling
0	No symptoms
1	Any symptom, however slight
2	Mild symptoms e.g. stomach awareness, but no nausea
3	Mild nausea
4	Mild to moderate nausea
5	Moderate nausea, but can continue
6	Moderate nausea, want to stop

Each minute you will also be asked **'what is moving?'** to which you will reply from the following table to indicate your perception of motion:

Perception of what is moving	You report:
Drum Only	You perceive that the only thing moving is the drum (real or virtual).
Drum and Self (intermittent)	You perceive the drum to be moving but also experience periods of self motion.
Drum and Self (continuous)	You perceive the drum to be moving and simultaneously experience continuous self motion.
Self Only	You perceive the drum to be stationary and experience continuous self motion only.

The experiment will end after 30 minutes, when you reach 6 on the motion sickness scale, or if you wish to stop for any other reason.

Experiment 2 (optokinetic drum with and without fixation)

Normal condition

The purpose of this experiment is to measure the illusion of motion and motion sickness, experienced during exposure to moving black and white stripes (shown on a virtual reality display). You will be seated in the chair under the drum. The drum will stay in its raised position. A strap will be placed around your head and attached to the chair in order to keep your head still. During exposure you will listen to 'white noise' on the headphones of the virtual reality display, and I will talk to you each minute through a microphone. Your eye movements will be measured using electro-oculography – which will be explained to you by the experimenter.

Please look straight ahead at the stripes at all times.

Do not deliberately follow the stripes. You should allow your eyes to naturally follow them.

You may stop the experiment at any time, for any reason, by removing the virtual reality display from your head, or by requesting that it should be stopped.

Each minute you will be asked **'how do you feel?'** to which you should reply from the following Table:

Subjective Response	Corresponding Feeling
0	No symptoms
1	Any symptom, however slight
2	Mild symptoms e.g. stomach awareness, but no nausea
3	Mild nausea
4	Mild to moderate nausea
5	Moderate nausea, but can continue
6	Moderate nausea, want to stop

Each minute you will also be asked **'what is moving?'** to which you will reply from the following table to indicate your perception of motion:

Perception of what is moving	You report:
Drum Only	You perceive that the only thing moving is the drum (real or virtual).
Drum and Self (intermittent)	You perceive the drum to be moving but also experience periods of self motion.
Drum and Self (continuous)	You perceive the drum to be moving and simultaneously experience continuous self motion.
Self Only	You perceive the drum to be stationary and experience continuous self motion only.

The experiment will end after 30 minutes, when you reach 6 on the motion sickness scale, or if you wish to stop for any other reason.

Fixation condition

The purpose of this experiment is to measure the illusion of motion and motion sickness, experienced during exposure to moving black and white stripes (shown on a virtual reality display). You will be seated in the chair under the drum. The drum will stay in its raised position. A strap will be placed around your head and attached to the chair in order to keep your head still. During exposure you will listen to 'white noise' on the headphones of the virtual reality display, and I will talk to you each minute through a microphone. Your eye movements will be measured using electro-oculography – which will be explained to you by the experimenter.

Please look straight ahead at the stationary cross at all times. Do not follow the stripes on the screen.

You may stop the experiment at any time, for any reason, by removing the virtual reality display from your head, or by requesting that it should be stopped.

Each minute you will be asked **'how do you feel?'** to which you should reply from the following Table:

Subjective Response	Corresponding Feeling
0	No symptoms
1	Any symptom, however slight
2	Mild symptoms e.g. stomach awareness, but no nausea
3	Mild nausea
4	Mild to moderate nausea
5	Moderate nausea, but can continue
6	Moderate nausea, want to stop

Each minute you will also be asked **'what is moving?'** to which you will reply from the following table to indicate your perception of motion:

Perception of what is moving	You report:
Drum Only	You perceive that the only thing moving is the drum (real or virtual).
Drum and Self (intermittent)	You perceive the drum to be moving but also experience periods of self motion.
Drum and Self (continuous)	You perceive the drum to be moving and simultaneously experience continuous self motion.
Self Only	You perceive the drum to be stationary and experience continuous self motion only.

The experiment will end after 30 minutes, when you reach 6 on the motion sickness scale, or if you wish to stop for any other reason.

Experiment 3 (normal and blurred stripes)

The instructions for experiment 3 are the same as those for experiment 2 - normal condition.

Experiment 4 (single and multiple dots)

Single dot condition

The purpose of this experiment is to measure the illusion of motion and motion sickness, experienced during exposure to a moving dot (shown on a virtual reality display). You will be seated in the chair under the drum. The drum will stay in its raised position. A strap will be placed around your head and attached to the chair in order to keep your head still. During exposure you will listen to 'white noise' on the headphones of the virtual reality display, and I will talk to you each minute through a microphone. Your eye movements will be measured using electro-oculography – which will be explained to you by the experimenter.

Please follow the single dot at all times.

You may stop the experiment at any time, for any reason, by removing the virtual reality display from your head, or by requesting that it should be stopped.

Each minute you will be asked **'how do you feel?'** to which you should reply from the following Table:

Subjective Response	Corresponding Feeling
0	No symptoms
1	Any symptom, however slight
2	Mild symptoms e.g. stomach awareness, but no nausea
3	Mild nausea
4	Mild to moderate nausea
5	Moderate nausea, but can continue
6	Moderate nausea, want to stop

You will also be asked **'how much are you moving?'** to which you should reply, with any number between 0 and 100% from the table below to indicate your perception of motion:

Perception of motion	You report:
You feel like you are stationary and it is the dot which appears to be moving only.	0%
You feel like you are moving a bit, but the dot is moving more	1-49%
You feel like you are moving at the same speed as the dot	50%
You feel like you are moving a lot and the dot is moving a bit	51-99%
You feel like you are moving and the dot appears stationary	100%

The experiment will end after 30 minutes, when you reach 6 on the motion sickness scale, or if you wish to stop for any other reason.

Multiple dot condition

The purpose of this experiment is to measure the illusion of motion and motion sickness, experienced during exposure to a moving dot (shown on a virtual reality display). You will be seated in the chair under the drum. The drum will stay in its raised position. A strap will be placed around your head and attached to the chair in order to keep your head still. During exposure you will listen to 'white noise' on the headphones of the virtual reality display, and I will talk to you each minute through a microphone. Your eye movements will be measured using electro-oculography – which will be explained to you by the experimenter.

Please track each dot as it passes, in the central row of dots. Do not skip any of the dots (demonstrated to the subject on the computer monitor).

You may stop the experiment at any time, for any reason, by removing the virtual reality display from your head, or by requesting that it should be stopped.

Each minute you will be asked **'how do you feel?'** to which you should reply from the following Table:

Subjective Response	Corresponding Feeling
0	No symptoms
1	Any symptom, however slight
2	Mild symptoms e.g. stomach awareness, but no nausea
3	Mild nausea
4	Mild to moderate nausea
5	Moderate nausea, but can continue
6	Moderate nausea, want to stop

You will also be asked **'how much are you moving?'** to which you should reply, with any number between 0 and 100% from the table below to indicate your perception of motion:

Perception of motion	You report:
You feel like you are stationary and it is the dot which appears to be moving only.	0%
You feel like you are moving a bit, but the dot is moving more	1-49%
You feel like you are moving at the same speed as the dot	50%
You feel like you are moving a lot and the dot is moving a bit	51-99%
You feel like you are moving and the dot appears stationary	100%

The experiment will end after 30 minutes, when you reach 6 on the motion sickness scale, or if you wish to stop for any other reason.

Experiment 5 (optokinetic drum with and without vision correction)

Both conditions

The purpose of this experiment is to measure the illusion of motion and motion sickness, experienced during exposure to moving black and white stripes (optokinetic drum). You will be seated in the chair under the drum. A strap will be placed around your head and attached to the chair in order to keep your head still. The drum will be lowered over you and rotated around you.

Please look straight ahead at the stripes at all times.

Do not deliberately follow the stripes. You should allow your eyes to naturally follow them.

You may stop the drum at any time, for any reason, by pressing your emergency stop button, or by requesting that it should be stopped.

Each minute you will be asked **'how do you feel?'** to which you should reply from the following Table:

Subjective Response	Corresponding Feeling
0	No symptoms
1	Any symptom, however slight
2	Mild symptoms e.g. stomach awareness, but no nausea
3	Mild nausea
4	Mild to moderate nausea
5	Moderate nausea, but can continue
6	Moderate nausea, want to stop

Each minute you will also be asked **'how much are you moving?'** to which you will reply from the following table to indicate your perception of motion:

Perception of motion (vection)	**You report:**
You feel like you are stationary and it is the dot(s) which appear to be moving only.	0%
You feel like you are moving a bit, but the dot(s) are moving more	1-49%
You feel like you are moving at the same speed as the dot(s)	50%
You feel like you are moving a lot and the dot(s) are moving a bit	51-99%
You feel like you are moving and the dot(s) appear stationary	100%

The experiment will end after 30 minutes, when you reach 6 on the motion sickness scale, or if you wish to stop for any other reason.

Experiment 6 (measurement of eye movements)

Instructions as per experiment 5. Subjects were verbally instructed about the IRIS eye measurement system.

Appendix D. Subject Data

Experiment 1. Normal condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	4.50	1.00	.00	80.00
2.00	15.00	1.00	.00	80.00
3.00	25.00	1.00	1.00	100.00
4.00	19.50	1.00	1.00	133.00
5.00	10.50	1.00	.00	50.00
6.00	8.00	1.00	.00	80.00
7.00	30.00	1.00	.00	80.00
8.00	22.50	1.00	1.00	100.00
9.00	24.00	1.00	1.00	100.00
10.00	8.00	1.00	.00	66.00
11.00	2.50	1.00	.00	50.00
12.00	17.50	1.00	.00	80.00
13.00	17.00	1.00	.00	50.00
14.00	22.50	1.00	1.00	100.00
15.00	17.00	1.00	1.00	100.00
16.00	30.00	.00	1.00	133.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	1.00	133.00	12.00
2.00	1.00	100.00	4.00
3.00	.00	50.00	4.00
4.00	1.00	100.00	2.00
5.00	.00	20.00	17.00
6.00	1.00	133.00	26.00
7.00	.00	80.00	16.00
8.00	.00	66.00	-2.00
9.00	1.00	100.00	14.00
10.00	1.00	100.00	16.00
11.00	.00	80.00	2.00
12.00	1.00	100.00	16.00
13.00	.00	20.00	8.00
14.00	1.00	100.00	3.00
15.00	1.00	100.00	56.00
16.00	.00	80.00	21.00

Experiment 1 – Virtual reality condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	8.50	1.00	.00	80.00
2.00	30.00	.00	.00	80.00
3.00	30.00	.00	1.00	100.00
4.00	30.00	.00	1.00	133.00
5.00	14.50	1.00	.00	50.00
6.00	28.00	1.00	.00	80.00
7.00	7.00	1.00	.00	80.00
8.00	30.00	.00	1.00	100.00
9.00	17.00	1.00	1.00	100.00
10.00	4.00	1.00	.00	66.00
11.00	7.00	1.00	.00	50.00
12.00	18.00	1.00	.00	80.00
13.00	16.50	1.00	.00	50.00
14.00	30.00	.00	1.00	100.00
15.00	3.50	1.00	1.00	100.00
16.00	30.00	.00	1.00	133.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	1.00	133.00	12.00
2.00	1.00	100.00	4.00
3.00	.00	50.00	4.00
4.00	1.00	100.00	2.00
5.00	.00	20.00	17.00
6.00	1.00	133.00	26.00
7.00	.00	80.00	16.00
8.00	.00	66.00	-2.00
9.00	1.00	100.00	14.00
10.00	1.00	100.00	16.00
11.00	.00	80.00	2.00
12.00	1.00	100.00	16.00
13.00	.00	20.00	8.00
14.00	1.00	100.00	3.00
15.00	1.00	100.00	56.00
16.00	.00	80.00	21.00

Subject number	Accumulated illness rating (Virtual reality)	Accumulated illness rating (real drum)	Accumulated vection score (virtual drum)	Accumulated vection score (real drum)
1	59.50	112.00	13.00	24.00
2	13.50	40.50	17.50	16.00
3	7.00	21.00	.00	2.00
4	6.50	21.00	2.50	37.00
5	42.50	46.50	9.00	5.00
6	28.00	55.00	30.50	17.50
7	47.00	8.00	.00	.50
8	27.50	35.50	57.00	37.00
9	35.50	36.00	32.50	43.00
10	71.50	119.50	7.50	31.00
11	67.50	145.00	43.50	56.00
12	40.00	38.50	25.50	45.00
13	42.00	36.50	19.00	16.00
14	17.00	3.00	57.00	45.00
15	118.00	153.50	33.00	37.00
16	.00	.00	.00	.00

Experiment 2. Normal condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	5.00	1.00	.00	33.00
2.00	30.00	.00	.00	66.00
3.00	7.00	1.00	.00	50.00
4.00	30.00	.00	1.00	100.00
5.00	16.00	1.00	.00	50.00
6.00	30.00	.00	1.00	100.00
7.00	2.00	1.00	.00	50.00
8.00	4.00	1.00	.00	10.00
9.00	22.00	1.00	.00	10.00
10.00	14.00	1.00	1.00	100.00
11.00	30.00	.00	1.00	133.00
12.00	5.00	1.00	.00	66.00
13.00	28.00	1.00	.00	10.00
14.00	9.00	1.00	.00	29.00
15.00	30.00	.00	1.00	100.00
16.00	14.00	1.00	1.00	133.00
17.00	10.00	1.00	.00	66.00
18.00	23.00	1.00	.00	33.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	.00	40.00	5.00
2.00	1.00	133.00	10.00
3.00	1.00	117.00	7.00
4.00	1.00	100.00	-2.00
5.00	1.00	117.00	7.00
6.00	1.00	100.00	23.00
7.00	1.00	100.00	-1.00
8.00	.00	33.00	7.00
9.00	.00	40.00	-1.00
10.00	1.00	117.00	50.00
11.00	1.00	133.00	15.00
12.00	1.00	133.00	54.00
13.00	.00	10.00	1.00
14.00	1.00	100.00	7.00
15.00	1.00	100.00	-2.00
16.00	1.00	133.00	1.00
17.00	1.00	100.00	12.00
18.00	1.00	133.00	15.00

Experiment 2. Fixation condition

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	12.00	1.00	.00	33.00
2.00	30.00	.00	.00	66.00
3.00	17.00	1.00	.00	50.00
4.00	30.00	.00	1.00	100.00
5.00	16.00	1.00	.00	50.00
6.00	30.00	.00	1.00	100.00
7.00	30.00	.00	.00	50.00
8.00	20.00	1.00	.00	10.00
9.00	17.00	1.00	.00	10.00
10.00	11.00	1.00	1.00	100.00
11.00	30.00	.00	1.00	133.00
12.00	4.00	1.00	.00	66.00
13.00	30.00	.00	.00	10.00
14.00	12.00	1.00	.00	29.00
15.00	30.00	.00	1.00	100.00
16.00	30.00	.00	1.00	133.00
17.00	27.00	1.00	.00	66.00
18.00	7.00	1.00	.00	33.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	.00	40.00	5.00
2.00	1.00	133.00	10.00
3.00	1.00	117.00	7.00
4.00	1.00	100.00	-2.00
5.00	1.00	117.00	7.00
6.00	1.00	100.00	23.00
7.00	1.00	100.00	-1.00
8.00	.00	33.00	7.00
9.00	.00	40.00	-1.00
10.00	1.00	117.00	50.00
11.00	1.00	133.00	15.00
12.00	1.00	133.00	54.00
13.00	.00	10.00	1.00
14.00	1.00	100.00	7.00
15.00	1.00	100.00	-2.00
16.00	1.00	133.00	1.00
17.00	1.00	100.00	12.00
18.00	1.00	133.00	15.00

Subject number	Accumulated illness rating (normal condition)	Accumulated illness rating (fixation condition)	Accumulated vection score (normal condition)	Accumulated vection score (fixation condition)
1.00	136.00	42.00	65.00	61.00
2.00	5.00	10.00	24.00	31.00
3.00	32.00	21.00	37.00	21.00
4.00	27.00	25.00	38.00	44.00
5.00	33.00	4.00	56.00	52.00
6.00	.00	.00	3.00	54.00
7.00	34.00	.00	70.00	60.00
8.00	87.00	39.00	4.00	6.00
9.00	33.00	39.00	60.00	81.00
10.00	28.00	43.00	20.00	36.00
11.00	24.00	.00	20.00	40.00
12.00	33.00	36.00	58.00	43.00
13.00	22.00	16.00	11.00	9.00
14.00	43.00	43.00	60.00	29.00
15.00	8.00	.00	5.00	.00
16.00	43.00	13.00	13.00	32.00
17.00	108.00	16.00	.00	9.00
18.00	37.00	3.00	3.00	.00

Experiment 3. Normal condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	30.00	.00	1.00	133.00
2.00	9.00	1.00	1.00	133.00
3.00	30.00	.00	.00	100.00
4.00	11.00	1.00	1.00	133.00
5.00	30.00	.00	1.00	133.00
6.00	30.00	.00	.00	100.00
7.00	30.00	.00	.00	100.00
8.00	16.00	1.00	1.00	133.00
9.00	30.00	.00	1.00	133.00
10.00	10.00	1.00	1.00	133.00
11.00	30.00	.00	1.00	133.00
12.00	30.00	.00	1.00	133.00
13.00	30.00	.00	1.00	133.00
14.00	30.00	.00	.00	100.00
15.00	13.00	1.00	.00	117.00
16.00	30.00	.00	1.00	133.00
17.00	4.00	1.00	1.00	133.00
18.00	30.00	.00	1.00	133.00
19.00	9.00	1.00	1.00	133.00
20.00	18.00	1.00	1.00	133.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	1.00	133.00	2.00
2.00	1.00	133.00	12.00
3.00	1.00	133.00	7.00
4.00	1.00	133.00	25.00
5.00	1.00	133.00	7.00
6.00	1.00	133.00	13.00
7.00	1.00	133.00	.00
8.00	1.00	133.00	22.00
9.00	1.00	100.00	4.00
10.00	1.00	133.00	50.00
11.00	1.00	133.00	17.00
12.00	1.00	133.00	65.00
13.00	1.00	133.00	8.00
14.00	1.00	100.00	4.00
15.00	1.00	133.00	-2.00
16.00	.00	50.00	4.00
17.00	1.00	133.00	8.00
18.00	1.00	133.00	13.00
19.00	1.00	133.00	44.00
20.00	.00	50.00	12.00

Experiment 3. Blurred condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	16.00	1.00	1.00	133.00
2.00	3.00	1.00	1.00	133.00
3.00	30.00	.00	.00	100.00
4.00	15.00	1.00	1.00	133.00
5.00	21.00	1.00	1.00	133.00
6.00	29.00	1.00	.00	100.00
7.00	30.00	.00	.00	100.00
8.00	16.00	1.00	1.00	133.00
9.00	30.00	.00	1.00	133.00
10.00	26.00	1.00	1.00	133.00
11.00	16.00	1.00	1.00	133.00
12.00	30.00	.00	1.00	133.00
13.00	16.00	1.00	1.00	133.00
14.00	11.00	1.00	.00	100.00
15.00	19.00	1.00	.00	117.00
16.00	30.00	.00	1.00	133.00
17.00	2.00	1.00	1.00	133.00
18.00	30.00	1.00	1.00	133.00
19.00	20.00	1.00	1.00	133.00
20.00	12.00	1.00	1.00	133.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	1.00	133.00	2.00
2.00	1.00	133.00	12.00
3.00	1.00	133.00	7.00
4.00	1.00	133.00	25.00
5.00	1.00	133.00	7.00
6.00	1.00	133.00	13.00
7.00	1.00	133.00	.00
8.00	1.00	133.00	22.00
9.00	1.00	100.00	4.00
10.00	1.00	133.00	50.00
11.00	1.00	133.00	17.00
12.00	1.00	133.00	65.00
13.00	1.00	133.00	8.00
14.00	1.00	100.00	4.00
15.00	1.00	133.00	-2.00
16.00	.00	50.00	4.00
17.00	1.00	133.00	8.00
18.00	1.00	133.00	13.00
19.00	1.00	133.00	44.00
20.00	.00	50.00	12.00

Subject number	Accumulated illness rating (normal condition)	Accumulated illness rating (blurred condition)	Accumulated vection score (normal condition)	Accumulated vection score (fixation condition)
1.00	39.00	42.00	18.00	23.00
2.00	57.00	59.00	59.00	49.00
3.00	24.00	8.00	.00	.00
4.00	45.00	39.00	36.00	48.00
5.00	23.00	35.00	34.00	44.00
6.00	7.00	17.00	18.00	21.00
7.00	29.00	27.00	47.00	52.00
8.00	36.00	46.00	48.00	48.00
9.00	13.00	16.00	28.00	50.00
10.00	48.00	35.00	37.00	39.00
11.00	17.00	47.00	27.00	27.00
12.00	25.00	12.00	.00	.00
13.00	24.00	37.00	2.00	.00
14.00	17.00	50.00	45.00	45.00
15.00	39.00	34.00	33.00	69.00
16.00	.00	.00	54.00	24.00
17.00	156.00	145.00	33.00	42.00
18.00	4.00	7.00	15.00	22.00
19.00	97.00	36.00	33.00	9.00
20.00	90.00	124.00	3.00	10.00

Experiment 4. Single dot condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	30.00	.00	1.00	133.00
2.00	30.00	.00	1.00	133.00
3.00	30.00	.00	1.00	133.00
4.00	15.00	1.00	1.00	133.00
5.00	18.00	1.00	1.00	133.00
6.00	14.00	1.00	.00	66.00
7.00	30.00	.00	1.00	133.00
8.00	23.00	1.00	1.00	133.00
9.00	30.00	.00	1.00	133.00
10.00	7.00	1.00	1.00	100.00
11.00	23.00	1.00	1.00	100.00
12.00	30.00	.00	1.00	133.00
13.00	30.00	.00	1.00	100.00
14.00	30.00	.00	1.00	133.00
15.00	30.00	.00	1.00	133.00
16.00	30.00	.00	1.00	100.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - M_{total}
1.00	.00	50.00	-2.00
2.00	1.00	133.00	12.00
3.00	1.00	133.00	29.00
4.00	1.00	133.00	12.00
5.00	1.00	100.00	25.00
6.00	1.00	100.00	27.00
7.00	1.00	133.00	18.00
8.00	1.00	133.00	35.00
9.00	1.00	133.00	37.00
10.00	.00	33.00	44.00
11.00	.00	20.00	12.00
12.00	1.00	133.00	7.00
13.00	.00	40.00	2.00
14.00	1.00	133.00	26.00
15.00	1.00	133.00	24.00
16.00	.00	50.00	5.00

Experiment 4. Multiple dot condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	30.00	.00	1.00	133.00
2.00	30.00	.00	1.00	133.00
3.00	18.00	1.00	1.00	133.00
4.00	30.00	.00	1.00	133.00
5.00	30.00	.00	1.00	133.00
6.00	9.00	1.00	.00	66.00
7.00	30.00	.00	1.00	133.00
8.00	25.00	1.00	1.00	133.00
9.00	30.00	.00	1.00	133.00
10.00	25.00	1.00	1.00	100.00
11.00	30.00	.00	1.00	100.00
12.00	30.00	.00	1.00	133.00
13.00	23.00	1.00	1.00	100.00
14.00	30.00	.00	1.00	133.00
15.00	30.00	.00	1.00	133.00
16.00	30.00	.00	1.00	100.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	.00	50.00	-2.00
2.00	1.00	133.00	12.00
3.00	1.00	133.00	29.00
4.00	1.00	133.00	12.00
5.00	1.00	100.00	25.00
6.00	1.00	100.00	27.00
7.00	1.00	133.00	18.00
8.00	1.00	133.00	35.00
9.00	1.00	133.00	37.00
10.00	.00	33.00	44.00
11.00	.00	20.00	12.00
12.00	1.00	133.00	7.00
13.00	.00	40.00	2.00
14.00	1.00	133.00	26.00
15.00	1.00	133.00	24.00
16.00	.00	50.00	5.00

Subject number	Accumulated illness rating (single dot condition)	Accumulated illness rating (multiple dots condition)	Accumulated vection score (single dot condition)	Accumulated vection score (multiple dots condition)
1.00	9.00	4.00	5.00	50.00
2.00	.00	.00	3.00	9.00
3.00	2.00	48.00	20.00	69.00
4.00	42.00	14.00	42.00	46.00
5.00	33.00	23.00	.00	.00
6.00	83.00	115.00	.00	37.00
7.00	8.00	19.00	.00	1.00
8.00	25.00	22.00	28.00	40.00
9.00	23.00	29.00	.00	.00
10.00	44.00	26.00	.00	48.00
11.00	24.00	6.00	80.00	45.00
12.00	25.00	28.00	23.00	24.00
13.00	.00	28.00	.00	23.00
14.00	.00	.00	.00	.00
15.00	.00	3.00	.00	25.00
16.00	1.00	.00	1.00	21.00

Experiment 5. Without vision correction condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	8.00	1.00	.00	10.00
2.00	10.00	1.00	.00	10.00
3.00	9.00	1.00	1.00	133.00
4.00	20.00	.00	.00	10.00
5.00	7.00	1.00	.00	40.00
6.00	11.00	1.00	.00	66.00
7.00	2.00	1.00	.00	10.00
8.00	6.00	1.00	.00	29.00
9.00	20.00	.00	1.00	100.00
10.00	6.00	1.00	.00	29.00
11.00	2.00	1.00	.00	5.00
12.00	18.00	1.00	1.00	100.00
13.00	14.00	1.00	.00	66.00
14.00	20.00	.00	1.00	133.00
15.00	5.00	1.00	.00	5.00
16.00	20.00	1.00	.00	66.00
17.00	20.00	.00	1.00	100.00
18.00	4.00	1.00	1.00	133.00
19.00	9.00	1.00	.00	10.00
20.00	7.00	1.00	.00	66.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	.00	10.00	33.00
2.00	.00	10.00	3.00
3.00	.00	29.00	41.00
4.00	.00	10.00	6.00
5.00	.00	10.00	36.00
6.00	1.00	133.00	11.00
7.00	.00	10.00	7.00
8.00	.00	29.00	14.00
9.00	.00	50.00	20.00
10.00	.00	20.00	1.00
11.00	.00	5.00	27.00
12.00	1.00	133.00	34.00
13.00	.00	50.00	4.00
14.00	.00	29.00	-2.00
15.00	.00	5.00	-2.00
16.00	.00	20.00	16.00
17.00	1.00	133.00	16.00
18.00	.00	40.00	11.00
19.00	.00	10.00	5.00
20.00	.00	66.00	39.00

Experiment 5. With vision correction condition.

Subject number	Survival time (time taken to reach 2)	Censor variable (1= did reach 2, 0= did not reach 2)	Visual acuity at the near point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the near point (%)
1.00	5.00	1.00	.00	133.00
2.00	20.00	.00	.00	133.00
3.00	11.00	1.00	.00	133.00
4.00	20.00	.00	.00	133.00
5.00	7.00	1.00	.00	133.00
6.00	17.00	1.00	.00	133.00
7.00	20.00	.00	.00	133.00
8.00	8.00	1.00	1.00	66.00
9.00	20.00	.00	.00	133.00
10.00	15.00	1.00	.00	100.00
11.00	3.00	1.00	.00	133.00
12.00	20.00	.00	.00	133.00
13.00	20.00	.00	.00	133.00
14.00	20.00	.00	.00	133.00
15.00	20.00	.00	.00	133.00
16.00	10.00	1.00	.00	100.00
17.00	19.00	1.00	.00	133.00
18.00	4.00	1.00	.00	133.00
19.00	12.00	1.00	.00	133.00
20.00	20.00	.00	.00	133.00

Subject number	Visual acuity at the far point (0=lower than 20:20, 1=20:20 or greater)	Visual acuity at the far point (%)	Motion sickness susceptibility - $M_{total.}$
1.00	.00	66.00	33.00
2.00	1.00	100.00	3.00
3.00	1.00	133.00	41.00
4.00	1.00	133.00	6.00
5.00	1.00	133.00	36.00
6.00	1.00	133.00	11.00
7.00	1.00	133.00	7.00
8.00	.00	66.00	14.00
9.00	1.00	133.00	20.00
10.00	1.00	100.00	1.00
11.00	1.00	133.00	27.00
12.00	1.00	133.00	34.00
13.00	1.00	133.00	4.00
14.00	1.00	133.00	-2.00
15.00	1.00	133.00	-2.00
16.00	1.00	133.00	16.00
17.00	1.00	133.00	16.00
18.00	1.00	133.00	11.00
19.00	1.00	133.00	5.00
20.00	1.00	133.00	39.00

Subject number	Accumulated illness rating (with vision correction)	Accumulated illness rating (without vision correction)	Accumulated vection score (with vision correction)	Accumulated vection score (without vision correction)
1.00	96.00	78.00	52.00	43.00
2.00	.00	7.00	55.00	72.00
3.00	22.00	18.00	84.00	20.00
4.00	.00	.00	58.00	48.00
5.00	52.00	60.00	65.00	65.00
6.00	23.00	42.00	53.00	57.00
7.00	19.00	41.00	31.00	51.00
8.00	27.00	23.00	90.00	85.00
9.00	1.00	26.00	87.00	86.00
10.00	5.00	79.00	79.00	94.00
11.00	73.00	92.00	78.00	86.00
12.00	.00	17.00	75.00	72.00
13.00	12.00	31.00	75.00	89.00
14.00	.00	2.00	43.00	68.00
15.00	.00	5.00	93.00	97.00
16.00	30.00	8.00	56.00	36.00
17.00	13.00	14.00	63.00	61.00
18.00	27.00	41.00	72.00	63.00
19.00	27.00	73.00	25.00	30.00
20.00	3.00	46.00	53.00	55.00

Experiment 5. Contrast sensitivity scores – without vision correction.

Subject number	0.3 cycles/°	0.6 cycles/°	1.25 cycles/°	2.5 cycles/°	5 cycles/°	10 cycles/°
1.00	12.00	15.00	16.00	16.00	13.00	15.00
2.00	12.00	15.00	17.00	25.00	25.00	25.00
3.00	16.00	12.00	12.00	10.00	11.00	11.00
4.00	14.00	13.00	13.00	18.00	25.00	25.00
5.00	11.00	12.00	14.00	14.00	15.00	15.00
6.00	7.00	9.00	9.00	10.00	10.00	11.00
7.00	13.00	13.00	19.00	25.00	25.00	25.00
8.00	13.00	12.00	13.00	11.00	14.00	25.00
9.00	13.00	12.00	12.00	11.00	14.00	11.00
10.00	14.00	11.00	13.00	13.00	13.00	15.00
11.00	16.00	15.00	19.00	25.00	25.00	25.00
12.00	14.00	11.00	13.00	13.00	10.00	13.00
13.00	12.00	14.00	13.00	14.00	16.00	15.00
14.00	13.00	11.00	11.00	14.00	11.00	11.00
15.00	15.00	13.00	19.00	25.00	25.00	25.00
16.00	16.00	13.00	13.00	13.00	12.00	13.00
17.00	10.00	11.00	11.00	13.00	14.00	15.00
18.00	13.00	13.00	15.00	15.00	16.00	19.00
19.00	15.00	14.00	14.00	19.00	25.00	25.00
20.00	13.00	14.00	14.00	14.00	17.00	19.00

Experiment 5. Contrast sensitivity scores – with vision correction.

Subject number	0.3 cycles/°	0.6 cycles/°	1.25 cycles/°	2.5 cycles/°	5 cycles/°	10 cycles/°
1.00	12.00	11.00	12.00	13.00	12.00	13.00
2.00	11.00	12.00	12.00	14.00	12.00	11.00
3.00	15.00	13.00	12.00	10.00	12.00	10.00
4.00	11.00	9.00	12.00	12.00	12.00	12.00
5.00	11.00	11.00	10.00	10.00	11.00	11.00
6.00	13.00	10.00	11.00	12.00	11.00	11.00
7.00	13.00	12.00	14.00	12.00	13.00	14.00
8.00	11.00	10.00	12.00	11.00	13.00	13.00
9.00	11.00	10.00	11.00	11.00	14.00	11.00
10.00	11.00	11.00	13.00	12.00	12.00	11.00
11.00	13.00	13.00	15.00	14.00	13.00	13.00
12.00	14.00	12.00	14.00	12.00	13.00	14.00
13.00	11.00	12.00	11.00	13.00	12.00	12.00
14.00	13.00	13.00	13.00	11.00	12.00	12.00
15.00	13.00	11.00	12.00	12.00	15.00	16.00
16.00	14.00	13.00	13.00	13.00	14.00	13.00
17.00	12.00	10.00	12.00	12.00	12.00	12.00
18.00	15.00	13.00	13.00	13.00	13.00	14.00
19.00	11.00	11.00	11.00	12.00	15.00	17.00
20.00	12.00	13.00	13.00	14.00	12.00	12.00

Experiment 6. Slow phase velocity and nystagmus frequencies for both conditions

Subject number	Slow phase velocity with vision correction	Slow phase velocity without vision correction	Nystagmus frequency with vision correction	Nystagmus frequency without vision correction
1	34.73	28.14	2.58	3.00
2	36.45	30.34	2.56	2.82
3	29.72	42.12	2.98	3.02
4	21.00	24.27	2.82	2.67
5	35.41	30.94	2.50	2.87
6	25.83	25.05	2.76	2.36
7	25.28	20.19	3.12	2.16
8	34.17	33.02	2.20	2.32
9	31.43	25.89	2.84	1.80
10	31.52	34.47	2.44	2.66
11	33.59	31.78	3.22	2.84
12	34.44	28.80	2.98	2.36
13	26.77	25.32	2.62	2.34

Experiment 6. Visual acuity and contrast sensitivity scores without vision correction

Visual acuity at the near point (%)	Visual acuity at the far point (%)	0.3 cycles/°	0.6 cycles/°	1.25 cycles/°	2.5 cycles/°	5 cycles/°	10 cycles/°
100.00	10.00	14.00	11.00	14.00	13.00	14.00	16.00
100.00	10.00	15.00	13.00	13.00	14.00	25.00	17.00
33.00	10.00	15.00	13.00	13.00	16.00	16.00	13.00
10.00	10.00	15.00	15.00	16.00	18.00	25.00	25.00
20.00	33.00	14.00	13.00	14.00	16.00	25.00	25.00
10.00	10.00	16.00	15.00	19.00	25.00	25.00	25.00
10.00	10.00	14.00	14.00	17.00	25.00	25.00	25.00
100.00	10.00	13.00	11.00	12.00	14.00	16.00	18.00
10.00	10.00	16.00	14.00	14.00	15.00	14.00	14.00
50.00	50.00	18.00	15.00	16.00	16.00	18.00	18.00
133.00	10.00	14.00	11.00	12.00	13.00	14.00	16.00
29.00	29.00	13.00	12.00	13.00	11.00	14.00	25.00
10.00	10.00	15.00	14.00	14.00	19.00	25.00	25.00

Experiment 6. Visual and contrast sensitivity scores with vision correction

Visual acuity at the near point (%)	Visual acuity at the far point (%)	0.3 cycles/°	0.6 cycles/°	1.25 cycles/°	2.5 cycles/°	5 cycles/°	10 cycles/°
133.00	133.00	13.00	12.00	13.00	13.00	12.00	13.00
133.00	133.00	13.00	11.00	11.00	11.00	9.00	10.00
133.00	133.00	14.00	13.00	13.00	13.00	13.00	11.00
133.00	133.00	16.00	14.00	14.00	15.00	15.00	15.00
133.00	133.00	13.00	12.00	13.00	13.00	12.00	11.00
133.00	133.00	13.00	13.00	15.00	14.00	13.00	13.00
133.00	133.00	14.00	11.00	13.00	13.00	13.00	14.00
133.00	133.00	13.00	13.00	14.00	14.00	16.00	15.00
133.00	133.00	15.00	12.00	13.00	13.00	13.00	13.00
133.00	133.00	17.00	5.00	16.00	16.00	15.00	15.00
133.00	133.00	14.00	13.00	11.00	12.00	12.00	13.00
66.00	66.00	11.00	10.00	12.00	11.00	13.00	13.00
133.00	133.00	15.00	14.00	14.00	19.00	25.00	25.00