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



Figure 2.1. Cross section of the eye.

"rods" and "cones". These are two different types of light receptors with different properties. Rods are by far the more numerous of the two There are receptors. 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. 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).

60

90

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



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

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).

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



Figure 2.5. Contrast sensitivity card from the Arden test.

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. А 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).

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 semicircular 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 а 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 - 3μ sec 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 $40,000^{\circ}$ /second² 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



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.

an average of 14 responses а 10°/second to 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°/sec² to reach a velocity of 6.1°/sec and а 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

used because one can study the initial reaction of the eye and the steady response.

Line 1 in Figure 2.11 shows

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 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 eye 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 afternystagmus induced by this motion. It was discovered that the velocity of afternystagmus 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. Vection experienced with fixation and restricted field of view. (Stern *et al.*, 1990)

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 reduction similar 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.

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. Telford et al. (1993) found that there was significantly more vection 1985). 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 <u>Sensory conflict theory</u>

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

	Category of conflict	
Type of conflict	Visual-Vestibular	Canal-Otolith
Туре 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

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

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.

	Category motion c	ue mismatch
	Visual (A) / Vestibular (B)	Canal(A)-Otolith(B)
ΤΥΡΕΙ	Watching waves from a ship Use of binoculars in a moving vehicle	Making head movements whilst rotating (Coriolis or cross- coupled stimulation)
A and B simultaneously give contradictory or uncorrelated information	Making head movements when vision is distorted by an optical device 'Pseudo-Coriolis' 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	Positional alcohol nystagmus
	Simulator sickness	Caloric stimulation of the semi- circular canals
	Circular vection	Vestibular disorders (e.g. pressure vertigo, cupulolithiasis)
TYPE IIB	Looking inside a moving vehicle without external visual reference (e.g. below deck in a boat)	Low frequency (< 0.5 Hz) translational oscillation
B signals in the absence of expected A signals	Reading in a moving vehicle	Rotating linear acceleration vector (e.g. barbecue spit rotation about an off-vertical axis)

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

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 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

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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 <u>Alternative theories of motion sickness</u>

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



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).

anaesthesia (anaesthetising the muscles behind the eve) 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 stimulation result in 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 still experience vection thev (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.

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 eve 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 antimotion 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 TTSscopolamine. The changes in gain were statistically significant between the different



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.

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.

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.

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



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

that 'the fixation group would experience more vection than the restricted visual field group and, therefore, would experience more symptoms'.

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 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

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 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 inbetween) 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 headmounted 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 headmounted 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°/s² 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 <u>Nauseogenicity of head-mounted displays versus computer monitors</u>

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 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 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.