

Chapter 10. Discussion and final model

10.1 Discussion

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

10.1.1 Vection and motion sickness

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

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

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

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

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

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

10.1.2 Visual acuity and eye movements

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

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

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

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

10.1.3 Visual acuity and motion sickness

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

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

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

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

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

10.1.4 Review of literature and experimental results

10.1.4.1 *Restricted field of view*

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

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

10.1.4.2 *Speed of rotation of the drum*

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

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

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

10.1.4.3 *Strobed lighting*

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

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

10.1.4.4 *Frequency of nystagmus and motion sickness.*

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

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

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

10.1.4.5 Image magnification errors in virtual reality

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

10.1.5 Virtual reality as a tool for motion sickness study

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

10.2 Final model

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

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

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

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

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

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

10.2.1 Explanation of the complete model in detail

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

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

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

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

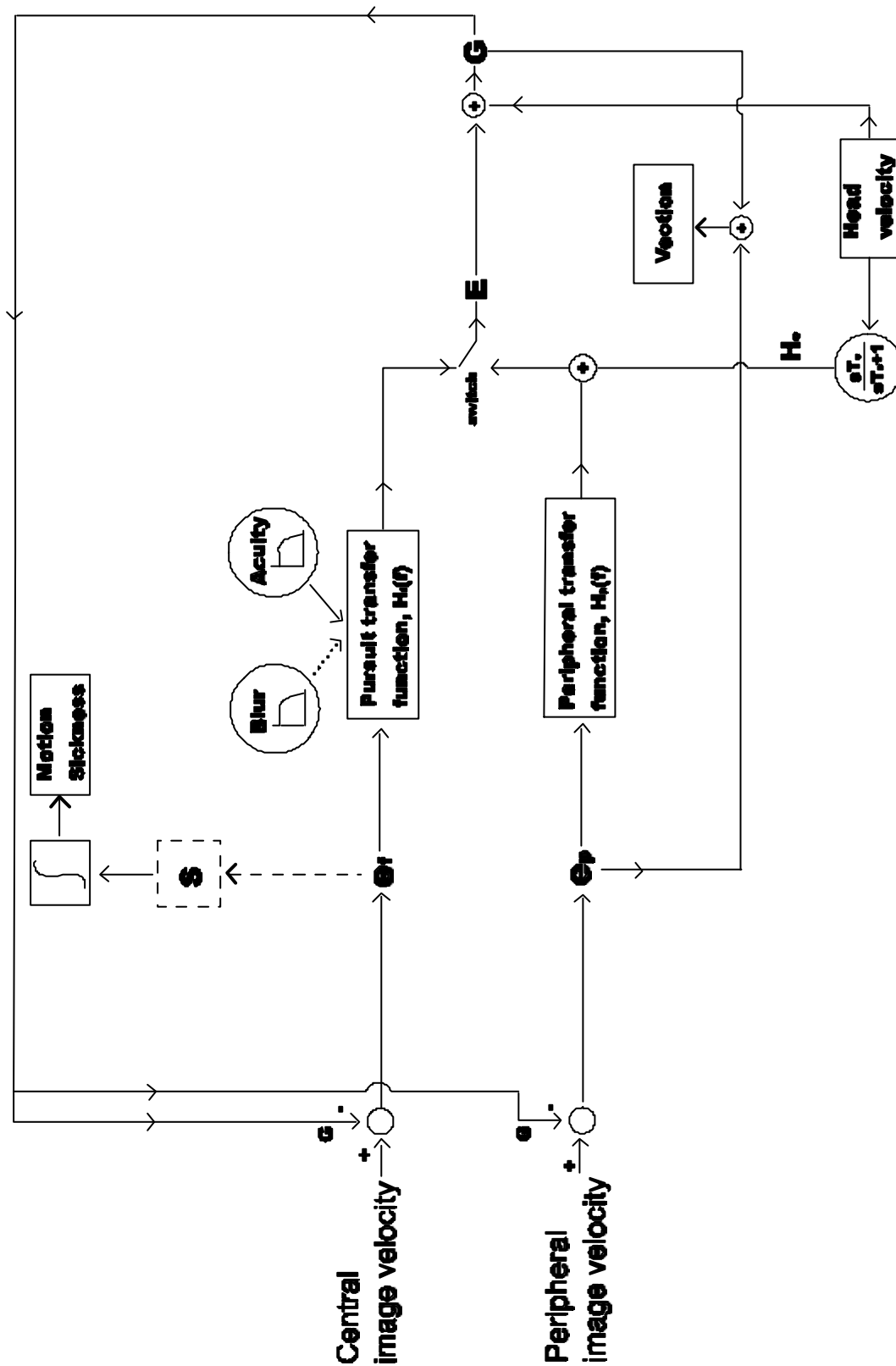


Figure 10.1. The final model, version 5. Head movements have been re-introduced from the original model of Robinson, 1981.

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

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

10.2.1.3 Experiment 3. Motion sickness with and without artificial blurring

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

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

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

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

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

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

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

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