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Vestibular Perception of Prolonged Rotational Stimuli

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Submitted January 1996
A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements of the degree of Master of Science
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As required above, here is an explicit statement of the kind and extent of the contribution made by co-authors of the manuscripts to be submitted for publication based on this thesis: Both Drs. G. Melvill Jones and B. Segal functioned in a supervisory capacity for the work presented in this thesis.
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Abstracts
1.1 Abstract

During the brief, high frequency, head rotations associated for example with the stepping jolts of normal locomotion, the vestibular semicircular canals accurately transduce instantaneous head angular velocity relative to space. However, during more prolonged rotations such as occur when walking along a curved pathway, the canal's biophysics lead to an exponential decay of the peripheral sensory signal with a human time constant on the order of 8 sec. Although this time constant is "neurally" augmented two or three fold in the brainstem, the decaying characteristic still incurs a progressively decreasing signal at this level in the CNS. The present study aimed to determine whether conscious human perception can correct for this form of error in the vestibular sensory signal.

Vestibular perception was estimated from a perception-based motor read out using two complimentary approaches. The first employed gaze saccades (either eyes alone or eye + head) to recapture a remembered target position after a passive head rotation of up to 8 sec, all conducted in complete darkness (Vestibular Memory Contingent Saccades = VMCS). The second study called for continuous "visual" fixation on an imagined earth-fixed target in the dark during the turn (Gaze Stabilization). In both studies final gaze error provided an index of volitional motor response accuracy.

The Results of both studies showed that final gaze position, and hence presumably vestibular perception, was on average not reduced from ideal (P< 0.01, 21 subjects) over the full range of rotational durations. However, the Gaze Stabilization study showed that slow-phase velocity of the compensatory vestibulo-ocular reflex (VOR) followed the expected brainstem signal decay. But this response error tended to be systematically corrected by internally generated saccades. It is inferred that while the slow-phase VOR follows the decaying brainstem signal, the success of both VMCS and Gaze Stabilization saccades in reaching the intended goal reflects veridical perceptual processing of the vestibular signal over the range of these experiments.
1.2 Resumé

Les canaux semicirculaires vestibulaires convertissent avec précision la vitesse angulaire instantanée de la tête relativement à l'espace, lors de brèves rotations à hautes fréquences de la tête qui sont causées entre autre par les mouvements entrecoupés de la locomotion normale. Lors de rotations plus prolongées telles que celles qui surviennent au moment où un individu suit un sentier courbé, la biophysique de ces canaux conduit à une décroissance exponentielle du signal sensoriel périphérique avec une constante de temps humaine à l'ordre de 8 secondes. Bien que cette constante de temps soit augmentée "neuralement" par deux ou trois fois dans le tronc cérébral, cette caractéristique de décroissance amène quand même une diminution progressive du signal à ce niveau du SNC. L'objectif de cette recherche fut de déterminer si la perception consciente humaine peut corriger cette forme d'erreur dans le signal vestibulaire sensoriel.

La perception vestibulaire fut estimée utilisant deux approches complémentaires en se servant d'un indicateur moteur basé sur cette perception. La première utilisa les mouvements saccadiques du regard (soit seulement l'œil, soit œil + tête) pour relocaliser de mémoire la position d'une cible après une rotation passive de la tête, pouvant durer jusqu'à 8 secondes, le tout exécuté en noirceur (Vestibular Memory Contingent Saccades = VMCS). La deuxième approche inclua une fixation "visuelle" continue de la cible immobile et imaginée dans la noirceur pendant la rotation. Dans chacune des études, l'erreur de la direction finale du regard a fourni un index de la précision de la réponse motrice volitive.

Le résultat de ces deux études démontra que la perception vestibulaire déterminée par la position du regard final, en moyenne n'était pas réduite par rapport à l'idéal (P<0.01, 21 sujets) sur l'ensemble des durées rotationnelles. Cependant, l'étude de "Gaze Stabilization" démontra que la vitesse de la phase-lente du réflexe vestibulo-oculaire (RVO) compensatoire suit la décroissance prévue du signal du tronc cérébral. Toutefois, cette erreur de rendement tend à être corrigée systématiquement par des saccades générées intérieurement. Il est inféré que lorsque la phase-lente du RVO suit le signal décroissant du tronc cérébral, les saccades de VMCS autant que celles de Gaze Stabilization réussissent à atteindre le but recherché, démontrant un traitement perceptuel véridique du signal vestibulaire dans l'ensemble de ces expériences.
Introduction and background
2.1 Biophysics of the end-organ lead to an exponentially decaying sensory signal

This investigation is concerned with human perceptual interpretation of the sensory response to natural rotational stimulation of the semicircular canals of the peripheral vestibular sense organs (Figure 2.1). Bear in mind that to a first approximation, these canals are involved purely with the detection of rotational motion (Wilson and Melvill Jones 1979, Schwartz 1986). Pertinent features of a single canal (Figure 2.2) include housing of a fluid, endolymph, within the closed circuit of the canal, and the existence of the cupula membrane within the ampullar area. This membrane achieves complete separation of the endolymph on either side of it. Although it is presently believed that cupula motion behaves as a purely elastic membrane attached all around the canal wall rather than like a swing door (as implied in the figure), either model realizes equivalent mechanical conclusions. First of all, no fluid can pass through or around the membrane. Secondly, there exists an elastic restoring force (like a spring) which "pulls" the cupula back to its initial zero position after a displacement in either direction (Steinhausen 1933, Van Egmond et al. 1949, Wilson and Melvill Jones 1979).

Peripheral canal response

There are three variables within the system that are relevant to these studies.

1. During angular acceleration in the plane of the canal there is an inertial force acting on the endolymph.

2. There is an opposing viscous force due to the induced relative flow of endolymph when in motion (the fluid flow is laminar, owing to the low Reynolds number of the system).

3. There is an elastic restoring force acting on the cupula membrane when it is deflected from its resting position.
Figure 2.1 Semi-Diagrammatic Drawing of the Vestibular Apparatus. The present studies are concerned with angular stimulation in the horizontal plane, which primarily stimulates the lateral semicircular canals. Lat. Canal in diagram denotes right lateral canal. This figure is reproduced from Brödel et al. (1946).

Figure 2.2 Schematic Drawing of a Single Semicircular Canal. The canal contains endolymph fluid. Within the ampullar area is the cupula membrane which achieves complete separation of the endolymph on either side. A head or canal rotation to the left leads to endolymph rotation to the right relative to the canal, which causes the cupula to be displaced. See text for details.
The biophysics of the canal system result in an integrating hydrodynamic response such that cupula position reflects instantaneous rotational velocity when neglecting the elastic restoring force. The canal can be referred to as an integrating accelerometer that outputs instantaneous velocity. Thus if the canal (or the whole framework) is rotating at a certain velocity, the endolymph is also rotating at a certain velocity because of frictional force acting between the walls of the canal and the endolymph. However, during acceleration to this velocity, there is an inertial force acting on the fluid in the opposite direction causing it to "stay behind" relative to the canal, until viscous forces, caused by this relative flow, oppose this relative flow. The result is a cupula displacement causing a neural signal output proportional to instantaneous velocity. This description of events is accurate as long as cupula elasticity is negligible, which is the case during high frequency or short duration head movements; however, with longer duration constant velocity rotations within the range of natural movement, cupula elasticity can no longer be ignored (reviewed in Wilson and Melvill Jones 1979, chapter 3). See Appendix A for the derivation of the second order equation describing these transduction characteristics of the semicircular canals as well as a Bode plot illustrating their response to sinusoidal stimulation.

Figure 2.3 illustrates what happens during a prolonged turn by plotting instantaneous (canal) rotational velocity (bottom curve) and predicted cupula displacement (top curve), both against time in seconds, during and after cessation of a 6 sec rotation. The initial (maximal) cupula response corresponds to the rotational velocity of 10 °/sec, one of the stimulus velocities used during the experiments described in this thesis. The velocity profile shows a step increase in velocity to 10 °/sec maintained constant for 6 seconds, and then decreased to 0 velocity by a step function again. If there was no elastic restoring force, the cupula membrane would follow the ideal, dashed line; it would be displaced by a constant amount during the
Figure 2.3 Cupula Response to Prolonged Step Angular Velocity Stimulus. A rotational velocity stimulus and the corresponding predicted cupula displacement, are both shown as a function of time. The stimulus velocity (bottom) is a step increase held constant for 6 sec and then returned to zero velocity. The cupula response (top) is shown as the solid line marked actual, assuming an 8 sec time constant of exponential decay. The dashed line marked ideal shows what the cupula response would be if it was not elastic. See text for details.
constant velocity rotation, finally to be "pushed" back to the initial zero position due to the deceleration back to zero velocity.

But in reality, due to its elasticity the cupula follows an exponential decay back towards its initial position, eventually reaching zero position if the velocity was held constant at 10 °/sec. The time constant for decay presented here is 8 sec which is in the higher range of estimates of the human time constant for the peripheral end organ (cf. section 2.3). Thus after 8 sec, the cupula position should be about 1/3 of its original value. But in this case, there is a step decrease in velocity at 6 sec equal in amplitude to the original increase. Hence the cupula is displaced by an equal amplitude to its original displacement, yet in the opposite direction. The membrane then follows an exponential positional decay back to zero again (since velocity is once again constant - this time at 0 °/sec).

If it is assumed that cupula displacement is sensed as head angular velocity, then to derive position from its response, an integration must be carried out. Consequently, the area under the cupula response curve would represent this derived positional signal. With the curve representing cupula position in the absence of an elastic restoring force, the total amplitude of rotation is integrated: 10 °/sec x 6 sec = 60°. However, the area under the actual cupula positional curve (continuous line) is on the order of 35°, the error introduced by cupula elasticity being about 25°. It is a remarkable fact that this mechanical response is more or less faithfully transduced through the complex train of mechano-electrical transductions in the vestibular sensory epithelium (e.g. Roberts et al. 1988, Howard et al. 1988), to yield similar dynamics in the primary afferent neural signal fed into the brainstem (Fernandez and Goldberg 1971).
Central neural response

However, this peripheral signal is altered when it reaches the brainstem level. Afferent input to the vestibular nuclei is presumed to be supplemented by input from an internal positive feedback loop, the effect of which has been characterized as "velocity storage" (Raphen and Cohen 1985). The result of this phenomenon is an extension of the time constant of the exponentially decaying peripheral signal to two to three times its original magnitude. Thus the CNS signal reaches 1/3 of its initial value after about 16 to 24 sec instead of 8 sec as it did in the periphery. Hence, after a 6 sec rotation at 10°/sec, the brainstem signal would register an angular displacement of about 46° for a 16 sec time constant, or about 50° if the time constant is approximately 24 sec. Thus under these stimulus conditions, the predicted brainstem level signal representing angular position would still be erroneous by about 14° to 10°.

2.2 Problem formulation

Nevertheless, normal individuals get along fine in this "vestibular error prone" environment. They do not appear to be disoriented during normal behaviour. The question therefore arises: How is veridical perception of spatial orientation achieved in these circumstances. Of course other cues such as proprioception, tactile sensations, and especially vision, may compensate for any vestibular deficiencies. For example with vision, much work has been done concerning the optokinetic system's role in compensating for peripheral vestibular dynamics (e.g. Robinson 1977, Raphen et al. 1979).

These experiments set out to determine whether perception of vestibular sensation follows this "false" vestibular output when there are no other sensory cues to correct for any error. Would these vestibular errors be accounted for, perhaps by some form of central "look up table" gained both from past sensory and motor experience? Or
would perception follow the central representation of the erroneous canal signal? Possibly the outcome would lie somewhere in between these two extremes.

2.3 Experimental estimates of peripheral and brainstem level time constants

Since it is not possible to measure directly the peripheral or brainstem level human vestibular time constant, secondary indices are used to estimate their values. For the brainstem level time constant in adults, the method most often used quantifies the profile of slow-phase eye movement decay, per or post rotation, with a step or impulse angular velocity stimulus. Optokinetic stimuli, that is prolonged full field visual movements (e.g. Leigh and Zee 1991), are also used to elicit slow-phase eye movements. Similar eye movements are examined in infants and newborns to estimate the human peripheral time constant, with the assumption that CNS modulation of its value has yet to occur. Estimates of both these time constants vary greatly owing to, amongst other things, the arousal level or the gaze direction goal of the subject. Peripheral values can range from about 6 to 7.5 sec (Ornitz et al. 1985, Weissman et al 1989). Brainstem level values can range from about 10 to 18 sec (Balo, Honrubia et al. 1984, Ornitz et al. 1985, Weissman et al 1989). For these studies, a 24 sec brainstem level time constant value was chosen when modeling brainstem processing. Such a high value was used to insure that a higher experimental perceptual gain, when compared to theoretical results derived from known brainstem level processing, would not be due to an error in underestimating the brainstem level time constant. Thus, it was decided to err on the side of caution by assuming a long brainstem level time constant.
2.4 Vestibular perception and low frequency rotational stimuli

The burgeoning space program of the 1960s was a strong impetus to study human subjective reactions to vestibular stimuli that spanned the full range of known canal dynamics (Niven et al. 1965, Benson 1968). Psychophysical studies have used magnitude estimation techniques, subjective measures of angular velocity, such as velocity matching, as well as subjective measures of angular displacement to measure perception of horizontal rotation during low frequency stimuli. The results from these studies are highly variable, with consistency often depending on extensive subject training for the required task (reviewed in Guedry 1974). However, these psychophysical estimates did mostly manifest an exponentially decaying signal and were often used to approximate the value of the peripheral time constant (Guedry 1974). Recently, Mergner et al. (1991 and 1993) have done extensive studies on spatial perception relating to interactions of vestibular and proprioceptive inputs. Their perceptual gains were based on magnitude estimates as well as subjective manipulation of external angular pointers. In their studies of purely vestibular stimuli using sinusoidal profiles, they found an attenuated perceptual gain during low frequency stimuli. However, their results showed gain attenuation to be linked to stimulus amplitude. Based on their results, they proposed a model that employed a perceptual threshold to account for the attenuated gain at low frequencies, rather than canal dynamics.

Keeping the above studies in mind, there were two main criteria that the present study aimed to meet: 1) The vestibular rotational stimuli to be used had to possibly occur during natural movement. Previous studies have shown that vestibular psychophysical results are more consistent when using experimental stimuli that approximate naturally occurring stimuli (Guedry 1974). 2) The perceptual indicator had
to be behaviourally natural. If it is hypothesized that some form of "look up table" will compensate for deficient canal dynamics, it is also reasonable to assume that such accurate perception will become apparent when using an appropriate motor indicator with a goal oriented task. It was therefore decided to employ a novel form of "Gaze Motor Readout" of the vestibular percept, using the two complimentary methods detailed below.
Measuring Vestibular Perception Using Vestibular Memory Contingent Saccades (VMCS)
3.1 Introduction

Normally, during a passive rotation in the dark, the vestibulo-ocular reflex (VOR) functions to help stabilize the direction of regard in space (Wilson and Melvill Jones 1979, Gauthier and Robinson 1975). The efficacy of the linear or rotational VOR depends on the arousal level (Torok 1970) and on the visual-motor goal of the rotating subject (Barr et al. 1976, Baloh, Lyerly et al. 1984, Melvill Jones et al. 1984, McKinley and Peterson 1985, Skipper and Barnes 1989, Fadlallah 1995); for example, VOR gain normally reaches close to ideal if the subject is instructed to maintain gaze fixation of a just seen earth-fixed target.

Presumably, subjects have some knowledge of their body position relative to space during such a rotation, based on vestibular perception. A particularly effective way to estimate this vestibular perception was introduced by Bloomberg et al. (1988) who used the Vestibular Memory Contingent Saccade (VMCS) method to quantify motor output of a goal directed task. The basic concept of the VMCS protocol is to suppress the above mentioned VOR during a passive rotation in the dark, and then to look back to the remembered position of a just seen earth-fixed target while still in the dark. Thus the goal directed motor output, gaze refixation of the remembered target, must be based only on the subject's perception of how much the subject was rotated. In turn, this perception is based solely on the vestibular sensory information incurred by the passive rotation. The present experiments set out to measure this perception based motor output during prolonged turns, as a means of estimating the accuracy of the perception of the stimulus.
3.2 Methods

3.2.0 General setup and procedure

a. Equipment

All subjects were seated in a servo-controlled rotating chair. For safety, subjects were secured by a quick release lap and shoulder harness as well as a leg restraint. They were also provided with an emergency servo shut off switch that cut off the power to the rotating chair. Chair angular position could be read off a compass rose attached to the chair's base as well as recorded from a rotatory potentiometer.

The chair was surrounded by a concentric cylindrical drum whose angular position could be read off an outer compass rose. It was covered on its inner surface by a visual scene encompassing a wide range of spatial frequencies and image intensities. Adhered to this scene, at the subject's eye level (and surrounding the subject), was a horizontal, marked reference tape calibrated in degrees and located at a radial distance of 57 cm from the subject's eyes (refer to Figure 3.1). With the chair and drum at their initial 0° positions, a visual target was affixed to the reference tape so that it appeared in the center of the subject's visual field. This target consisted of a filled in red circle with a radius of 2 mm, surrounded by a white border with a width of 4 mm, again surrounded by a red border of 4 mm width. To the left and right of the target, marked on the tape in 5° intervals, were short vertical lines labeled with angular displacement from the 0° target position. To the left of the target, these lines and numbers were labeled in blue, from 5° to 180°; to the right, in red, from 5° to 175°.

Above the subject's head, was a radially oriented chair-fixed metal arm, that ended in an adjustable red light emitting diode (LED), approximately 1.5 cm from the inner surface of the drum. At the beginning of a trial, this LED was positioned directly
Figure 3.1 Schematic top view of seated subject surrounded by cylindrical drum and affixed reference tape
under the target, in such a way, that neither the arm nor the LED itself impeded the subject's view of the target.

b. Data acquisition and recording

Variable-duration VMCS protocol:

Changes in chair angular position were determined from the compass rose at the chair's base and recorded by hand by the experimenter. The subject's gaze, or direction of regard, was determined using the concentric reference tape as described below in the section entitled "Training procedure". Subjects called out their gaze position verbally and the numbers were recorded by the experimenter.

Variable-velocity VMCS protocol:

Data were acquired as in the variable-duration VMCS protocol with the additional recording of change in drum angular position recorded by the experimenter from the outer compass rose; however, corroborating data were also acquired as follows. DC bitemporal electrooculography (EOG) was used for measuring horizontal eye movements relative to the head. A shaft-mounted precision (1% full scale error) potentiometer measured the chair's angular position relative to space. A Watson Industries' angular velocity transducer, with a full field range of (+/-)1000°/sec output as (+/-)10 volts, was firmly mounted on a snug headband and was used to measure horizontal head angular velocity. The voltage signals controlling the drum light switch as well as the LED switch were also recorded. Data were digitally recorded at 166.6 Hz on a PDP 11/73 computer using VAN¹ software, after fourth order, low pass filtering at 100 Hz (adequate parameters for subsequent analysis).

c. Training procedure

Before being tested, the subject (S) was "trained" in a few essential skills until feeling completely comfortable performing these tasks while strapped in to the chair:

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¹Written by Robert Douglas
1) During rotations, S's head was kept still relative to the body by grasping the chin with one hand while that hand was hugged to the body at the elbow by the other hand. This method of head stabilization was adopted because in some elements of the protocol S was required suddenly to release the head and move it relative to the body. S practiced keeping the head stationary, as in Figure 3.2, while being passively and abruptly rotated at different velocities (up to 60°/sec). A control was performed on one subject who was affixed to a goniometer measuring displacement of head relative to chair; no relative movement could be seen. In the protocols in which such recordings occurred, the integrated head angular velocity and chair angular position profiles were identical.

2) S was submerged in darkness and asked to visually "fixate" on an imagined earth-fixed target at any point (at eye level) around her or him. The lights were then turned on and S had to decide on the position on the tape that S was looking at (possibly by pointing to this position with a finger if it was more comfortable doing so). This procedure was repeated until S was confident in being able to accurately identify the point fixated in darkness, once the lights were turned on. Finally S was asked to call out the angular displacement from the 0° target position that S had fixated upon, using the lines and numbers as a reference (e.g. "31.5° blue" would indicate fixation of a point 31.5° left of the target). S practiced this procedure until feeling confident in accurately being able to determine the position of fixation, within a 0.5° error range.

3) The third training procedure consisted of centering the head on the target. S was asked to center the head approximately on the target. Then S was asked to close the right eye, and look as far right as possible. The bridge of the nose would thus cut off the visual field to the right and S would determine how many degrees could be seen on the right (using the reference tape). This procedure was then repeated closing the left
eye, and determining the left visual field. Thus if S's nose cut off the right visual field (with the right eye closed) at 40°, and there was a 30° leftward visual field (with the left eye closed), S would rotate the head 5° to the left, i.e. until there was a 35° visual field on either side. S practiced this procedure of centering the head on different marked lines on the reference tape, until feeling comfortable doing it quickly.

4) Finally, S was coached slowly through the experimental procedures described below until feeling at ease with the routine.

d. General protocol

The experimental procedure required the subjects to undergo a passive change in whole body angular position. During the rotation, the vestibulo-ocular reflex was effectively suppressed by fixation of a chair-fixed, and thus head-fixed, LED. After cessation of rotation, the task then required subjects to match a saccadic gaze movement to the amount of "vestibularly" perceived passive head movement in order to re-fixate an original target. Thus subjects were required to make voluntary saccades (purely oculomotor or with head and eye together), in darkness, to the position of a previously seen earth-fixed target following a passive change in whole body (and thus head) angular position. Trials were only continued if making saccades was comfortable for S, i.e. well within S's gaze motor range as allowed by the protocol.

The trial began, in the light, by centering the chair and torso on a protocol-dependent predetermined point relative to the target. A subject then centered the head on another protocol-dependent predetermined point on the reference tape as trained, and then stabilized the head as in Figure 3.2. S was then asked to relax while maintaining this position. S then fixated the target and was told to remember where it was. Visual fixation was then transferred to the chair/head-fixed LED, directly underneath the target.
Figure 3.2 Head Stabilization Method
Within 1 sec, but at an exact time unknown to S, the drum lights were extinguished and simultaneously the servo-controlled chair began to rotate at a constant velocity. During the rotation, S suppressed the VOR by maintaining fixation on the still visible chair-fixed LED. S also continued to keep the head stationary relative to the chair and torso throughout the passive rotation as described above. After a predetermined rotational duration that was dependent on the protocol, the chair decelerated to 0 °/sec in another step velocity change.

Instantaneously, the LED shut off. This cue signaled S to make a voluntary gaze saccade to the perceived (remembered) position of the original earth-fixed target, while still in complete darkness. This gaze saccade was either purely oculomotor or head and eye combined, depending on the protocol. In the case of a pure oculomotor gaze saccade, S continued keeping the head stationary as trained. 1.5 sec after the LED was turned off, the drum light turned on and S called out the estimated point of visual fixation as earlier trained. S's gaze position thus yielded a presumed motor read-out of the remembered vestibular percept in the form of an error in degrees off of target.

Immediately after calling out the point of fixation, all illumination was extinguished and S was passively rotated under identical stimulation parameters but in the opposite direction. This second rotation was performed to minimize any post-rotational error built up by continuous unidirectional displacement of the cupula membrane. After this second rotation was completed, signaling the end of the trial, S stayed relatively immobile for a minimum duration of 60 sec to allow the decay of any residual post-rotational vestibular stimulation, before starting another trial. Direction of rotation was not randomized because of the fact that larger amplitude turns necessitated a protocol that revealed the direction of the impending turn, at the beginning of the trial. These constraints will become apparent in the following, more detailed, protocol descriptions.
3.2.1 Variable-Duration VMCS Protocol

Nine subjects (aged 20-67 years) participated in this experimental protocol. Subjects were seated such that the chair's axis of rotation was orthogonal to the direction of vision and passed through the subject's mid interocular point. At the end of an individual trial, a subset of the subjects was asked to state a number out of a scale of 10 describing confidence in the accuracy of the just performed saccade, that is confidence before the lights came on. This number was duly recorded at the end of the trial.

a. Eyes straight, oculomotor saccade

In this series, 8 subjects participated in the procedure outlined in Figure 3.3. The objective was to overlap with the stimulus parameters of previous studies (Bloomberg et al. 1988, Bloomberg et al. 1991b). To start the trial (left Figure 3.3) the chair and torso, head, and eyes were *all* positioned on the target/LED. Thus the eyes were "straight" relative to the head. The trial proceeded as described in the general protocol (center Figure 3.3) the servo-controlled chair rotating at 10°/sec. In this diagram, rotation was to the left. Recall that during the rotation, the subject suppressed the VOR by maintaining fixation on the chair-fixed LED. The rotational duration was 2 sec and thus the rotational amplitude was 20°. The LED shutting off signaled the subject to immediately make a voluntary pure oculomotor saccade to the perceived (remembered) position of the original earth-fixed target. In the diagram (right Figure 3.3), the fixation point is indicated as 5° blue, giving an undershooting error of 5°. This undershooting would indicate a diminished vestibular perception compared to actual passive rotation.
Figure 3.3 Variable-Duration VMCS: Example of a Single Trial in the Eyes Straight, Oculomotor Saccade Protocol. Refer to Figure 3.1 for a description of the setup. Chair and torso, head, and eyes all begin straight ahead on target/LED (left in diagram). The chair rotates (ccw in this example) at 10°/sec for 2 sec in the dark, as the subject maintains LED fixation (center in diagram). At the end of the rotation the LED switches off signalling the subject to look back to the perceived position of the earth-fixed target with an oculomotor saccade, in the dark. 1.5 sec later the lights come on (right in diagram) and the subject calls out gaze position as determined from the reference tape.
b. Eyes deviated, oculomotor saccade

There were three related series of trials in this protocol. Here the objective was to extend the durations and angles of rotation. In this protocol, 9 subjects participated; however, one subject was unable to complete the last (largest rotational amplitude) series because he wished to make saccades beyond his oculomotor range. Figure 3.4 refers to the last series in the protocol, but may be used to illustrate all three series. The chair and torso were positioned facing 10° for the first series, 20° for the second, and 30° for the third, to either the left or right of the target, depending on the trial. The chair-fixed LED was adjusted to be directly under the target. S then centered and stabilized the head at the same position as the chair and torso, 30° left (or blue) in the diagram and then fixated the LED/target by turning the eyes 30° to the right. Thus the eyes were deviated 10°, 20°, and 30° relative to the head in the progressing series. The rest of the trial was identical to the previous eyes straight protocol (10°/sec rotation), except for the stimulation duration. The first series of this protocol had a rotational duration of 2 sec to overlap with the eyes straight protocol. The second and third series had durations of 4 and 6 sec respectively. Rotation was always in the opposite direction to initial head deviation relative to target (i.e. in the same direction as eye deviation relative to head). In the center diagram, S undergoes a 60° rotation in darkness to the right so that chair, torso, and head end up facing 30° right (or red), whilst the eyes are (gaze is) directed 60° right relative to the target. On extinguishing the LED, S attempts to relocate the original target with a saccade to the left; a 60° saccade would fixate the target. The voluntary oculomotor saccade has undershot again in this fictional example, this time giving an error of 10°.

The purpose of beginning the trial with eyes deviated relative to head was to allow saccades to be made within an extended oculomotor range (full field range of approximately 90°). Thus by beginning with eyes deviated 30°, a 60° oculomotor
Figure 3.4 Variable-Duration VMCS: Example of a Single Trial in the Eyes Deviated, Oculomotor Saccade Protocol. Refer to Figure 3.1 for a description of the setup. This example corresponds to the last series in this protocol which had the longest duration stimulus. For this example, the chair and torso and head all begin centered on 30° blue (or ccw), while the eyes are on target/LED (left in diagram). The chair rotates (cw in this example) at 10°/sec for 6 sec in the dark, as the subject maintains LED fixation (center in diagram). At the end of the rotation the LED switches off signalling the subject to look back to the perceived position of the earth-fixed target with an oculomotor saccade, in the dark. 1.5 sec later the lights come on (right in diagram) and the subject calls out gaze position as determined from the reference tape.
saccade was possible by sweeping the eyes across the midline. Unfortunately, use of this method precluded randomization of rotational direction since S was aware, by the direction of eye deviation, of which way the rotation would be. The previous protocol (eyes straight) served as a control for the first series within this protocol, initial eye deviation being the only variable altered.

c. Head and eye saccade

Six subjects participated in this series, which aimed to extend the range of the experiment by introducing combined head-eye gaze saccades to locate the perceived target.

The trial began by positioning the chair and torso on 40° blue or red. In Figure 3.5, the deviation is 40° red (right of the target). S then centered the head on the 30° mark (right in the diagram) with the eyes on LED/target at 0°. Thus the eyes were deviated 30° relative to the head, which was itself deviated 10° relative to the torso, bringing gaze 40° off the torso and on LED/target. The rest of the trial continued as in the other series, however, with a stimulation duration of 8 sec (rotation was to the left in the diagram). Thus at the end of rotation in the dark in Figure 3.5, the chair and torso were on 40° blue (left of the target), the head on 50° blue, and the LED and thus eyes (gaze) on 80° blue. The immediate extinguishing of the LED signaled the subject to saccade back to the perceived target, in the dark, but this time with a combined head and eye movement. Thus, at the end of the trial, individual deviations of eye or head from the target were unknown; but, gaze fixation, or head plus eye fixation was indicated by the number and colour called out by the subject after illumination (right Figure 3.5). In the diagram, the subject undershot by 7.5°. It should be pointed out that before this series began, the subject was "trained" in making combined head and eye saccades (i.e. gaze saccades) until a satisfactory feeling of comfort was reached. Note
Figure 3.5 Variable-Duration VMCS: Example of a Single Trial in the Head and Eye Saccade Protocol. Refer to Figure 3.1 for a description of the setup. For this example, the chair and torso begin on 40° red (or cw), the head is centered on 30° red (or cw), while the eyes are on target/LED (left in diagram). The chair rotates (ccw in this example) at 10 °/sec for 8 sec in the dark, as the subject maintains LED fixation (center in diagram). At the end of the rotation the LED switches off signalling the subject to look back to the perceived position of the earth-fixed target with a combined head and eye saccade, in the dark. 1.5 sec later the lights come on (right in diagram) and the subject calls out gaze position as determined from the reference tape.
that the head was stabilized relative to torso/chair until the LED shut off, signaling S to release the head and make a saccade.

3.2.2 Variable-Velocity VMCS Protocol

These trials were performed as a separate set of experiments from the variable-duration VMCS protocol, at a later date. This protocol was comparable to the last series of the variable-duration VMCS protocol. That is, perception of rotation was determined using a head and eye combined gaze saccade at the cessation of rotation. The overall variable-velocity VMCS protocol differed from the variable-duration VMCS protocol in that duration of rotation was kept constant (at 6 sec) while velocity of rotation was varied (between 7.5'/sec and 15'/sec), in order to assess linearity of vestibular perception. Furthermore, in this series, computer aided data acquisition was implemented, allowing one to calculate gaze (eye + head) shifts from separate eye and head records. Note that the subjective "confidence" values asked of some subjects in the variable-duration VMCS protocol were not a part of this protocol.

All 5 subjects (aged 20-68 years), 2 of whom overlapped from the variable-duration VMCS protocol, were similarly seated in the servo-controlled rotating chair. However, this time the chair's rotational axis was aligned with the yaw axis of rotation of the subject's head when rotating relative to the body, determined as described below.

During the training procedure for this protocol, S was asked to rotate her or his head back and forth in yaw so that the experimenter could mark its axis of rotation with a circular sticker on top of the head. This sticker was used as the marker with which to align the chair's axis of rotation once S was seated in the chair.

There was only one series of experimentation and all 5 subjects participated. The trial began by deviating the chair/torso 60° relative to the target. The head was then centered 10° relative to the target and thus 50° relative to the torso. The eyes were on
LED/target. The rest of the trial proceeded as in the last head and eye combined saccade series of the variable-duration VMCS protocol, except the stimulation parameters were different.

All rotations lasted 6 seconds, but the velocity of rotation could be 7.5°/sec, 10°/sec, 12.5°/sec, or 15°/sec resulting in rotational amplitudes of 45°, 60°, 75°, or 90° respectively. The velocities were presented in a pseudo-random order so that S would not know the amplitude of the imminent rotation. There were 20 trials in one direction with the 4 randomized velocities, and then similarly 20 trials in the other direction. There were thus 10 trials for each velocity split 5 and 5 in each direction. All trials, no matter what amplitude, began in the same position for one stimulus direction. Thus initial gaze (eye + head) deviation re torso/chair was chosen to ensure there would be no physical difficulty in refixating the target, even after a maximum chair rotation of 90° with eyes fixed on the LED. Specifically in the case of maximum amplitude, since gaze began deviated 60° relative to the torso, only a further 30° gaze deviation across the "torso's midline" would be needed to redirect gaze onto the target.

Another feature of this protocol was that there was no possibility of post-test cognitive feedback of perceptual error. During the passive stimulus rotations in the dark, the surrounding concentric drum, which was also attached to a servo-controlled motor, was rotated known amounts to the experimenter, as read off a separate compass rose. Thus the number that S called out, was arbitrary relative to target error as far as S was concerned. This procedure was implemented because of evidence that it is possible to cognitively modulate the VOR and hence VMCS gain depending on feedback of performance (Fadlallah 1995, Fadlallah and Melvill Jones 1990). The end of the trial was followed, as before, with a counter rotation in the dark using the same stimulus parameters, to offset any canal error buildup.
Calibration trials for the EOG system and the angular velocity transducers were performed at the beginning of the experiment and at least after every 10 trials. A calibration trial consisted of S stabilizing the head relative to the torso (and chair) as previously trained, and then fixating the target in the light. The chair, initially starting centered on the target, was then manually rotated back and forth through 40° peak to peak amplitude rotations, as S maintained gaze fixation of the target in the light. These records were later used to calibrate the channels for eye relative to head, and head as well as chair relative to space.

3.2.3 Data analysis

a. Variable-duration VMCS protocol

Stimulus amplitude in degrees was determined from the change in chair angular position read off the compass rose. Gaze error, in degrees off of target, was determined from the number and colour called out by S. The convention adopted was such that a positive error indicated undershooting and a negative error indicated overshooting. Total gaze saccadic amplitude in degrees was determined by taking the stimulus amplitude and subtracting the gaze error. The gaze saccade was obviously in the opposite direction to the stimulus rotation; keeping this fact in mind, the gaze saccade vector amplitude had its sign (direction) reversed before being compared to the stimulus rotation vector amplitude. Thus dividing the gaze saccadic amplitude by the stimulus amplitude yielded the standardized VMCS gain.

b. Variable-velocity VMCS protocol

Values for stimulus amplitude, gaze error, and gaze saccadic amplitude were determined as in the variable-duration VMCS protocol. However, the change in position of the concentric drum, recorded by the experimenter, was used along with the number and colour called out by S in determining the gaze error value. Values for
stimulus amplitude and gaze saccadic amplitude were also determined from the computer acquired data as follows.

The VAN software language was used for data manipulation and analysis. All recorded channels from an individual trial were displayed on a graphic terminal for characterization as described below. To determine head angular position, the head angular velocity signal was digitally integrated. This head position signal was used to determine stimulus amplitudes rather than the chair position signal; however, the chair position signal was used to verify head stabilization during the turn. Figure 3.6 shows an individual trial for subject E, a 7.5 °/sec rotation for 6 sec. The eye position re head, integrated head velocity re space, and gaze position (eye + head) re space signals are shown. To minimize data storage requirements, a drum light marker signal was combined with an LED-off marker, the start of which indicated when the LED was extinguished. At the top of the figure, the drum light and LED signals are shown: The shaded area indicates that the drum lights are off, and the LED-off marker "bump" shows when the LED was extinguished. Stimulus amplitude was measured as the change in integrated head velocity, or head position, from stimulus start to stop; gaze saccadic amplitude was measured as the change in gaze position from when before the LED was extinguished to just before the drum lights came back on. Once again, ultimately the magnitudes of the two values were compared after reversing the direction of the gaze saccadic amplitude. As for the variable-duration protocol, dividing the gaze saccadic amplitude by the stimulus amplitude yielded the standardized VMCS gain.

c. Analysis of variance

Analysis of variance (Scheffe 1959, SAS software package2), or ANOVA, was used to evaluate whether various elements of the above protocols (e.g. stimulus direction) and/or inter-subject variability significantly influenced VMCS gains. The

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Figure 3.6 A Single Variable-Velocity VMCS Protocol Trial. A single trial of a 7.5 °/sec rotation for 6 sec is shown for subject E. Eye re head, head re space, and gaze (eye + head) re space position signals are shown. The shaded bar at the top of the figure indicates the period of darkness. The marked "bump" on the same bar shows when the LED was extinguished signaling performance of the VMCS with the head released. Note that gaze follows the head during the passive turn as the VOR is suppressed by LED fixation.
method used was to assume that VMCS gains could be accounted for by various linear models in which gain was a function of several independent variables (e.g. stimulus direction).

3.3 Results

3.3.1 Variable-Duration VMCS Protocol

Figure 3.7 shows the relationship between the VMCS amplitude and imposed head displacement for one subject (E) for all the trials in the variable-duration VMCS protocol. Each symbol represents one trial. So each symbol is a plot of the motor output or gaze saccadic amplitude in degrees, reflecting vestibular perception, against the stimulus amplitude in degrees, expressed as head rotation relative to space. Note that on the abscissal axis, an increase in stimulus amplitude also implies an increase in stimulus duration. The scatter of points for this subject is a bit less than the rest of the subject population for the first 3 stimulus durations, and a bit more for the last, 8 second, stimulus. However, the tight fit of a 1st order regression line \( r^2 = 0.988 \) to the progressive increase in gaze amplitude with stimulus duration is typical for the whole population. This regression line has been fitted to the data from the eyes deviated protocol for the 2, 4, and 6 second stimuli and has a slope of 0.948 (and a y intercept of 2.00). Note that the 2 second stimulus duration includes both eyes straight (squares) and eyes deviated (circles) trials, and that the 8 second stimulus corresponds to the head free gaze saccade trials (triangles). The black symbols represent clockwise trials and the white symbols, counterclockwise trials. Table 3.1 compares results for this subject with other subjects in terms of VMCS gain means and standard deviations.
Figure 3.7 VMCS Amplitude Vs. Stimulus Amplitude for One Subject. This figure plots all the trials for subject E in all protocols of the Variable-duration VMCS experiment. The ordinate axis shows the gaze motor output based on perception, and the abscissa axis shows the stimulus amplitude. Note that an increase in stimulus amplitude also corresponds to an increase in stimulus duration, as all trials had a 10 °/sec stimulus velocity. The dashed line marked ideal shows where points would lie if the motor output perfectly matched the stimulus input. Black symbols and white symbols represent clockwise and counterclockwise trials respectively. Squares, circles, and triangles correspond to eyes straight, eyes deviated, and head free saccade protocols respectively. The regression line is fitted to the eyes deviated protocol data.
Table 3.1 Variable-Duration VMCS Gain Means: Variable-duration VMCS subject VMCS gain means (and standard deviations) by stimulus duration (and protocol). "All" signifies population VMCS gain means (and standard errors of the mean). All stimulus velocities were 10 °/sec. A VMCS gain mean of unity implies ideal stabilization. Means with standard deviations typically are based on 10 trials; means with standard errors typically are based on 90 trials.

<table>
<thead>
<tr>
<th>Subject</th>
<th>2 sec eyes straight</th>
<th>2 sec</th>
<th>4 sec</th>
<th>6 sec</th>
<th>8 sec head + eye saccade</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1.09 (0.176)</td>
<td>1.18 (0.163)</td>
<td>0.940 (0.0984)</td>
<td>0.942 (0.0995)</td>
<td>0.896 (0.108)</td>
</tr>
<tr>
<td>B</td>
<td>1.12 (0.122)</td>
<td>1.09 (0.119)</td>
<td>1.04 (0.177)</td>
<td>0.864 (0.106)</td>
<td>0.954 (0.0591)</td>
</tr>
<tr>
<td>C</td>
<td>1.01 (0.0222)</td>
<td>1.01 (0.0404)</td>
<td>0.968 (0.0354)</td>
<td>0.932 (0.0781)</td>
<td>0.982 (0.0384)</td>
</tr>
<tr>
<td>D</td>
<td>1.15 (0.329)</td>
<td>1.43 (0.284)</td>
<td>1.10 (0.149)</td>
<td>0.958 (0.0746)</td>
<td>1.05 (0.137)</td>
</tr>
<tr>
<td>E</td>
<td>1.07 (0.0425)</td>
<td>1.05 (0.0527)</td>
<td>0.996 (0.0302)</td>
<td>0.981 (0.0453)</td>
<td>1.02 (0.108)</td>
</tr>
<tr>
<td>F</td>
<td>0.986 (0.0452)</td>
<td>1.00 (0.318)</td>
<td>0.877 (0.140)</td>
<td>0.908 (0.0657)</td>
<td>0.995 (0.0154)</td>
</tr>
<tr>
<td>G</td>
<td>0.848 (0.123)</td>
<td>1.06 (0.207)</td>
<td>1.15 (0.129)</td>
<td>1.01 (0.136)</td>
<td>0.891 (0.152)</td>
</tr>
<tr>
<td>H</td>
<td>1.53 (0.0787)</td>
<td>1.52 (0.191)</td>
<td>1.35 (0.132)</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>I</td>
<td>na</td>
<td>0.898 (0.0682)</td>
<td>0.802 (0.0897)</td>
<td>0.762 (0.0769)</td>
<td>na</td>
</tr>
<tr>
<td>All</td>
<td>1.10 (0.0263)</td>
<td>1.14 (0.0279)</td>
<td>1.03 (0.0202)</td>
<td>0.920 (0.0126)</td>
<td>0.970 (0.0133)</td>
</tr>
</tbody>
</table>
Figure 3.8 (axes as in Figure 3.7) shows the population results, with each letter representing the mean for all trials for each of the nine subjects in the protocol for that duration. Thus each letter symbol typically comprises the mean of 10 trials. Note that the 2 second stimulus has the mean of trials for the eyes straight protocol as well as a separate mean for the eyes deviated protocol (duplicate letter symbol used). Take note that subject H was not able to complete the protocols for durations passed the 4 second stimulus as his intended gaze saccades were out of his motor range as allowed by the experimental protocol. Also, subject I did not participate in the eyes-straight protocol nor the head free gaze saccade protocol. The linear regression line fitted to the subject average values with a least squares method ($r^2=0.881$) has a slope of 0.815 (and a y intercept of 7.12) and once again corresponds to the eyes deviated trials for the 2, 4 and 6 second stimuli.

Figure 3.9 (axes as in Figure 3.7) shows the population results, with each point representing the mean for all trials in the protocol for that duration. Thus each point typically comprises the mean of 70 to 90 trials. The black circles represent the means for the eyes deviated protocol with the pure oculomotor saccade at the end of the trial. The white circle represents the mean for the eyes straight protocol (only with a 2 sec stimulus duration) and the white square the mean for the head free saccade protocol. Standard error bars (in the x and y direction) are included for each mean. The regression fit is the same as for Figure 3.8. Note that the head free saccade protocol mean seems to be closer to the ideal dashed line than the 6 sec stimulus (pure oculomotor) mean.

The theoretical results of a subject whose perception follows an exponentially decaying signal have also been presented, for three different time constants of decay. These results were determined by feeding step velocity profiles into simple models of exponential decay. The upright white triangles, upside down white triangles, and white
Figure 3.8 VMCS Amplitude Vs. Stimulus Amplitude for All Subjects. This figure plots subject averages for the population in all protocols of the Variable-duration VMCS experiments, with letter symbols denoting each subject. Note that there are duplicate letter symbols at the 2 sec (20 deg) stimulus because it includes both eyes straight and eyes deviated protocol trials. The regression line is fitted to the eyes deviated protocol data.
Figure 3.9 VMCS Amplitude Vs. Stimulus Amplitude for Population. This figure plots population averages for all protocols in the Variable-duration VMCS experiment. Standard error bars are included in the x and y direction. The white and black circles represent eyes straight and eyes deviated protocols respectively. The white square plots the head free saccade results. The regression line is fitted to the eyes deviated protocol data. The theoretical results of a subject whose perception follows three different exponentially decaying signals have also been presented. The three time constants of decay are 8, 16, and 24 sec, represented by the upright white triangles, upside down white triangles, and white diamonds respectively. All experimental means (except for the 2 sec eyes straight protocol) are significantly higher (p < 0.05) than the corresponding theoretical value for a 24 sec time constant of decay.
diamonds show theoretical results for time constants of 8, 16, and 24 seconds respectively. All experimental means (except for the 2 sec eyes straight protocol) are significantly \( p < 0.05 \) higher than corresponding theoretical values for a 24 second time constant of decay.

Table 3.2 shows, by subject, the results of an Analysis of Variance (ANOVA) of VMCS gain values from all trials. Results from the 8 sec stimulus trials have not been included in these ANOVAs since they were derived from head free saccades as noted below. The dependent variable in these ANOVAs was VMCS gain. The independent variables in the ANOVA model are represented in the labels of different columns. The first column shows the subject whose VMCS gain values were placed in the ANOVA model ("All" implying the whole population). Independent variables found to contribute significantly to the variation of the dependent variable, VMCS gain, within the model are signified by asterisks in the appropriate column; the null hypothesis is that the independent variable does not contribute: ** for \( p \leq 0.01 \), * for \( p \leq 0.05 \), - for \( p > 0.05 \), and na for "not applicable". The column entitled "Model" shows whether the ANOVA model itself is statistically significant, that is, whether the model significantly accounts for variance of VMCS gain. The last two rows show ANOVA results for the whole population of trials excluding the 8 sec stimulus trials. The very last row shows results from a population ANOVA with one more independent variable, Subject. This variable is comprised of 9 levels for the 9 subjects. As would be expected, inter-subject variability significantly affects VMCS gain.

Note that both population ANOVAs as well as all subject ANOVAs save one, show Duration as a significant independent variable. This result was implied by the less than unity slope of the linear regression fit in Figure 3.9 which shows that as duration increases, the gaze saccadic amplitude decreases compared to the stimulus amplitude. The reason why the 8 sec stimulus trial gains were not included in the model was to
Table 3.2 Variable-Duration VMCS ANOVAs of VMCS Gain for 2, 4, and 6 sec Stimulus Durations: The results of an Analysis Of Variance (ANOVA) of the Variable-duration VMCS gain values by subject as well as for the whole population. This ANOVA did not include the 8 second head and eye combined saccade protocol. Note that subject H only did 2 and 4 sec trials. VMCS gain was the dependent variable and the independent variables were as follows: Direction of stimulus - clockwise or counterclockwise; Eye deviation - yes or no (the eyes were deviated for one of the 2 second protocols); Duration - 2, 4, or 6 sec stimulus.

The last row shows a separate ANOVA done on the whole population at once where there was another independent variable, Subject. This variable comprised the subject initials above - 9 subjects and thus 9 levels to the variable.

The column labeled Model shows whether the overall ANOVA model was significant. In all cases, ** represents statistical significance at the 0.01 level, * represents statistical significance at the 0.05 level, - represents no statistical significance (P>0.05), and na stands for "not applicable". Note that adding Subject as an independent variable in the population ANOVA makes whether or not the eyes are deviated statistically significant; refer to Table 3.4.
show the statistical significance of the stimulus duration's affect on VMCS gain, in the same light as the regression fit of Figure 3.9. The regression fit in that figure does not include values from the 8 sec stimulus since it was a separate protocol, which seemed to have "better" results. From the ANOVA results, of the 9 subjects, 5 subjects' net gains depend on the direction of stimulus and 2 subjects have different results depending on whether or not the eyes are deviated at the beginning of the trial. The following tables examine these results more closely.

Table 3.3a shows results from similar ANOVAs as Table 3.2, except all trials, including the 8 sec stimulus trials, have been added to the models. Furthermore, another independent variable was added, namely whether or not the saccade was head free. Unfortunately, the model was unable to determine whether this variable contributed to the variation of VMCS gain values, because there was only one stimulus duration during which the head was released for saccades, and there were no pure oculomotor saccade trials at this stimulus duration of 8 sec for comparison. The only minor effect of adding the 8 sec stimulus trials, and presumably the extra independent variable, was the loss of a significant influence of stimulus direction on one of the subjects' net gains (A).

Table 3.3b takes the subjects from Table 3.3a who showed that VMCS gain was influenced by the stimulus direction of the trial, and presents means and standard deviations for all the trials in the whole experiment by stimulus direction. It also shows the results from t tests of whether the two directional means are significantly different. Three of the 4 subjects have significantly different means but it is important to note that only one subject (G) has means that are functionally different: about a 0.2 difference in mean gains compared to less than 0.1 differences in mean gains for the other subjects. This difference in directional gain of subject G is of the same magnitude as the directional asymmetries occurring in the variable-velocity protocol to be presented
Table 3.3a Variable-Duration VMCS ANOVAs of VMCS Gain for All Protocols: The results of ANOVAs done by subject and for the whole population for the Variable-duration VMCS protocol including all trials in the experiment. Once again VMCS gain was the dependent variable and the independent variables were as described for the previous Table 3.2, except as follows: The Duration variable - 2, 4, 6, or 8 sec stimulus (the previous table did not include the 8 sec trials); Head and Eye Saccade - yes or no, only the 8 sec stimulus had a head and eye saccade. Since the 8 sec stimulus did not also have trials where there was no head and eye saccade, the ANOVA cannot determine the statistical significance of adding this variable to the protocol. Significance levels are as described for Table 3.2.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Model</th>
<th>Direction Stimulus</th>
<th>Eye deviation</th>
<th>Duration</th>
<th>Head and Eye Saccade</th>
<th>Subject</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>**</td>
<td>-</td>
<td>-</td>
<td>**</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>B</td>
<td>**</td>
<td>**</td>
<td>-</td>
<td>**</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>C</td>
<td>**</td>
<td>*</td>
<td>-</td>
<td>**</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>D</td>
<td>**</td>
<td>-</td>
<td>**</td>
<td>**</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>E</td>
<td>*</td>
<td>-</td>
<td>**</td>
<td>*</td>
<td>na</td>
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</tr>
<tr>
<td>F</td>
<td>-</td>
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<td>na</td>
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</tr>
<tr>
<td>G</td>
<td>**</td>
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<td>*</td>
<td>na</td>
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</tr>
<tr>
<td>H</td>
<td>*</td>
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<td>na</td>
<td>na</td>
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<td>I</td>
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<tr>
<td>All</td>
<td>**</td>
<td>-</td>
<td>**</td>
<td>**</td>
<td>na</td>
<td>na</td>
</tr>
</tbody>
</table>

Table 3.3b Variable-Duration VMCS A Subgroup of Directional Means and t Tests: Means (and standard deviations) are given for subjects who had a statistically significant difference in VMCS gain with different stimulus directions in the ANOVAs above. Significance levels from a group mean t test are also given for these subjects. Note that only subject G has a relatively large functional difference in VMCS gain depending on direction.

<table>
<thead>
<tr>
<th>Subject</th>
<th>VMCS gain counterclockwise stimulus</th>
<th>VMCS gain clockwise stimulus</th>
<th>Group mean t test</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>0.965 (0.144)</td>
<td>1.06 (0.144)</td>
<td>*</td>
</tr>
<tr>
<td>C</td>
<td>0.966 (0.0573)</td>
<td>0.995 (0.0466)</td>
<td>-</td>
</tr>
<tr>
<td>F</td>
<td>0.903 (0.130)</td>
<td>1.00 (0.176)</td>
<td>*</td>
</tr>
<tr>
<td>G</td>
<td>1.09 (0.132)</td>
<td>0.889 (0.132)</td>
<td>**</td>
</tr>
</tbody>
</table>
(compare to Table 3.6b where the differences in directional least squares mean gains range from about 0.16 to over 0.4).

Table 3.3c shows a similar table to Table 3.3b, except it presents results for subjects who showed a significant contribution of eye deviation towards the variation of VMCS gain values. Only the 2 sec stimulus duration trials are presented. Of the two subjects, only one subject (G) had significantly different VMCS gain means for the different protocols at the same duration, as determined by a t test. If this finding is compared to those of Table 3.4, this relationship is consistent; only G has a significant contribution of eye deviation. This table shows the results from ANOVAs done on the 2 sec stimulus trials, with only one independent variable (except for the first row). Having one independent variable with two levels, eye deviation here, is equivalent to doing a t test. Note that the population results only show a significant contribution of eye deviation to VMCS gain variation, when Subject is also included as an independent variable. This relationship is consistent with all the ANOVAs presented in previous tables. Perhaps, adding Subject as an independent variable simply makes the overall model sensitive to the one subject (G) who had a significant difference in VMCS gains for the two protocols.

There was no significant correlation between subjective confidence and VMCS gain, for the trials in which subjects gave a number out of a scale of 10 representing confidence in the saccade made.

3.3.2 Variable-Velocity VMCS protocol

Figure 3.10 compares the VMCS amplitude data acquired using EOG and an angular velocity transducer, to the VMCS amplitude data acquired using the subject's own visual estimates from the reference tape, for subject J. The ordinate axis shows the
Table 3.3c Variable-Duration VMCS A Subgroup of Means and t Tests for the 2 sec Stimulus: Means (and standard deviations) are given for subjects who had a statistically significant difference in VMCS gain depending on whether or not the eyes were deviated in the ANOVAs above. Significance levels from a group mean t test are also given for these subjects.
Table 3.4 Variable-Duration VMCS ANOVAs of VMCS Gain for the 2 sec Stimulus: Results of simple ANOVAs done on only the 2 sec stimulus VMCS gain values. Once again the dependent variable is VMCS gain. Note that for the whole population, whether the eyes are deviated or not is no longer significant when Subject is dropped as an independent variable (compare rows one and two). Having Eye deviation as the sole independent variable is equivalent to doing a t test comparing the eyes straight protocol to the eyes deviated protocol. Note that subject I did not do the eyes straight protocol.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Model</th>
<th>Eye deviation</th>
<th>Subject</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>**</td>
<td>*</td>
<td>**</td>
</tr>
<tr>
<td>All</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>A</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>B</td>
<td>-</td>
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</tr>
<tr>
<td>C</td>
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<td>D</td>
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<tr>
<td>E</td>
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<td>-</td>
<td>-</td>
</tr>
<tr>
<td>F</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>G</td>
<td>*</td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>H</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>na</td>
<td>na</td>
<td></td>
</tr>
</tbody>
</table>

Note: Table values represent statistical significance levels. ** indicates p < 0.01, * indicates p < 0.05.
Figure 3.10 Objective VMCS Amplitude Vs. Subjective VMCS Amplitude for One Subject. All trials for subject J are shown in this figure plotting gaze amplitude as acquired by computer with EOG and a velocity transducer, and as acquired by verbal reports of reference tape fixation. Black and white symbols represent clockwise and counterclockwise trials respectively. The regression line's slope of 1.06 closely straddles the ideal dotted line.
results obtained by computer aided data acquisition and the abscissal axis shows the
data acquired using the subjects verbal reports. The solid line shows a regression fit (r^2
= 0.916) with a slope of 1.06 (and a y intercept of -3.59). Ideally, for each trial, the x
value would equal the y value, giving a line with a slope of 1. Figure 3.11 shows a
similar graph with all trials for all subjects in the variable-velocity VMCS protocol. The
regression fit (r^2 = 0.954) has a slope of 0.935 (and a y intercept 2.67).

Figure 3.6, already referred to in Methods section 3.2.3 b, shows an individual
trial for subject E. The VMCS gain for this trial was 1.05.

Figure 3.12 shows all the trials in the variable-velocity VMCS protocol for one
subject (E). Each symbol represents one trial of gaze saccadic amplitude plotted against
stimulus amplitude. Note that an increase in stimulus amplitude also implies an increase
in stimulus velocity, not an increase in stimulus duration as it did in the variable-
duration VMCS protocol. Black symbols represent clockwise stimuli and white
symbols represent counterclockwise stimuli. The scatter of points for this subject is
typical for the population. The 1st order regression fit (r^2=0.440) has a slope of 0.542
(and a y intercept of 34.9). The VMCS gain means and standard deviations for
different velocities for subject E is included in Table 3.5.

Figure 3.13 (axes as in Figure 3.12) shows the population results, with each
point representing the mean for all trials in one stimulus direction and one stimulus
velocity for one of the five subjects. Subject E from the Figure 3.12 is represented by
the square symbols. Thus each point typically comprises the mean of 5 trials. Black
symbols signify a clockwise stimulus and white symbols signify a counterclockwise
stimulus. The dashed regression lines are fitted to all the data for one subject in one
stimulus direction. Note the stimulus direction dependent asymmetry of VMCS gain (as
seen in Table 3.6a and 3.6b) that is idiosyncratic to each subject. The solid regression
Figure 3.11 Objective VMCS Amplitude Vs. Subjective VMCS Amplitude for Whole Population. All trials for all 5 subjects are shown in this figure plotting computer acquired, and verbally acquired gaze amplitudes against each other, as described for Figure 3.10. The regression line's slope of 0.935 again is close to ideal.
Figure 3.12 VMCS Amplitude Vs. Stimulus Amplitude for One Subject. This figure plots all the trials for subject E in the Variable-velocity VMCS experiment. The ordinate axis shows the gaze motor output based on perception, and the abscissal axis shows the stimulus amplitude. Note that an increase in stimulus amplitude also corresponds to an increase in stimulus velocity, as all trials had a 6 sec stimulus duration. The dashed line marked ideal shows where points would lie if the motor output perfectly matched the stimulus input. Black symbols and white symbols represent clockwise and counterclockwise trials respectively. The regression line is fitted to all the data.
Table 3.5 Variable-Velocity VMCS Gain Means: Variable-velocity VMCS subject VMCS gain means (and standard errors) for the 4 different stimulus velocities. "All" signifies the population VMCS gain means (and standard errors of the mean). All stimulus durations were 6 sec.

<table>
<thead>
<tr>
<th>Subject</th>
<th>7.5°/sec</th>
<th>10°/sec</th>
<th>12.5°/sec</th>
<th>15°/sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>J</td>
<td>1.04 (0.162)</td>
<td>0.907 (0.201)</td>
<td>0.922 (0.197)</td>
<td>0.854 (0.131)</td>
</tr>
<tr>
<td>E</td>
<td>1.30 (0.233)</td>
<td>1.05 (0.148)</td>
<td>1.03 (0.163)</td>
<td>0.966 (0.152)</td>
</tr>
<tr>
<td>G</td>
<td>1.37 (0.244)</td>
<td>1.27 (0.136)</td>
<td>1.26 (0.0933)</td>
<td>1.16 (0.0694)</td>
</tr>
<tr>
<td>K</td>
<td>1.70 (0.446)</td>
<td>1.54 (0.369)</td>
<td>1.36 (0.221)</td>
<td>1.36 (0.203)</td>
</tr>
<tr>
<td>L</td>
<td>1.05 (0.368)</td>
<td>0.850 (0.308)</td>
<td>0.866 (0.241)</td>
<td>0.822 (0.240)</td>
</tr>
<tr>
<td>All</td>
<td>1.29 (0.0542)</td>
<td>1.12 (0.0495)</td>
<td>1.09 (0.0377)</td>
<td>1.03 (0.0367)</td>
</tr>
</tbody>
</table>
Figure 3.13 VMCS Amplitude Vs. Stimulus Amplitude for All Subjects, Separated by Stimulus Direction. This figure plots subject averages by stimulus direction for the Variable-velocity VMCS experiment, with axes as in Figure 3.12. The dashed line marked ideal shows where points would lie if the motor output perfectly matched the stimulus input. Black symbols and white symbols represent clockwise and counterclockwise averages respectively. Dashed lines are regression fits to all trials in one direction for one subject. Note the idiosyncratic asymmetries in response for all subjects. The solid line is a regression fit to the subject averages not separated by direction.
line $\left( r^2=0.388 \right)$ is fitted to the subject averages with both stimulus directions included (cf. Figure 3.14), and has a slope of 0.761 (and a y intercept of 24.1).

Figure 3.14, like Figure 3.13, shows the population results by subject; however, in this case, the subject means have not been separated by stimulus direction. Thus each letter symbol typically comprises the mean of 10 trials. The regression line is fitted to these averages with parameters as described in Figure 3.13. Table 3.5 shows the mean and standard deviation values of VMCS gain by stimulus velocity, for each subject.

Figure 3.15 (axes as in Figure 3.12) shows the population results, with each point representing the mean for all trials at one of the four stimulus velocities. Thus each point typically comprises the mean of 50 trials. Standard error bars (in the x and y direction) are included for each mean. The regression fit is as in Figure 3.13.

Once again the theoretical results of a subject whose perception follows an exponentially decaying signal have also been presented, for three different time constants of decay, as calculated by feeding step angular velocity stimuli through simple transfer functions representing exponentially decaying signals. The symbols for the time constants are the same as in Figure 3.10. All experimental means are significantly higher (p<0.05) than the corresponding theoretical values for a 24 second time constant of decay. In fact, when looking at the VMCS gain mean for all trials for all subjects (1.13), it is significantly higher than 1 (p<0.01).

Table 3.6a shows the results of ANOVAs done by subject and for the whole population where the dependent variable is VMCS gain and the independent variables are direction of stimulus, velocity of stimulus, and when the whole population is looked at, Subject. All trials in the experiment were considered for the ANOVA models. Note that direction of stimulus is a significant contributor to the variance of VMCS gains for all subjects individually, but not for the population as a whole.
Figure 3.14 VMCS Amplitude Vs. Stimulus Amplitude for All Subjects. This figure plots subject averages without differentiating between stimulus directions, for the Variable-velocity VMCS experiment. The solid line is a regression fit to these subject averages.
Figure 3.15 VMCS Amplitude Vs. Stimulus Amplitude for Population. This figure plots population averages in the Variable-duration VMCS experiment with standard error bars included in the x and y direction. The theoretical results of a subject whose perception follows three different exponentially decaying signals again have been presented. The three time constants of decay are 8, 16, and 24 sec, represented by the upright white triangles, upside down white triangles, and white diamonds respectively. All experimental means are significantly higher ($p < 0.05$) than the corresponding theoretical value for a 24 sec time constant of decay.
Table 3.6a Variable-Velocity VMCS ANOVAs of VMCS Gain: The results of ANOVAs by subject as well as for the whole population where the dependent variable is VMCS gain. Independent variables are as follows: Direction of stimulus - clockwise or counterclockwise; Velocity - 7.5, 10, 12.5, or 15 °/sec; and Subject (when doing an ANOVA of the whole population) which has 5 levels. Note that the direction of stimulus is not statistically significant when looking at the population as a whole.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Model</th>
<th>Direction stimulus</th>
<th>Velocity</th>
<th>Subject</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>**</td>
<td></td>
<td>**</td>
<td>**</td>
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<tr>
<td>All</td>
<td>**</td>
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<tr>
<td>J</td>
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<td>**</td>
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<tr>
<td>E</td>
<td>**</td>
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<tr>
<td>G</td>
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<tr>
<td>K</td>
<td>**</td>
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<tr>
<td>L</td>
<td>**</td>
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</tbody>
</table>

Table 3.6b Variable-Velocity VMCS ANOVA Least Squares Means of VMCS Gain for Stimulus Direction: The Least squares means, i.e. marginal means that would be expected had the experimental design been balanced for the independent variable, stimulus direction. The ANOVA model used was the one in Table 3.6a above (not including Subject as an independent variable). The t test tests the hypothesis that the two LS means are equal and is rejected in all cases except when looking at the entire population.

<table>
<thead>
<tr>
<th>Subject</th>
<th>LS mean clockwise stimulus</th>
<th>LS mean counterclockwise stimulus</th>
<th>t test</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>1.15</td>
<td>1.12</td>
<td>-</td>
</tr>
<tr>
<td>J</td>
<td>1.03</td>
<td>0.830</td>
<td>**</td>
</tr>
<tr>
<td>E</td>
<td>0.966</td>
<td>1.20</td>
<td>**</td>
</tr>
<tr>
<td>G</td>
<td>1.34</td>
<td>1.18</td>
<td>**</td>
</tr>
<tr>
<td>K</td>
<td>1.73</td>
<td>1.24</td>
<td>**</td>
</tr>
<tr>
<td>L</td>
<td>0.669</td>
<td>1.13</td>
<td>**</td>
</tr>
</tbody>
</table>
Furthermore, for all but one subject (J), velocity is also a significant contributor of variance, as it is for the population as a whole. Once again, this result follows from the less than unity slope of Figure 3.14, which implies a velocity-dependent gain.

Table 3.6b shows the least squares means of VMCS gain by direction, that is, marginal means that would be expected had the experimental design been balanced for the independent variable, stimulus direction. The ANOVA models used to calculate these means were from Table 3.6a (not including Subject as an independent variable). The t test column shows whether the hypothesis that the two directional means are equal can be rejected; it is rejected in all cases except when looking at the population as a whole.

3.4 Discussion

The primary object of these experiments was to investigate the accuracy with which human subjects can perceive passive rotational stimuli which are sufficiently prolonged to evoke markedly decaying vestibular sensory signals, but which can occur during natural behaviour. The Results demonstrate that, rather than following the presumed decaying vestibular signal, subjects tended to achieve veridical perception of the magnitude of imposed turns, despite the fact that relevant sensory information was confined to the vestibular system.

However, given this general outcome the following factors need to be addressed in greater detail for the separate protocols.
3.4.1 Variable-Duration VMCS Protocol

Accuracy of Perception

As reported in the Results section, all experimental means at the different stimulus durations are significantly higher than theoretical results based on perception presuming a 24 sec time constant of decay. In fact they generally cluster round the ideal dotted lines of Figures 3.8 and 3.9. It is however interesting that in these figures the solid lines depicting averaged data indicate a less than "ideal" slope of the regression fits. This trend might imply a progressively underestimating perception with longer turns. However, the undershooting estimates of longer turns may not be due to the turns being longer, but due to them being larger. In fact, the estimates of the 2 second stimulus actually significantly overshoot (p<0.01) the proper value. Such perceptual overestimation of smaller stimuli and underestimation of larger stimuli can be explained by the range effect (Poulton 1975, Bloomberg 1989).

Range Effect

The basic postulate of the range effect is that perceptual estimations through a variety of different sensory modalities consistently lead to overestimation of the lower values in a range, and underestimation of the higher values in the range. The middle values tend to be the most accurate. Looking at the regression fit line in Figure 3.9, the range effect phenomenon can clearly be seen, reassuring us that the VMCS protocol is indeed a perceptual measure. In fact when looking at VMCS gains for the population, not only is the 2 second stimulus mean (1.12) significantly above the ideal value of 1, the 6 second stimulus mean (0.920) is significantly below 1 (p<0.01) and the midrange 4 second stimulus mean (1.03) is not significantly different from ideal. Thus, assuming that the range effect is influencing the results of our subject population, correcting for this effect would rotate the regression line in Figure 3.9 counterclockwise with the 4
second stimulus point as the "fulcrum". Such a rotation would lead to the "corrected" results generally overlapping with the ideal dashed line. However, one cannot conclusively say that the less than ideal slope does not signify a legacy of progressive error in velocity transduction with increased stimulus duration. Nevertheless, it is important to keep in mind that the range effect is a perceptual phenomenon, and presumably would be functioning during studies such as these. Also, it has been shown before to function within the saccadic system (Kapoula and Robinson 1986, Kapoula 1985), including the VMCS protocol (Bloomberg 1989).

Eye Deviation

A concern raised with part of the VMCS protocol was that the necessary off center deviation of eyes pre- and per- rotation might affect the results. However, the extra trials done at the 2 second stimulus comparing eyes deviated (VMCS gain mean of 1.14) and eyes straight (VMCS gain mean of 1.10) show no significant difference in results for the population as a whole, and no significant difference in results individually in all subjects save one. Hence it seems unlikely that this factor played a role in the overall results.

Head and Eye Saccades

An interesting feature seen in Figure 3.9 is the markedly reduced error associated with head free estimates of target location, compared to the longest pure oculomotor trial (p<0.01) or compared to an extension of the regression fit to the three previous durations (p<0.01). Although derived from only one rotational amplitude, the finding is remarkable on account of the fact that this "improved" data point was obtained with the longest duration of rotation, which necessarily incurs the largest real canal error (see diamonds and triangles in Figure 3.9). The population VMCS gain mean of 0.970 for this head free trial was just significantly below an ideal value of 1 (p<0.05).
Allowing combined eye-head saccadic movement was considered justifiable on account of the following three factors: It is "behaviourally" more natural; it alleviates mechanical oculomotor constraints allowing longer stimulus durations and hence larger canal errors; there is strong evidence that saccadic motor programming for visually fixating targets is made in coordinates of gaze (head plus eye) and not solely those of eye (Guitton et al. 1984, Guitton et al. 1986, Guitton and Volle 1987, Guitton and Cullen 1995). Furthermore, vestibular stimulation caused by head movement while making the saccade does not have time to build up a significant error signal to add to the existing one. It is possible that releasing the head did result in the subject making a "behaviourally" more natural saccade. A more natural motor output of spatial perception might have provided easier access to a learned central "lookup table". Access might have been facilitated since such a table presumably would be based on interactions between sensory signals and motor behaviour as well.

Subjective Confidence of Individual Perceptual Estimates

Although the data for the subjective confidence levels could not be correlated to accuracy in perception trial by trial, in general subjects were confident in the accuracy of the saccades they made. However, an incidental experimental observation should be noted: Even though subjects expressed confidence in the accuracy of saccades made after longer duration stimuli, they found the task of keeping a mental image of target position more difficult during the longer turns. With longer turns, subjects reported an urge to make a gaze saccade to the imagined target position, before the turn was completed. Thus visual fixation of the LED was essential not only to suppress the VOR (Barnes and Grealy 1992), but also to suppress this urge. Furthermore, previous experiments have shown that visual suppression of the VOR is actually more effective during lower, rather than higher, frequency rotational stimuli (Barnes and Edge 1984).
Some subjects reported that in the head-free saccade trials, they found it easier to suppress the per-rotation saccadic urge, and easier to keep an imagined target position in mind, knowing that the head would be free during the saccade. This observation supports the concept of gaze saccadic motor programming being done in terms of head and eye rather than eye alone.

Possible neural activity at the superior colliculus during a VMCS trial, based on the Guitton and Munoz model

It would be interesting at this point to propose a hypothesis of neural activity at the level of the superior colliculus (SC), which could partially explain both of the incidental observations above. The observations were 1) an urge to make a saccade before completion of the turn, and 2) a better ability to suppress this urge when it was known that the head would be released for the eventual saccade.

Guitton and Munoz have proposed a still controversial closed loop model of collicular commands for gaze saccades (Guitton et al. 1990; reviewed in Guitton 1991), outlined below. The model proposes that an instantaneous motor error signal (closed loop) is available at the SC level rather than only the initial motor error signal assumed by conventional open-loop models. Their model proposes that 'orientation' tecto-reticular neurons and tecto-reticulo-spinal neurons (together referred to as TR(S)Ns) in the deep layers of the SC are active for a particular vector error between the intended saccade target and the visual axis. These TR(S)Ns project onto long-lead burst neurons (LLBNs) in the gaze motor system, with an excitatory signal encoding saccade amplitude. LLBNs project to the premotor neurons responsible for a saccade. In the rostral SC are TR(S)Ns active for a vector error of 0° which they call 'fixation' TR(S)Ns. These TR(S)Ns prevent initiation of saccades during attentive fixation by conveying excitation to omnipause neurons (OPNs) also in the gaze motor system.
OPNs function as a gate, tonically inhibiting the premotor neurons responsible for a gaze saccade. OPNs and LLBNs reciprocally inhibit each other.

In their model, the dynamic gaze motor error signal reaching the SC is derived from subtracting an actual gaze position signal (G) from a desired gaze position signal (Gd). They propose that before a gaze saccade, there is TR(S)N activity in the rostral 'fixation' area (Munoz and Wurtz 1992) representing present gaze position, and a build up of activity at a more caudal area representing the intended saccade. Commencement of a saccade is contingent upon the attenuation of activity in the rostral gaze fixation area. What is unique to their model is the proposition that during the gaze shift, the TR(S)N activity representing gaze vector error and hence gaze saccade amplitude signal, actually moves towards the rostral fixation area until target fixation is accomplished. This movement of neural activity would be based upon the dynamic error signal (= Gd - G) reaching the SC. Thus during a gaze shift, it is G, the actual gaze position, that is changing. To build upon their model, perhaps during a VMCS protocol G is once again changing the gaze motor error signal, this time because of the passive rotation. Thus in this case G solely would be based upon interpretation of the sensory vestibular signal since the eye is not moving relative to head (LED fixation), but head is moving relative to space.

Steps 5-7 in Figure 3.16 represent the traditional Guitton-Munoz "movement of neural activity" during a gaze shift by using a schematic representation of gaze motor error on the SC motor map. The zone of TR(S)N activity (represented by the black hill) moves towards the rostral 'fixation' area during the gaze shift. The movement of activity is based upon the dynamic gaze motor error signal.

Steps 1-4 in Figure 3.16 show a hypothetical addition to what might be happening at the SC motor map level during the passive rotation of the VMCS protocol. It is suggested that a hill of neural activity remains on the rostral 'fixation' area
Figure 3.16 A hypothetical representation of dynamic gaze motor saccade signal on SC motor map, during the VMCS protocol. Zones of TR(S)N activity are represented by the shaded hills. The activity in the rostral "fixation" area represents fixation of the LED and is maintained throughout the turn (steps 1-4). The movement of an area of activity away from the rostral zone in steps 1-4 represents the increasing amplitude of the intended gaze saccade as the turn progresses. This "movement of activity" is ultimately based upon interpretation of the stimulus vestibular signal. Steps 5-7 show dynamic gaze motor error readout during the course of the gaze shift. This "movement of activity" is based upon a dynamic gaze motor error signal as the subject makes the saccade. The time course of steps 1-4 is greater than steps 5-7. The hills are tilted in the diagram merely to represent direction of activity movement. Adapted from Guitton (1991).
throughout the turn, because of LED fixation. Concurrently, a hill of neural activity moves away caudally from the rostral 'fixation' area during the turn. The movement of this second hill of activity is once again based on dynamic "gaze motor error", which we can rename dynamic "intended saccade amplitude" for the VMCS task. This intended saccade amplitude is ultimately based upon interpretation of the stimulus vestibular signal. At the end of the turn (steps 4-5), the LED extinguishes and the rostral 'fixation' activity disappears. The gaze shift commences and proceeds as described above (steps 5-7).

We now apply this hypothesis to the subjective impressions during the VMCS task. It is possible that the subjective urge to make the VMCS before the end of the turn could be related to the "balance" between the two hills of activity represented in steps 1-4 of Figure 3.16. The rostral hill represents LED fixation and the caudally moving hill represents growing amplitude of the VMCS. There is growing evidence of reciprocal inhibition between rostral and caudal SC areas (Istvan et al. 1995). Furthermore, this urge seems to be related to the dynamic property of the task, since Bloomberg (1989) showed that up to an 8 minute delay between LED extinguishment and making a VMCS, while sitting immobile in the dark, did not prove difficult. It is commonly known that static sensations habituate much quicker than dynamic sensations(e.g. tactile sensation). Perhaps it is inherent in the dynamic nature of the "intended saccade" activity to be stronger than the static "fixation" activity, unless consciously suppressed.

The second subjective observation was that knowing that the VMCS would be head free alleviated some of this "saccadic urge". The conjectured explanation is that the further the neural activity strays from the rostral fixation zone, the more the intended gaze saccade will depend on a head motor component. Thus with a consciously
intended pure oculomotor saccade, there might be an impetus to trigger the saccade when neural activity leaves "oculomotor boundaries" on the SC motor map.

3.4.2 Variable-Velocity VMCS Protocol

This protocol aimed to answer several questions additional to that of the main study. For example, how linear is the vestibular perceptual system response? Does post-test visual error feedback affect the results? How accurately do subjective visual reports of gaze position compare with objective estimates of gaze position derived from recorded eye and head movements?

Linearity

Since the vestibular system receives a velocity modulated input signal it was desirable to examine the linearity of response as a function of this variable rather than that of duration. The results plotted in Figures 3.13 - 3.15 do in fact demonstrate strongly linear trends over the range of the experiment. This linearity of response as a function of stimulus velocity, concurs with the linearity of response as a function of stimulus duration, found in the variable-duration VMCS experiments discussed above. These similar results confirm, as expected, that the linearity of response when varying stimulus duration is not due to non-vestibular duration cues, but rather reflects a linearity of vestibular perception.

Post-Test Visual Error Feedback

In the variable-duration VMCS experiments, there was a possibility that subjects might use post-test visual information to improve subsequent performance, even though they could not predict the magnitude of a given rotational stimulus. As described under Methods, this potential was obviated in the present series by rotating the concentric drum in an unpredictable way during experiments. In view of the
comparable overall results of Figures 3.9 and 3.15, it seems that this potential factor
did not materially influence the overall variable-duration VMCS results, and was not
the determining force behind the unreduced stabilization gains. However, a question
arises as to whether a lack of error feedback was somehow related to the individual
stimulus direction dependent asymmetries (see below under Asymmetry).

Subjective and Objective Gaze Recording

Figures 3.10 and 3.11 compare results obtained from the subjective visual
method of verbal recording (Figures 3.1 and 3.3 - 3.5) and simultaneous objective
recording of eye (EOG) and head (Watson angular velocity transducer) movements
(Fig. 3.6). It is gratifying to see how well correlated the two data sets are in the plots.
The finding recommends this novel subjective method as a simple, inexpensive one
which might prove useful in some clinical settings, where suitable patient cooperation
can be obtained.

Inspection of recorded eye and head movements during trials (e.g. Figure 3.6)
showed consistent patterns of gaze saccades amongst subjects using the VMCS
paradigm. The profiles showed initiation of a post-rotational saccadic eye movement
roughly at the same time and in the same direction as the corresponding head saccadic
movement. Then, at the moment when gaze arrived at the intended target, the VOR
was switched in at unity gain, holding gaze on target during the remainder of the head
movement. Such gaze saccade profiles are similar to previously described saccades
made to visual (Barnes 1979) and non-visual (Guitton and Volle 1987) targets.

Accuracy of Perception

From Figure 3.15 it is seen that the experimental data are significantly raised
(p<0.05) from theoretical results assuming a 24 second time constant of decay.
Actually, the overall VMCS gain for the population (1.13) is increased (p<0.05)
slightly from an ideal gain of 1 as is apparent from the upwards "offset" of the
regression fit in the figure. It is not clear why perceptual estimates of the stimulus rotation would be augmented. Recall that one subject in the variable-duration VMCS protocol wished to make saccades out of his oculomotor range and so could not complete the full protocol. However, in the case of the whole population, the mean gain of 1.13 is raised minimally from ideal.

**Range Effect**

Looking at the slope (0.761) of the regression line in Figure 3.15, it seems the range effect is again apparent. The regression line (solid line) is tilted clockwise relative to an ideal slope of 1 (dashed line) as would be expected if the range effect is functioning. Recall that in the case of the variable-velocity VMCS protocol, all stimuli were of the same duration. Thus the larger turns presumably would not be associated with a proportionately more decayed brainstem signal relative to the smaller turns, as they would be for the variable-duration VMCS protocol. It follows that in this case a decreased perceptual gain for larger turns cannot be explained by an exponentially decaying sensory velocity signal. In this light, it also seems appropriate to assume that the range effect largely can account for the less than ideal slope (0.815) in Figure 3.9 of the variable-duration VMCS protocol.

**Asymmetry**

An interesting result became apparent in the variable-velocity VMCS protocol that was not present in the variable-duration VMCS protocol: a stimulus direction dependent asymmetry of VMCS gain emerged for each subject. This asymmetry was not an "error" in the protocol that biased all the subjects' results in one direction, as the directions and amplitudes of the asymmetries were idiosyncratic to the subjects. Figure 3.13 and Table 3.6b show the differences. It was also hypothesized that the asymmetries may have been brought about by the imposed motor task of having the head deviated during the trial, necessitated by the protocol. It seems this hypothesis can
be rejected as elaborated in Discussion of the gaze stabilization experiments (cf. 4.4). On the other hand it is possible that the stimulus direction-dependent asymmetries might have become manifest because of the lack of post-test error feedback. Perhaps visual cues in the surrounding environment that normally lead to a full awareness of spatial position, also function to "reset to zero" any directional bias that normally might exist in a subjects' impression of straight ahead. This possibility opens up another avenue of investigation, which is not addressed in this study.

3.4.4 General VMCS discussion

Habituation

The question arises whether the repeated rotational stimuli of these experiments could lead to classical habituation of response. However this possibility would seem unlikely in view of the brief durations of rotational stimuli employed compared with a relatively long habituation time constant on the order of 80 sec (Malcolm and Melvill Jones 1970).

Adaptive Plasticity

A routine manoeuvre in the main experiments was visual VOR suppression during the brief rotational stimuli. Given time, this would likely induce lasting adaptive attenuation of both the vestibulo-ocular (reviewed in Melvill Jones 1985, Melvill Jones and Berthoz 1985) and vestibular perceptual (Bloomberg et al. 1991b) responses. However, this potential effect would also seem unlikely to have interfered with the present results, on account of the short durations of stimulation employed compared with time constants on the order of hours and days associated with the adaptive phenomenon (Gonshor and Melvill Jones 1976a, Gonshor and Melvill Jones
1976b). Indeed, overall results show augmentation, rather than attenuation, relative to the presumed vestibular signal in the brainstem (Figures 3.9 and 3.15).

3.5 Summary

The two VMCS studies show that during the prolonged turns of these experiments the vestibular perceptual response did not follow the time constant of exponential decay to be expected in the brainstem. Rather this response remained close to, or even above, the ideal value of unity gain. However there was a tendency for the vestibular perceptual response gain to decrease with increasing stimulus amplitude in both sets of experiments. But this result can be explained readily by the range effect phenomenon discussed above.
Part 4.

Measuring Vestibular Perception using Gaze Stabilization
4.1 Introduction

As stated in the Introduction for the VMCS experiments (cf. 3.1), the gain of the VOR can be modulated by what the subject is trying to "look at" (i.e. the mental set) during a passive rotation in the dark (Barr et al. 1976, Baloh, Lyerly et al. 1984, Melvill Jones et al. 1984, McKinley and Peterson 1985, Fadlallah 1995). Segal and Katsarkas (1988a) reported that inappropriate VOR-associated slow-phase eye movements tended to be supplemented by goal directed saccadic eye movements during a rotation in which the task was to maintain visual fixation on an unseen earth-fixed target in the dark. These supplementing saccades brought gaze, which they now defined as net gaze, towards and/or on to target. Thus they introduced the concept of net gaze stabilization, which was equal to slow-phase + saccadic eye movements, during this goal-oriented task.

Segal and Katsarkas (1988b) also found that saccades tended to supplement deficient slow-phase eye movements, thereby improving gaze stabilization in patients with unilateral loss of peripheral vestibular function. Bloomberg et al. (1991a and 1991b) continuing along this line of thinking used a VOR plastic adaptation paradigm which had long periods of "forced" VOR suppression, to examine the relationship between VMCS and net gaze stabilization. They found that along with a reduced slow-phase component of gaze stabilization that was dependent on the adaptation paradigm, the VMCS gain was also diminished. They interpreted these results as implying a reduced vestibular perception. Furthermore, VMCS gain was consistently indistinguishable from net gaze stabilization gain, before and after plastic adaptation paradigms. Israel et al. (1993) later showed that this uniform relationship between VMCS and net gaze stabilization gains held over different stimulus amplitudes and axes of rotation. Thus if it is assumed that the VMCS protocol accurately estimates vestibular
perception, it is reasonable to extrapolate that the same may be said for net gaze stabilization.

The following experiments were conducted with the Segal and Katsarkas concept of net stabilization in mind: It was speculated that during a prolonged turn, the slow-phase component of gaze stabilization might be characterized by dependency on an exponentially decaying velocity signal. In turn, this inadequate slow-phase might be "corrected for" by saccadic eye movements, based on a proper vestibular perception of the stimulus. In the Bloomberg et al. experiments (1991a), the reduced slow-phase was "artificially" produced with adaptation paradigms; in the Segal and Katsarkas experiments (1988b), the slow-phase reduction was the result of a pathology; in the present experiments, it is the prolonged nature of the testing stimulus that attempts to manifest a diminished slow-phase. Possibly, all of the above scenarios might invoke common motor mechanisms that employ correcting saccades to help reduce gaze error.

Thus the gaze stabilization experiments were performed after the VMCS experiments, to investigate the sequence of continuous eye movements throughout a prolonged gaze stabilization task. Since the VMCS experiments implied veridical perception during prolonged turns, it was hypothesized that the gaze stabilization experiments would show similar results. Along with this general approach, the possible consequences of having the neck deviated relative to torso during a turn were also examined. It was hypothesized that such a neck deviation might have caused the directional asymmetries seen in the variable-velocity VMCS experiments. The following sections elaborate the methods used, the ensuing results, and their interpretations.
4.2 Methods

4.2.0 General setup and procedure

a. Equipment
The equipment setup was identical to that employed during the VMCS experiments (cf. 3.2.0 a. Equipment) except that the LED and attached arm were not present for these runs.

b. Data acquisition and recording
In this protocol, only computer-acquired objective recordings were employed, as described for the variable-velocity VMCS protocol (cf. 3.2.0 b. Data acquisition and recording). Thus in these experiments, the visual reference tape of Figure 3.1 was not used to estimate gaze position at the end of a trial.

c. Training procedure
Subjects were trained to stabilize the head relative to torso/chair, as well as to center the head on certain points on the reference tape as described for the VMCS experiments (cf. 3.2.0 c. Training procedure). Subjects were then coached through the experimental procedure until comfortable with the protocol.

d. General protocol
The VMCS protocols described previously, measured perception of rotation with a task that suppressed the VOR. The gaze stabilization protocol did the same without suppressing the VOR. Once again, gaze motor output was the index used to measure vestibular perception after a passive rotation in the dark. However in this case, the nature of online gaze motor output during the goal oriented task was studied as well. This gaze stabilization protocol was based on protocols previously used in different labs (e.g. Gauthier and Robinson 1975, Segal and Katsarkas 1988a and 1988b, Bloomberg et al. 1991a). The protocol employed passive rotational velocity...
steps from and back to zero velocity in the dark, while the subject (S) maintained gaze fixation on the remembered position of a just-seen earth fixed target. An important difference between this set of gaze stabilization experiments and previous ones is the fact that the earth-fixed target was not re-illuminated at the end of the trial. Previously S was able to refixate the target in the light; but here, such post-test error feedback was avoided by keeping the lights off. As was done during the variable-velocity VMCS protocol, this error feedback was denied to prevent cognitive modification of the gaze stabilization net gain (Fadlallah 1995). The stimulus parameters used, were identical to some of those employed during the variable-velocity VMCS protocol of the VMCS experiments (cf. 3.2.2). There were 3 protocols in this experiment.

All 14 subjects, aged 21-68 years, participated in the first two protocols which employed long duration stimuli; 12 subjects participated in the third protocol which employed short duration stimuli. Five of the subjects also participated in the variable-velocity VMCS protocol of the VMCS experiments, 2 of these 5 participated in the variable-duration VMCS protocol, and another 1 of the 14 only participated in the variable-duration VMCS protocol. All subjects were secured in the chair as described for the variable-duration VMCS protocol, with the chair's axis of rotation aligned with the mid interocular axis of the subject (cf. 3.2.0 a. Equipment and 3.2.1).

Calibration trials occurred at the beginning of the experiment, and after every 10 experimental trials. Along with calibrations, any spontaneous nystagmus was characterized by asking the subject to stabilize the head and then "look forward" in total darkness. The EOG calibration trial was then conducted in the light: S looked back and forth between points 25° to the left and right of the target on the experimenter's command. During this calibration trial, the head was stabilized and centered on the target, and the chair/torsio was also centered on the target. Thus pure oculomotor saccades across the midline were made with a peak to peak amplitude of 50°. Finally, a
calibration trial similar to that employed during the variable-velocity VMCS protocol was conducted (cf. end of 3.2.2): S was swung back and forth through a 50° peak to peak amplitude rotation while fixating the target in the light and with head stabilized relative to torso.

4.2.1 Protocols

a. Long duration, with head straight re torso

The subject began the trial with the torso (chair) and head centered 25° to the left or right of the target and with the head stabilized relative to torso. Eyes were on target and thus deviated 25° relative to the head and torso. S was told to maintain fixation of the target. Within 1 second of this command but at an exact time unknown to S, the lights were extinguished and the chair was rotated in the same direction as eye deviation. As always the rotation was a step velocity change at start and stop. The stimulus angular velocity of 7.5°/sec lasted 6 sec, yielding a rotational amplitude of 45°. At stop, the lights were not turned on, but the subject continued to maintain fixation of the unseen (imagined) target in the dark for another 6 sec in order to record any post-rotational eye movements. Then the subject was advised that the trial was over and underwent an identical stimulus rotation, but in the opposite direction, all in the dark. This counter-rotation was performed, as in the VMCS experiments, to counteract any post-rotational error built up by continuous unidirectional displacement of the cupula membrane. The lights were turned on and again there was at least a 60 sec immobile period between trials. Five trials were conducted in one direction, then 5 in the other.

b. Long duration, with head deviated re torso

This protocol was identical to the previous one, except that the chair/torso began centered 75° relative to the target; the head was centered only 25° relative to the target
(in the same direction); and the eyes were on target. Thus the head was deviated 50° relative to the torso, and the eyes were deviated 25° relative to the head.

c. Short duration head straight re torso

This protocol was identical to the first protocol, except that the stimulus was a 40 °/sec rotation lasting 1.1 sec resulting in a rotational amplitude of 44°.

4.2.2 Data analysis

Data were manipulated and analyzed using programs specially written for this study with the NEXUS software language (Hunter and Kearney 1984). All recorded channels from an individual trial were displayed on a graphic terminal to characterize the variables described below. As in the VMCS experiments (cf. section 3.2.3 b), values for stimulus amplitude were determined from the integrated head velocity signal. Net gaze stabilization amplitudes were calculated similarly to the gaze saccadic amplitude of the VMCS experiments. "Slow"-phase gaze stabilization amplitudes were assessed as described below. Refer to Figure 4.1 which shows a typical 1.1 sec turn (top ) and a typical 6 sec turn (bottom ) for subject Q. Eye position, integrated head velocity (i.e. head position) and gaze (eye + head) position signals are shown. The shaded bars on the bottom of the figures show when the drum lights were extinguished. The horizontal solid line represents target position and hence both the starting position of gaze and its ideal location throughout the trial. The stimulus amplitude was estimated as the difference between initial and final head angle. The net gaze stabilization amplitude was estimated as the difference between initial eye angle and that which occurred 1.5 sec after stopping the rotation (* in figure). This point corresponds to the time when the drum lights came on in the VMCS experiments, but recall that in the present experiments the lights remained off.
Figure 4.1 Gaze stabilization trials for short and long duration stimuli. A 1.1 sec turn at 40 °/sec (top) and 6 sec turn at 7.5 °/sec (bottom) are shown for subject Q. Head re space, eye re head, and gaze (eye + head) re space position signals are shown. The solid line represents target position and thus the starting position of gaze and ideally where it should have remained throughout the trial. The trace marked "Slow-phase gaze" is drawn by hand and shows gaze position minus identified saccades. The shaded bars at the bottom of the figures show when the lights were extinguished. The asterisk shows the point in time when stabilization gains were calculated, and corresponds to 1.5 sec after stopping the rotation.
The gaze trace was used to mark saccadic eye movements. If there was a rapid shift in gaze position, the beginning and end of this shift was marked and the corresponding points on the eye trace were deemed the beginning and end of the saccadic eye movement. Differentiation between quick-phase and saccadic eye movements (Leigh and Zee 1991) was not made; thus direction of the rapid movement could be in the same direction as, or opposite to, the head - it was still identified as "saccadic". This definition of a saccade has been used previously in similar studies (e.g. Segal and Katsarkas 1988a). Whether a gaze shift was rapid enough in nature was judged by the experimenter, the approximate criterion being at least a 20 °/sec slope of gaze position. This method is consistent with previous saccade marking techniques (Leigh and Zee 1991). However, if there was any doubt at all in the nature of the gaze shift, it was not marked as saccadic. Thus any error of judgment resulted in a saccadic eye movement being included in the slow-phase eye movement category, since for this study "slow-phase" movements simply represent all eye movements excluding those deemed saccadic. Note that this definition of slow-phase differs from the traditional definition, and actually necessarily includes traditional slow-phase eye movements as well as other "non saccadic" eye movements. The conservative method of identifying saccadic eye movements described above was adopted as a precautionary measure. That is, since there were only two categories of eye movements defined, one did not want to artificially reduce the slow-phase component because of saccade marking error. And since most "non-saccadic but still rapid" eye movements included as "slow-phase" were in the same direction as the slow-phase, the most probable outcome was a marginal boost in the slow-phase gain values. Consequently in the Results section, values of slow-phase gain for a couple of subjects are actually higher than the traditional upper range of slow-phase gain. However, note that the net stabilization value which is made
up of slow-phase plus saccadic components, would not be altered by slight variances in the partitioning of eye movements into these two categories.

The trace marked "slow-phase gaze" in Figure 4.1 is drawn by hand, and approximates where the subject's gaze or direction of regard would be without the saccadic gaze shifts. When looking at the gaze and slow-phase gaze traces on the bottom figure, one can see a gaze shift at the end of the turn that was not judged rapid enough to be included in the saccadic category, and hence ended up in the slow-phase category. Net stabilization gain and slow-phase stabilization gain were calculated as the ratio of change in eye position to change in stimulus head position; for example, the slow-phase gain would be the net gaze stabilization amplitude, minus the total saccadic eye movement amplitude, all divided by the stimulus amplitude. The change in eye position vectors were reversed before dividing by change in head position, so that a net or slow-phase stabilization gain below unity implied undershooting, and above unity implied overshooting, with unity representing ideal stabilization of the eye on target.

A general purpose simulation package (MATLAB, SIMULINK3) was used to predict brainstem level velocity signals, assuming such signal behaviour can be modeled by a first order high pass characteristic with 8, 16, and 24 sec time constants. The stimulus head signals from several trials for 3 differently behaving subjects were placed into these models of an exponentially decaying velocity signal. Theoretical values of gaze stabilization gains if following a decaying velocity signal were estimated from these models and compared to actual experimental values for the same trials. As well, MATLAB generated step velocity stimulus signals were passed through the same models for comparison to the results from the whole population.

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3The MathWorks Inc.
4.3 Results

In Figure 4.1 described above, for the 1.1 sec turn (top), the net and slow-phase gains were 0.93 and 0.78 respectively. For the 6 sec turn (bottom), net gain was not reduced at 1.0 but the slow-phase gain of 0.63 was reduced.

Figure 4.2 on the left plots slow-phase gain against the two stimulus turn durations of 1.1 and 6 sec. It shows the population results as subject means for the 12 of the 14 subjects who participated in protocols at both stimulus durations. Note that the trials for the two protocols which employed a 6 sec stimulus duration have been pooled into one mean for each subject. The dashed line at the gain value of 1.0 shows where points would lie if slow-phase eye movements alone perfectly compensated for the stimulus head movement. The solid lines simply connect the means for the two stimulus durations for each subject. All but one of the subjects showed a decrease in gain with increase of stimulus duration. As shown in Table 4.2b, 8 of the individual decreases proved statistically significant, as did the overall trend ($P < 0.01$).

The right hand figure shows similar plots of net gain for the same 12 subjects. The main feature here is that in contrast to the slow-phase data, the population means did not differ significantly from one another, whilst only 5 subjects showed significant gain reduction at the longer duration (Table 4.2b). Table 4.1 shows the means and standard deviations of stabilization gains for the three different protocols.

Figure 4.3 compares individual subject mean gains obtained from the slow-phase and net data at the longer stimulus duration of 6 sec in which all 14 subjects participated. Ten out of the 14 showed higher net than slow-phase gains (Table 4.1). This relationship is further explored for three subjects in the following figures.

Figure 4.4a shows all the data obtained from one subject, M, following the method of Segal and Katsarkas (1988a). Saccadic gain is plotted against slow-phase
Figure 4.2 Slow-phase and Net Stabilization Gains Vs. Stimulus Duration. Slow-phase (left) and net (right) stabilization average gains are plotted against the two stimulus durations of 1.1 and 6 sec, for the 12 subjects who participated in trials at both stimulus durations. Note that trials for the two protocols with a 6 sec stimulus have been pooled into one mean per subject. The dashed line at a gain of 1.0 shows where points would lie with ideal stabilization. 8 subjects showed a significant decrease in slow-phase gain with increased stimulus duration, while only 5 showed a significant decrease in net gain (Table 4.2b).
<table>
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<td>0.930 (0.193)</td>
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<td>0.751 (0.265)</td>
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<td>1.06 (0.0903)</td>
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<td>P</td>
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<td>0.962 (0.0859)</td>
<td>0.959 (0.201)</td>
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<td>0.692 (0.0893)</td>
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<td>G</td>
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<td>1.21 (0.207)</td>
<td>0.775 (0.130)</td>
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</table>

Table 4.1 Gaze Stabilization Gain Means: Gaze stabilization means (and standard deviations) for both net stabilization and slow-phase stabilization gain. "All" signifies population means (and standard errors of the mean). The methods for calculating both these values are explained in the main text. 6 sec stimuli had a stimulus velocity of 7.5 °/sec and 1.1 sec stimuli, 40 °/sec. Note that subjects E and G did not participate in the short duration stimulus protocol.
Figure 4.3 Slow-phase and Net Stabilization Gains for the 6 sec Stimuli. This figure compares subject mean gains for all 14 subjects who participated in the 6 sec stimulus trials. 10 of the 14 showed higher net than slow-phase gains (Table 4.1).
Figure 4.4 Saccadic Gain Vs. Slow-phase Gain for Three Subjects. Figures 4.4a, 4.4b, and 4.4c show all gaze stabilization trials for subjects M, B, and R respectively. Symbols for the different protocols are shown to the left. Note that subject B did not participate in the short duration protocol. The dashed lines (marked ideal in 4.4b) represent the equation $y = (1 - x)$ and signify the whole potential range of compensatory saccadic and slow-phase combinations that yield a unity net gain. Note that for all three subjects, there is a trend for points to straddle the ideal dashed line. This method of plotting the results facilitates identification of the idiosyncratic asymmetries for each subject. See text for details.
gain for each experimental trial. Thus the dashed line marked ideal, \( y = (1 - x) \), which intersects unity gain on both axes, represents the whole potential range of compensatory saccadic and slow-phase combinations which could theoretically yield unity net gain. Points lying above and to the right of the line represent a net gain above 1 (i.e. overshooting), while those below and to the left represent undershooting. The black and white symbols denote clockwise (cw) and counterclockwise (ccw) rotational stimuli respectively. The circles and squares indicate 6 sec trials with head-straight and head-deviated respectively. Triangles indicate 1.1 sec trials.

The first noteworthy feature in this figure is that all trials cluster around the dashed line marked ideal. The short duration trials (triangles) are near a slow-phase gain of unity to the right of the figure; minimal saccadic gain contribution is implied by the fact that most of the triangles do not stray far above a zero saccadic gain. On the other hand, results from the long duration trials (squares and circles) having a reduced slow-phase gain tend to the left, but are moved up along the dashed line by an appropriate saccadic eye contribution, for retention of net unity gain.

Another interesting feature in this figure is the emergence of a direction dependent asymmetry of response, manifest as a predominant distribution of open symbols (ccw) up and to the left along the dashed line marked ideal. Thus ccw turns tended to produce lower slow gains than cw turns, a characteristic which is most evident in the longer duration trials of this subject (squares and circles in the figure, and Table 4.2d). Nevertheless, as already mentioned, this reduction of slow-phase gain tends to be appropriately compensated by an augmented saccadic component. Significantly the asymmetry does not appear to be linked to the introduction of head deviation; nor indeed are there any other obvious features dependent on head deviation.
Figure 4.4b similarly plots results for subject E, who did not participate in the short (1.1 sec) duration protocol. The decrease in slow-phase gain is not as extensive as it was for subject M; however, when the slow-phase gain is reduced, the saccadic gain increases, again compensating with a net gain nearer the ideal dashed line. In this case, the subject's asymmetry is strongly manifest in the net gain, with cw (black) trials being concentrated above the ideal dashed line and the ccw (white) trials below. However, a slight slow-phase gain asymmetry is still evident with the ccw turns having a marginally augmented slow-phase gain relative to the cw turns. Interestingly (Table 4.2d), the direction of the asymmetry is different for net and slow-phase gains. The reason becomes clear when looking at the figure: The saccadic compensation for the reduced slow-phase gain of the cw turns is greater than for that of the ccw turns. Once again, there are no obvious features dependent on head deviation.

Figure 4.4c similarly plots the data for subject R. Here, symbols associated with each direction tend to cluster around the ideal dashed line, showing compensation for a reduced slow-phase gain with an increased saccadic gain. This subject also had an asymmetry in slow-phase gain as well as net gain (Table 4.2d).

Figure 4.5 shows population averages for both net (top of white bar) and slow-phase (top of hatched bar) gains, plotted for both stimulus durations. In each set, the white bars represent the saccadic gain contribution. Standard error bars showing variation of net and slow-phase gain are included. Note the significant (p<0.01) decline of slow-phase gain with the longer duration stimulus, but notably no corresponding decline of net gain, the latter being not reduced and indistinguishable from the ideal value of unity gain for the short and long duration turns respectively (p>0.05). Particularly interesting is the fact that the net stabilization gains for both stimulus durations are significantly higher (p<0.05) than the expected results of a hypothetical subject whose perception follows a decaying velocity signal with a decay
Figure 4.5 Population Means for Net and Slow-phase Gains at Both Stimulus Durations. Slow-phase and net gains, with standard errors, are plotted together at the two different stimulus durations. Saccadic contributions to net gains are represented by the (white) differences between slow-phase (top of hatched bars) and net (top of white bars) gains. There is a significant (p < 0.01) decline of slow-phase gain with stimulus duration but no corresponding decline of net gain. In fact, net gain is not reduced, and indistinguishable from unity gain at the 1.1 and 6 sec stimuli respectively.
time constant of 24 sec. However, the slow-phase gain means for both stimulus durations are not significantly different from such theoretical "vestibular signal decay" results. Both these latter conclusions were arrived at by comparing results to step angular velocity inputs fed through a simple 1st order Matlab model.

Table 4.2a shows the results of ANOVAs done with either net gaze stabilization or slow-phase gaze stabilization gain as the dependent variable, by subject and for the whole population. The independent variables are Duration of stimulus, Head deviation, Direction of stimulus, and in the case of the whole population, Subject. Note that for the whole population, the slow-phase stabilization gains are shown to be significantly dependent on the duration of the stimulus, whether or not Subject is included as an independent variable. However, net stabilization gain is no longer significantly affected by stimulus duration, when the independent variable, Subject, is omitted (see top of Table 4.2a). The results that emerge from this ANOVA model, in terms of each independent variable, are further explored in the following tables.

Table 4.2b shows net and slow-phase gaze stabilization least squares means for stimulus duration, using the ANOVAs presented in Table 4.2a and without Subject as an independent variable. These means are the expected marginal means had the experimental design been balanced for stimulus duration. Also included are significance levels for t tests testing the hypothesis that pairs of least squares means are equal. Note that for the whole population, not only is the slow-phase gain least squares mean reduced by 0.164 with increased stimulus duration as compared to 0.053 for the net gain, but only the slow-phase component is significantly reduced with the increase in stimulus duration. When looking at the 12 subjects who underwent trials at both stimulus durations (E and G did not participate in short duration trials), 8 subjects had significantly reduced slow-phase gains with an increase in stimulus duration, while
Table 4.2a Gaze Stabilization ANOVAs of Net Stabilization and Slow-phase Stabilization: The results of ANOVAs done of net stabilization as well as slow-phase stabilization gains by subject and for the whole population, done for the gaze stabilization experiments. The dependent variable is either net stabilization or slow-phase stabilization gain. The independent variables are as follows: Duration of stimulus - either 6 sec or 1.1 sec (corresponding to 7.5 and 40°/sec stimuli respectively); Deviation of the head - either yes or no; Direction of stimulus - clockwise or counterclockwise; and Subject with 14 levels. Note that for the population results ("all"), when Subject is not included as an independent variable, the net stabilization gain is no longer significantly different for the two stimulus durations although it still is for the slow-phase gain. This result corresponds to the t test results showing slow-phase gain is significantly different for the two stimulus durations while the same is not true for net stabilization gain.
<table>
<thead>
<tr>
<th>Subject</th>
<th>LS mean 1.1 sec</th>
<th>LS mean 6 sec</th>
<th>t test</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>1.05</td>
<td>0.997</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>slow 0.948</td>
<td>0.784</td>
<td>**</td>
</tr>
<tr>
<td>M</td>
<td>1.00</td>
<td>0.972</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>slow 0.836</td>
<td>0.619</td>
<td>**</td>
</tr>
<tr>
<td>N</td>
<td>0.962</td>
<td>0.906</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>slow 0.977</td>
<td>0.849</td>
<td>**</td>
</tr>
<tr>
<td>O</td>
<td>1.48</td>
<td>1.75</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>slow 1.16</td>
<td>1.08</td>
<td>-</td>
</tr>
<tr>
<td>J</td>
<td>1.16</td>
<td>0.650</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 1.08</td>
<td>0.719</td>
<td>**</td>
</tr>
<tr>
<td>P</td>
<td>1.15</td>
<td>0.914</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 1.09</td>
<td>0.981</td>
<td>*</td>
</tr>
<tr>
<td>Q</td>
<td>0.784</td>
<td>1.00</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 0.761</td>
<td>0.717</td>
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<td>E</td>
<td>1.02</td>
<td>na</td>
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<td>slow 0.725</td>
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<td>na</td>
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<td>F</td>
<td>1.07</td>
<td>0.777</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 1.03</td>
<td>0.698</td>
<td>**</td>
</tr>
<tr>
<td>R</td>
<td>0.974</td>
<td>0.933</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>slow 0.785</td>
<td>0.745</td>
<td>-</td>
</tr>
<tr>
<td>G</td>
<td>1.24</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td></td>
<td>slow 0.822</td>
<td>na</td>
<td>na</td>
</tr>
<tr>
<td>K</td>
<td>1.02</td>
<td>0.959</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>slow 0.880</td>
<td>0.758</td>
<td>*</td>
</tr>
<tr>
<td>T</td>
<td>1.19</td>
<td>0.623</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 1.09</td>
<td>0.678</td>
<td>**</td>
</tr>
<tr>
<td>U</td>
<td>0.904</td>
<td>1.13</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 0.816</td>
<td>0.880</td>
<td>-</td>
</tr>
<tr>
<td>L</td>
<td>1.05</td>
<td>0.785</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 1.03</td>
<td>0.705</td>
<td>**</td>
</tr>
</tbody>
</table>

Table 4.2b Gaze Stabilization ANOVA Least Squares Means for Stimulus Duration: The Least squares means, i.e. marginal means that would be expected had the experimental design been balanced for the independent variable, stimulus duration. The ANOVA model used was the one in Table 4.2a above (not including Subject as an independent variable). The t test tests the hypothesis of whether the two LS means are equal.
only 5 of the 12 had significantly reduced net gains, although 8 did have significantly different net gains for the two stimulus durations.

Table 4.2c is similar to Table 4.2b except that the marginal means represent values had the experimental design been balanced for head deviation. Although head deviation does seem to cause a significant difference in net and slow-phase stabilization gain in a substantial proportion of the subjects, that functional difference is not consistent from subject to subject. Furthermore, there is no difference for the population as a whole. Table 4.3, discussed below, further explores head deviation's relation to directional asymmetries of subjects.

Table 4.2d is once again similar to Tables 4.2b and 4.2c, except that the marginal means represent values had the design been balanced for stimulus direction. Although the population as a whole has no directional asymmetry, 12 of the 14 subjects have a significant difference in directional gains for net or slow-phase stabilization (or both), idiosyncratic to each subject. Subjects who also participated in the variable-velocity VMCS experiments have their results compared to their gaze stabilization results, in terms of directional asymmetries, in Table 4.4, to be discussed below.

Table 4.3 shows results from ANOVAs similar to those discussed in Table 4.2a. However, another independent variable has been added: the interaction between direction of stimulus and head deviation. The head straight and head deviated protocols with the 6 sec stimulus duration were originally done with the intention of seeing whether head deviation was causing/correlated with the stimulus direction dependent asymmetries in stabilization gain present in the variable-velocity VMCS experiments. As can be seen from the table, there was no consistent trend of stimulus direction dependent variance coinciding with a significant interaction with head deviation; furthermore, only 5 subjects showed any significant interaction at all, independent of whether it be net or slow-phase gain. Of the 5 subjects that also participated in the
### Table 4.2c Gaze Stabilization ANOVA Least Squares Means for Head Deviation

The Least squares means, i.e. marginal means that would be expected had the experimental design been balanced for the independent variable, head deviation. The ANOVA model used was the one in Table 4.2a above (not including Subject as an independent variable). The t test tests the hypothesis of whether the two LS means are equal.

<table>
<thead>
<tr>
<th>Subject</th>
<th>LS mean head straight</th>
<th>LS mean head deviated</th>
<th>t test</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td>1.01</td>
<td>1.04</td>
<td>-</td>
</tr>
<tr>
<td>slow</td>
<td>0.854</td>
<td>0.878</td>
<td>-</td>
</tr>
<tr>
<td>M</td>
<td>1.03</td>
<td>0.944</td>
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<td>slow</td>
<td>0.778</td>
<td>0.677</td>
<td>-</td>
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<tr>
<td>N</td>
<td>0.896</td>
<td>0.972</td>
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<tr>
<td>slow</td>
<td>0.874</td>
<td>0.952</td>
<td>-</td>
</tr>
<tr>
<td>O</td>
<td>1.49</td>
<td>1.73</td>
<td>-</td>
</tr>
<tr>
<td>slow</td>
<td>0.998</td>
<td>1.24</td>
<td>*</td>
</tr>
<tr>
<td>J</td>
<td>0.802</td>
<td>1.00</td>
<td>*</td>
</tr>
<tr>
<td>slow</td>
<td>0.783</td>
<td>1.00</td>
<td>**</td>
</tr>
<tr>
<td>P</td>
<td>0.986</td>
<td>1.08</td>
<td>*</td>
</tr>
<tr>
<td>slow</td>
<td>1.02</td>
<td>1.06</td>
<td>-</td>
</tr>
<tr>
<td>Q</td>
<td>0.990</td>
<td>0.795</td>
<td>**</td>
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<tr>
<td>slow</td>
<td>0.792</td>
<td>0.686</td>
<td>-</td>
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<td>E</td>
<td>0.992</td>
<td>1.05</td>
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<tr>
<td>slow</td>
<td>0.710</td>
<td>0.739</td>
<td>-</td>
</tr>
<tr>
<td>F</td>
<td>0.823</td>
<td>1.02</td>
<td>**</td>
</tr>
<tr>
<td>slow</td>
<td>0.785</td>
<td>0.939</td>
<td>*</td>
</tr>
<tr>
<td>R</td>
<td>0.985</td>
<td>0.921</td>
<td>-</td>
</tr>
<tr>
<td>slow</td>
<td>0.818</td>
<td>0.712</td>
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</tr>
<tr>
<td>G</td>
<td>1.26</td>
<td>1.21</td>
<td>-</td>
</tr>
<tr>
<td>slow</td>
<td>0.869</td>
<td>0.775</td>
<td>*</td>
</tr>
<tr>
<td>K</td>
<td>1.01</td>
<td>0.963</td>
<td>-</td>
</tr>
<tr>
<td>slow</td>
<td>0.855</td>
<td>0.783</td>
<td>-</td>
</tr>
<tr>
<td>T</td>
<td>0.772</td>
<td>1.04</td>
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<tr>
<td>slow</td>
<td>0.753</td>
<td>1.01</td>
<td>**</td>
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<td>U</td>
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<td>0.841</td>
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<tr>
<td>slow</td>
<td>0.924</td>
<td>0.772</td>
<td>*</td>
</tr>
<tr>
<td>L</td>
<td>0.822</td>
<td>1.01</td>
<td>*</td>
</tr>
<tr>
<td>slow</td>
<td>0.806</td>
<td>0.931</td>
<td>*</td>
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</tbody>
</table>
The table below presents the Least Squares Means for stimulus direction, along with the associated t tests. The ANOVA model used did not include Subject as an independent variable.

<table>
<thead>
<tr>
<th>Subject</th>
<th>LS mean counterclockwise stimulus</th>
<th>LS mean clockwise stimulus</th>
<th>t test</th>
</tr>
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<tbody>
<tr>
<td>All</td>
<td>net 1.01</td>
<td>1.04</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>slow 0.869</td>
<td>0.863</td>
<td>-</td>
</tr>
<tr>
<td>M</td>
<td>net 0.973</td>
<td>1.00</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>slow 0.587</td>
<td>0.868</td>
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</tr>
<tr>
<td>N</td>
<td>net 1.08</td>
<td>0.791</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 0.961</td>
<td>0.865</td>
<td>*</td>
</tr>
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<td>O</td>
<td>net 1.63</td>
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<tr>
<td></td>
<td>slow 1.12</td>
<td>1.11</td>
<td>-</td>
</tr>
<tr>
<td>J</td>
<td>net 0.801</td>
<td>1.00</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 0.831</td>
<td>0.956</td>
<td>*</td>
</tr>
<tr>
<td>P</td>
<td>net 1.16</td>
<td>0.903</td>
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</tr>
<tr>
<td></td>
<td>slow 1.10</td>
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<td>Q</td>
<td>net 0.986</td>
<td>0.800</td>
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</tr>
<tr>
<td></td>
<td>slow 0.764</td>
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<td></td>
<td>slow 0.792</td>
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<td>F</td>
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<td>slow 0.940</td>
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<tr>
<td>R</td>
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<td>1.05</td>
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</tr>
<tr>
<td></td>
<td>slow 0.718</td>
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<tr>
<td>G</td>
<td>net 1.17</td>
<td>1.30</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>slow 0.802</td>
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<td>-</td>
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<tr>
<td>K</td>
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<td>1.07</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 0.804</td>
<td>0.834</td>
<td>-</td>
</tr>
<tr>
<td>T</td>
<td>net 0.824</td>
<td>0.992</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>slow 0.818</td>
<td>0.949</td>
<td>*</td>
</tr>
<tr>
<td>U</td>
<td>net 0.988</td>
<td>1.05</td>
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<tr>
<td></td>
<td>slow 0.864</td>
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<td>L</td>
<td>net 0.890</td>
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</tr>
<tr>
<td></td>
<td>slow 0.944</td>
<td>0.794</td>
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</table>

Table 4.2d Gaze Stabilization ANOVA Least Squares Means for Stimulus Direction: The Least squares means, i.e. marginal means that would be expected had the experimental design been balanced for the independent variable, stimulus direction. The ANOVA model used was the one in Table 4.2a above (not including Subject as an independent variable). The t test tests the hypothesis of whether the two LS means are equal.
Table 4.3 Gaze Stabilization ANOVAs of Net Stabilization and Slow-phase Stabilization Including Interactions: The results of ANOVAs done of net stabilization as well as slow-phase stabilization gains by subject and for the whole population, done for the gaze stabilization experiments. The dependent variable is either net stabilization or slow-phase stabilization gain. The independent variables are as in Table 4.2a as well as an independent variable comprising the interaction between direction of stimulus and head deviation, shown in the last column.
variable-velocity VMCS experiments, only 2 (J and L) showed a significant interaction of head deviation and stimulus direction on net gain. However, consideration of Table 4.4 shows that it is only subject L of these two who is consistent in the direction of his asymmetry between the two experiment.

Table 4.4 shows directional VMCS net stabilization means, by subject, from all of the variable-velocity VMCS experiment and from part of the gaze stabilization experiment. All trials in the variable-velocity VMCS experiment had a 6 sec stimulus duration and the head deviated throughout the stimulus rotation. Directional net stabilization means from the 6 sec head deviated trial of the gaze stabilization experiments are also shown, for these same 5 subjects. Only subjects E and L had a consistent direction of asymmetry between the two experiments.

The results from three subjects, who had idiosyncratic asymmetrical gain manifestations, were further analyzed to assess whether any asymmetries in stimulus profiles of head movement might have caused such asymmetries. Subjects M, Q, and E had asymmetries in the slow-phase gain, net gain, and both slow-phase and net gain respectively (Table 4.2d). Several head velocity stimulus profiles from clockwise and counterclockwise trials for these subjects were chosen at random from each protocol and used as the inputs to a high-pass filter model having a first order exponential decay with a time constant of 24 sec. This model was an approximation of brainstem level behaviour. In the 16 trials that were studied, no asymmetry was seen in the simulated responses, and thus the profiles could not account for the asymmetries that still persisted in the actual gaze stabilization values for these same trials.

Table 4.5 shows mean net stabilization gains for subjects who participated in all three experimental protocols (two VMCS and one gaze stabilization), for those trials having a 6 sec stimulus duration. The two variable-duration VMCS means are lower than their other experimental counterparts, possibly owing to the manifestation of the
<table>
<thead>
<tr>
<th>Subject and experimental protocol</th>
<th>Counterclockwise stimulus net stabilization</th>
<th>Clockwise stimulus net stabilization</th>
</tr>
</thead>
<tbody>
<tr>
<td>J Variable-velocity VMCS</td>
<td>1.03 (0.1510)</td>
<td>0.830 (0.154)</td>
</tr>
<tr>
<td>J Gaze stabilization</td>
<td>0.564 (0.144)</td>
<td>0.938 (0.224)</td>
</tr>
<tr>
<td>E Variable-velocity VMCS</td>
<td>0.966 (0.1390)</td>
<td>1.20 (0.211)</td>
</tr>
<tr>
<td>E Gaze stabilization</td>
<td>0.961 (0.112)</td>
<td>1.14 (0.123)</td>
</tr>
<tr>
<td>G Variable-velocity VMCS</td>
<td>1.34 (0.166)</td>
<td>1.19 (0.127)</td>
</tr>
<tr>
<td>G Gaze stabilization</td>
<td>1.14 (0.200)</td>
<td>1.29 (0.195)</td>
</tr>
<tr>
<td>K Variable-velocity VMCS</td>
<td>1.73 (0.310)</td>
<td>1.24 (0.141)</td>
</tr>
<tr>
<td>K Gaze stabilization</td>
<td>0.838 (0.0599)</td>
<td>1.03 (0.0775)</td>
</tr>
<tr>
<td>L Variable-velocity VMCS</td>
<td>0.669 (0.149)</td>
<td>1.13 (0.222)</td>
</tr>
<tr>
<td>L Gaze stabilization</td>
<td>0.760 (0.145)</td>
<td>1.00 (0.156)</td>
</tr>
</tbody>
</table>

Table 4.4 Comparable Variable-Velocity VMCS & Gaze Stabilization Subject Means of Gains by Stimulus Direction: VMCS and net stabilization gain means (and standard deviations) by direction for overlapping subjects for all Variable-velocity VMCS trials (all with a 6 sec stimulus) and the comparable 6 sec stimulus trials for Gaze stabilization that also had head deviated. Note only subjects E and L had consistent directional asymmetries between the two experiments.
<table>
<thead>
<tr>
<th>Subject</th>
<th>Variable-duration VMCS net stabilization</th>
<th>Variable-velocity VMCS net stabilization</th>
<th>Gaze stabilization net stabilization</th>
</tr>
</thead>
<tbody>
<tr>
<td>J</td>
<td>na</td>
<td>0.930 (0.181)</td>
<td>0.660 (0.240)</td>
</tr>
<tr>
<td>E</td>
<td>1.02 (0.0683)</td>
<td>1.08 (0.213)</td>
<td>1.04 (0.141)</td>
</tr>
<tr>
<td>G</td>
<td>0.992 (0.184)</td>
<td>1.27 (0.165)</td>
<td>1.24 (0.196)</td>
</tr>
<tr>
<td>K</td>
<td>na</td>
<td>1.49 (0.344)</td>
<td>0.959 (0.142)</td>
</tr>
<tr>
<td>L</td>
<td>na</td>
<td>0.898 (0.297)</td>
<td>0.785 (0.201)</td>
</tr>
</tbody>
</table>

Table 4.5 Variable-Duration VMCS, Variable-Velocity VMCS, and Gaze Stabilization Net Stabilization Gain Means for 6 sec Stimuli for Overlapping Subjects: VMCS and net stabilization gain means (and standard deviations) from all experiments, comparing 6 sec stimuli.
range effect in the variable-duration VMCS 6 sec stimuli, as a reduction of the corresponding means. Of the five subjects that took part in the other two experiments, only two subjects (J and K) do not seem to have consistent means. No solid statistical conclusions can be drawn across the three experiments based on these sparse data.

4.4 Discussion

The previous experiments of Part 3 used Vestibular Memory Contingent Saccades (VMCS) to investigate the perceptual accuracy of vestibular memory after completion of a prolonged turn. The present Gaze Stabilization study serves the complementary role of addressing the question: "What is the vestibular perceptual response during a prolonged turn?" The transfer function of neural response associated with rotational stimulation of the semicircular canals leads to exponential signal decay during prolonged turns, as is explained under "Introduction and background" (cf. 2.1 and appendix A). Does conscious perception of this response follow the brainstem signal? Or, given a consciously chosen goal, can perceptual processes correct for the inherent deficiencies of the sensory response throughout the turn?

Accuracy of perception

The main outcome for the subject population of the study is exemplified in Fig 4.5. Whereas the slow-phase component of the compensatory VOR decayed according to expectation, the net response did not. In fact, net gain proved not to be reduced from the ideal value of unity at both short (1.1 sec) and long (6.0 sec) durations.

In the studies of Bloomberg et al. (1991b) referred to earlier (cf. 3.1 and 4.1), the net response of gaze stabilization was found to be indistinguishable from the corresponding VMCS response. Since the latter was dependent solely on the consciously perceived memory of a preceding vestibular signal, it was inferred that net gaze stabilization also reflected the percept of that signal available while the subject was
in the process of turning. Consequently it is now inferred from the present results that a veridical percept was available to subjects during turns which were long enough to invoke significant decay of the corresponding brainstem signal, at least over the 6 sec duration of these experiments.

Saccadic and slow-phase eye movements

Although some individual subjects did have reduced net gains, there was a consistent trend to compensate for inadequate slow-phase stabilization with saccades (Figures 4.3 and 4.4). Slow-phase stabilization gains for the population were reduced with the longer duration rotations as judged by ANOVAs, t tests, and figures. Furthermore, these gains were not significantly different from expected results for a 24 sec time constant of decay. Although some subjects did not have greatly reduced slow-phase gains, they usually had an appropriately small saccadic gaze stabilization component. Thus, in general, the concept of saccadic eye movements correcting for inadequate slow-phase eye movements holds.

The proposed perceptual lookup table

The problem formulation of "Introduction and background" (cf. 2.2) referred to a possible "learned lookup table" based on past experience that provides correct vestibular perception during prolonged turns. An example of the kind of neural mechanisms necessary for such velocity-error correction is already available at the brainstem level, referred to as "velocity storage". The models of velocity storage have positive (Robinson 1977) or negative (Raphen et al. 1979) feedback that function to increase the vestibular peripheral time constant of decay, two or threefold. It is reasonable to assume that a proposed lookup table in a higher center than the brainstem could also function the same way, with a gain adjustment that extended the time constant indefinitely.
In the present experiments, this proposed lookup table seems to be relying on the saccadic system as its main gaze motor output. Perhaps a learned vestibular lookup table exists in the posterior parietal cortex and is the source of spatial accuracy for saccades, based on the following evidence: 1) The possible role of the posterior parietal cortex in vestibular perception has been gaining support (Bloomberg 1989, Tropper et al. 1991, Israel et al. 1992); 2) function of the posterior parietal cortex appears to be linked to sensorimotor integration, that is, motor programming based on sensory guidance (Anderson 1987); 3) and the lateral intraparietal area within has been implicated in saccadic eye movements (Gnadt and Anderson 1988, reviewed in Anderson and Gnadt 1989). Furthermore, the frontal eye field (FEF) is primarily concerned with saccadic motor targeting and also its triggering (reviewed in Goldberg and Segraves 1989). So perhaps information from the proposed lookup table in the parietal cortex could reach the superior colliculus (SC) saccade motor map via known projections to the FEF (reviewed in Goldberg and Segraves 1989 and in Anderson and Gnadt 1989) and through more direct pathways (reviewed in Anderson and Gnadt 1989).

Conjectured neural activity at the superior colliculus

It has been previously suggested that vestibular perception projects onto the saccadic motor map of the SC (Melvill Jones 1992). With this idea in mind, here is another conjectured progression of neural activity on the SC motor map, based on the Guitton and Munoz model of dynamic gaze motor error. Referring back to Figure 3.16 in Part 3, we can imagine the dynamic progression of neural activity outlined in steps 1-7 occurring several times throughout a gaze stabilization turn. The only necessary change to the figure is removal of the rostral "hill" of activity representing LED fixation in steps 2-4, since there was no such fixation in this protocol. The rostral activity remains in step 1 at initiation of the turn because it represents accurate gaze on the
(unseen) target. Then steps 2-4 (without the rostral 'hill') represent build up of gaze motor error as the turn progresses and slow-phase eye movements fail to properly stabilize gaze on the perceived (unseen) target. This gaze error information ultimately would be derived from the proposed vestibular lookup table, possibly in the posterior parietal lobe. Initiation and progression of the saccade "correcting" gaze is represented by steps 5-7, possibly triggered through the FEF. We are then back to step 1 with zero gaze error and the cycle progresses again as the turn continues and gaze error builds up again. It would be interesting to investigate the actual neural activity on the SC motor map of properly trained animals performing the tasks of both the VMCS and gaze stabilization protocols described above.

Neck deviation and individual directional gain asymmetries

Although stimulus direction dependent asymmetries in both net and slow-phase stabilization gains were again present, no correlation could be made to neck deviation. Furthermore, this asymmetry was only consistent in one subject overlapping with the variable-velocity VMCS experiments. The origin of these asymmetries could not be determined from these experiments. Possibly the asymmetry could be associated with the lack of post-test error feedback at the end of a trial. In both the gaze stabilization, and variable-velocity VMCS experiments, in which idiosyncratic directional asymmetries become noticeable, subjects did not receive reinforcement from seeing correct target position at the end of the trial. Possibly such a lack of error feedback could allow a directional bias to build up.
Summary and Overall Conclusions
A prolonged turn produces a velocity coded vestibular sensory signal that decays exponentially with time. And yet during everyday life, this situation does not seem to interfere with our perception of movement. Accordingly, the basic question asked by this study was "How accurate is vestibular perception during naturally occurring prolonged turns?" It was decided a natural goal-directed motor task was necessary to measure perception. Two sets of experiments were carried out, the vestibular memory contingent saccade (VMCS) protocols and the gaze stabilization protocol. Both required a gaze motor output that was based solely on a vestibular sensory input. The VMCS experiments required a subject to fixate the remembered position of an earth-fixed target after a passive rotation in the dark, and the gaze stabilization experiment required the same during a passive rotation in the dark.

**VMCS experiments**

**VMCS with Variable-duration stimuli**

From this protocol, it is concluded that perception was not decreased but rather was virtually ideal with a superimposed range effect. It is also concluded that having the eyes deviated at the beginning of a trial did not affect the results significantly. Furthermore, when head and eye combined saccades were used as the required motor output, they were more accurate than eye-only saccades, perhaps due to their being a more natural way of producing saccadic gaze shifts.

**VMCS with Variable-velocity stimuli**

Along with the general question of this study, the following questions were also asked by this protocol: How linear is the relationship between vestibular perception and the magnitude of the velocity stimulus? Does post-test error feedback affect the results? Are visual reports of gaze related to actual eye-head movements?
The conclusions are the following: Perception was not decreased and was close to ideal with a range effect present, even with the removal of post-test error feedback; the perceptual response to velocity stimuli was linear over the range tested; substantial individual directional asymmetries of perceptual response were observed; and subjective visual and objective eye-head position estimates of gaze position were well correlated.

**Gaze stabilization experiment**

Vestibular perceptual response was measured during the passive head turn. Also, the following relationships were investigated: saccadic and slow-phase eye movements; neck deviation relative to torso and individual directional asymmetries.

It is found that net stabilization, and thus presumably perception was not decreased during long turns compared to short turns. This perceptual response was close to ideal since, although slow-phase stabilization was decreased as expected during long turns, saccades consistently tended to compensate for the slow-phase underperformance. Also, whether or not the neck was deviated during a turn was uncorrelated with the individual directional asymmetries. The origin of these asymmetries remains unknown.

**Conclusions**

Veridical vestibular perception was available for controlling goal-directed gaze motor output during the prolonged turns of this study. Possibly the source of this proper spatial percept is a learned lookup table based on past sensory and motor experience.
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Appendix A: Canal frequency response relationship

The dimensions and characteristics of the semicircular canal, endolymph fluid, and components within have been extensively studied (Steinhausen 1933, Van Egmond et al. 1949; reviewed in Wilson and Melvill Jones 1979). During an angular acceleration applied to the whole system, there are three significant moments in balance:

An inertial moment of the endolymph fluid equal (but opposite) to the combination of a viscous moment of the fluid plus an elastic moment of the cupula membrane. If we assume the whole canal system undergoes an angular acceleration \( q'' \) in time \( t \), this situation is well characterized by the equation:

\[
I \cdot p'' = B \cdot \theta' + K \cdot \theta 
\]  
(A.1)

where \( p'' \) is the absolute angular acceleration of fluid relative to space in time \( t \); \( \theta \) and \( \theta' \) are respectively the relative angular displacement and velocity of endolymph fluid with respect to the canal walls, in time \( t \); \( I \) is the moment of inertia of endolymph fluid contained within the canal; \( B \) is the moment of viscous friction per relative angular velocity (\( \theta' \)) of fluid; and \( K \) is the cupula elastic restoring force per relative angular displacement (\( \theta \)) of fluid.

If we take \( q, q', \) and \( q'' \) to be the angular position, velocity, and acceleration of the canal in its own plane, and \( p \) to be the absolute angle of fluid displacement relative to space, then

\[
p = q - \theta
\]  
(A.2)

Therefore, assuming linearity,
\[ p'' = q'' - \theta'' \]  

(A.3)

Where \( \theta'' \) is the relative angular acceleration of fluid with respect to the canal walls.

Substituting in equation (A.1) for \( p'' \)

\[ I \cdot (q'' - \theta'') = B \cdot \theta' + K \cdot \theta \]  

(A.4)

which rearranges to

\[ q'' = \theta'' + \frac{(B/I) \cdot \theta'}{1} + \frac{(K/I) \cdot \theta}{1} \]  

(A.5)

Analysis of the above differential equation is simplified by converting from the time domain to the Laplace frequency domain using the corresponding Laplace transfer function

\[ q''(s) = \theta(s) \cdot (s^2 + \frac{B}{I} \cdot s + \frac{K}{I}) \]  

(A.6)

where \( q''(s) \) and \( \theta(s) \) represent the Laplace transforms of \( q''(t) \) and \( \theta(t) \). Rearranging again

\[ \frac{\theta}{q''}(s) = \frac{1}{s^2 + \frac{B}{I} \cdot s + \frac{K}{I}} \]  

(A.7)

and multiplying both numerator and denominator on the right side of the equation by \( I/K \)
\[(\theta / q')(s) = (I/K) \cdot \frac{1}{[(I/K) \cdot s^2 + (B/K) \cdot s + 1]} \] (A.8)

Now factorizing the right hand side denominator (within the square brackets)

\[(\theta / q')(s) = (I/K) \cdot \frac{1}{[(T_1 \cdot s + 1) \cdot (T_2 \cdot s + 1)]} \] (A.9)

where \(T_1 \cdot T_2 = I/K\) and \(T_1 + T_2 = B/K\). The value of \(T_1\), the "short" time constant, can be shown to be equal to \(I/B\), by classical hydrodynamic analysis. This value has been calculated to be about 3 msec on the basis of experimental data determining \(I\), the inertial moment and \(B\), the viscous moment of the fluid within the semicircular canals (Wilson and Melvill Jones 1979). \(T_1\) represents the time constant of approach to an equilibrium of forces due to inertia and viscosity, and at a value of 3 msec is too brief to influence the present studies. Van Egmond et al. (1949) first showed that the value of \(T_2\) was in the order of seconds rather than milliseconds. Thus the value \(T_1 + T_2 = B/K\) closely approximates \(T_2\) alone. So we may then approximate

\[T_2 = B/K \] (A.10)

which allows us to approximate

\[T_1 \cdot T_2 = T_1 \cdot (B/K) = I/K\]

and so again \(T_1\) emerges as

\[T_1 = I/B \] (A.11)
Equation (A.9) relates fluid displacement ($\theta$), and hence cupula displacement, to angular acceleration ($q''$), the input to the system. To get the relationship between fluid displacement and instantaneous angular velocity, we may substitute $q' \cdot s = q''$ into equation (A.9) to yield

$$\frac{\theta}{q'}(s) = \frac{(I/K) \cdot s}{[(T_1 \cdot s + 1)(T_2 \cdot s + 1)]}$$  \hspace{1cm} (A.12)

Furthermore, since $I/K = T_1 \cdot T_2$, we may substitute into equation (A.12) to obtain

$$\frac{\theta}{q'}(s) = \frac{[T_1 \cdot T_2 \cdot s]}{[(T_1 \cdot s + 1)(T_2 \cdot s + 1)]}$$  \hspace{1cm} (A.13)

Figure A.1 reproduced from Wilson and Melvill Jones (1979) gives a plausible Bode plot of the frequency response of this system over a range of sinusoidal stimulus frequencies, 0.01 to 10.0 Hz. The forcing function has been expressed in terms of angular velocity ($q'$). The extrapolated dashed lines intersect one another at the asymptotic cut-off frequencies

$$\omega_1 = 1/T_1 = B/I \text{ and } \omega_2 = 1/T_2 = K/B$$  \hspace{1cm} (A.14)

where $\omega$ represents frequency in radians/sec ($\omega = 2\pi \cdot \text{Hz}$). The gain is expressed in terms of fluid (or cupula) displacement in relation to instantaneous angular velocity. In this figure, a 0.003 sec value for $T_1$ has been used to yield the upper cut-off frequency

$$\omega_1 = 1/0.003 \text{ sec} = 333 \text{ rad/sec}$$
Figure A.1 Bode plot of the frequency response of equation (A.13), representing the dynamic response of a 2nd order model of the semicircular canal. \( \theta \) is equal to endolymph or cupula angular displacement relative to the canal. Both gain and phase are expressed with respect to head angular velocity \( (q') \). \( T_1 = I/B \). \( T_2 = B/K \). (Reproduced from Wilson and Melvill Jones 1979).
which is about 50 Hz. For convenience, a value of 10 sec (Van Egmond et al. 1949) has been used for $T_2$ yielding

$$\omega_2 = 1/10 \text{ sec} = 0.1 \text{ rad/sec}$$

approximating the lower cut-off frequency with which this study is concerned. 0.1 rad/sec is 0.016 Hz.

The flat middle portion of the figure, in which velocity gain remains at unity and phase shift is close to zero, corresponds to the frequency range in which cupula response functions as a closely veridical instantaneous velocity transducer. From the Bode plot of Figure A.1 it is interesting to note the following: Cupula position at very low frequencies veridically transduces angular acceleration of the system, and cupula position at very high frequencies veridically transduces angular position of the system, with shifting gains in between. This finding is also implied by the 90' phase shifts of response at very low and very high frequencies. The natural head stimuli of this study enter into the lower frequency range where such a shift begins to occur.
Appendix B: Additional studies

It should be mentioned that two other separate experimental protocols were performed as part of this study but not included in this thesis. The first was an investigation of vergence eye movements during prolonged turns using a VMCS protocol, with targets at different distances from the subject. Separate eye movements were measured using the magnetic search coil technique with equipment in the Clinical Neurosciences Department at the University of Calgary.

The second experiment was the implementation of a VMCS protocol on a large sample of patients complaining of dizziness at the Otolaryngology Department of the Montreal Jewish General Hospital. The goal of this clinical study was to find a correlation between VMCS gain, other vestibular function tests, and an examining doctor's assessment of the patient.