Effects of Viewing Distance on the Responses of Horizontal Canal–Related Secondary Vestibular Neurons During Angular Head Rotation

CHIJU CHEN-HUANG AND ROBERT A. McCREA
Department of Neurobiology, Pharmacology and Physiology, University of Chicago, Chicago, Illinois 60637

Chen-Huang, Chiju and Robert A. McCrea. Effects of viewing distance on the responses of horizontal canal–related secondary vestibular neurons during angular head rotation. J. Neurophysiol. 81: 2517-2537, 1999. The eye movements generated by the horizontal canal–related angular vestibuloocular reflex (AVOR) depend on the distance of the image from the head and the axis of head rotation. The effects of viewing distance on the responses of 105 horizontal canal–related central vestibular neurons were examined in two squirrel monkeys that were trained to fixate small, earth-stationary targets at different distances (10 and 150 cm) from their eyes. The majority of these cells (77/105) were identified as secondary vestibular neurons by synaptic activation following electrical stimulation of the vestibular nerve. All of the viewing distance–sensitive units were also sensitive to eye movements in the absence of head movements. Some classes of eye movement–related vestibular units were more sensitive to viewing distance than others. For example, the average increase in rotational gain (discharge rate/head velocity) of position-vestibular-pause units was 20%, whereas the gain increase of eye-head-velocity units was 44%. The concomitant change in gain of the AVOR was 11%. Near viewing responses of units phase lagged the responses they generated during far target viewing by 6–25°. A similar phase lag was not observed in either the near AVOR eye movements or in the firing behavior of burst-position units in the vestibular nuclei whose firing behavior was only related to eye movements. The viewing distance–related increase in the evoked eye movements and in the rotational gain of all unit classes declined progressively as stimulus frequency increased from 0.7 to 4.0 Hz. When monkeys canceled their VOR by fixating head-stationary targets, the responses recorded during near and far target viewing were comparable. However, the viewing distance–related response changes exhibited by central units were not directly attributable to the eye movement signals they generated. Subtraction of static eye position signals reduced, but did not abolish viewing distance gain changes in most units. Smooth pursuit eye velocity sensitivity and viewing distance sensitivity were not well correlated. We conclude that the central premotor pathways that mediate the AVOR also mediate viewing distance–related changes in the reflex. Because irregular vestibular nerve afferents are necessary for viewing distance–related gain changes in the AVOR, we suggest that a central premotor pathway that mediates the AVOR also mediates viewing distance–related changes in the reflex.

INTRODUCTION

The vestibuloocular reflex (VOR) functions to stabilize images on the retina during head movements by rotating the eyes. The angular vestibuloocular reflex (AVOR) is evoked by angular rotations of the head that stimulate the vestibular semicircular canals. The eye movements evoked by the AVOR vary inversely as a function of viewing distance (Chen-Huang and McCrea 1998b; Snyder and King 1992; Viirre et al. 1986). Several observations suggest that the increase in AVOR eye movement gain during near viewing reflects changes in the processing of vestibular signals in brain stem VOR pathways. During near target viewing, sudden step changes in head velocity evoke eye movements with an enhanced gain at latencies that are shorter than even the shortest visual feedback-driven eye movements (Crane and Demer 1997, 1998; Snyder et al. 1992; Viirre et al. 1986). A viewing distance–related increase in AVOR gain has been demonstrated to occur before the convergent eye movements that produce a change in viewing distance (Snyder et al. 1992). This gain increase persists in the absence of a target in complete darkness (Chen-Huang and McCrea 1998b; Hine and Thorn 1987; Snyder et al. 1992). Finally, viewing distance gain increases in the AVOR are significantly reduced when irregular vestibular afferents are silenced (Chen-Huang and McCrea 1998b). These observations have led to the suggestion that some, or possibly all of the central vestibular signals that drive the VOR are multiplied by an internal estimate of vergence angle or viewing distance (Chen-Huang and McCrea 1998b; Paige and Tomko 1991; Viirre et al. 1986). If the changes in the AVOR are due to parametric changes in central pathways that mediate the reflex, it is not clear how or where these changes occur. Recently, McConville et al. (1996) carried out a single-unit recording study in rhesus monkeys in which the AVOR responses of eye movement–related units were recorded during viewing of near and far targets. They found that some classes of eye movement–related units were more sensitive to viewing distance than others. Specifically, the rotational responses of eye-head-velocity (EHV) units (whose response to vestibular stimulation typically reverses when the VOR is voluntarily canceled or suppressed) were significantly enhanced during near target viewing. On the other hand, the responses of position-vestibular-pause (PVP) units were usually unaffected by viewing distance, once the eye position sensitivity of those cells was taken into account. The rotational responses of units that were not sensitive to eye movements were not affected by viewing distance (Tomlinson et al. 1996). Although based on a relatively small sample of cells, these observations suggest that...
Viewing distance–related changes in the AVOR may be mediated by a specialized subset of the available VOR pathways.

In this study we examined in further detail the viewing distance–related changes in the angular head rotation sensitivity of different classes of horizontal canal–related secondary vestibular neurons. We focused particularly on eye movement–related neurons in the rostral part of the medial vestibular nucleus of the squirrel monkey, because such neurons are likely to be related to the VOR (Cullen et al. 1993; Scudder and Fuchs 1992). We report that the signals generated by most of the horizontal canal–related neurons putatively involved in mediating the VOR change as a function of viewing distance. The changes in sensitivity to head rotation appear to be more than sufficient to produce viewing distance–related changes in the VOR. We hypothesize that viewing distance modifications in the AVOR are produced by modifying the gain of indirect inhibitory vestibular afferent inputs to VOR pathways.

**METHODS**

**Surgical procedures**

Two squirrel monkeys were prepared for chronic recording of bilateral eye movements, single-unit activity and for electrical stimulation of both labyrinths. Surgeries were carried out under sterile conditions on anesthetized animals (pentobarbital sodium, 20 mg/kg, initial dose; supplemental doses of 1–2 mg/kg administered as necessary). A flattened stainless steel bolt, to be used for head restraint in the plane of the horizontal semicircular canal, was attached to the occipital bone with dental acrylic. A Plexiglas cylindrical recording chamber (15 mm diam), was stereotaxically implanted on the surface of the left parietal bone with dental acrylic. A search coil was sutured to the sclera of the right eye, and the twined leads of the coils were soldered to a connector that was cemented to the skull.

On a subsequent day, a second search coil was implanted on the left eye, and bipolar stimulating electrodes were implanted into both labyrinths. One of each pair of labyrinthine stimulating electrodes (chlorided silver wire, 0.25 mm diam and Teflon insulated to within 1 mm of tip) was inserted into the perilymphatic space through a hole in the bony promontory midway between the round and oval windows.
The second electrode was placed in the ventral aspect of the middle ear. Leads from both electrodes were led out of the middle ear near the insertion of the tympanic membrane and soldered to a skull-mounted connector.

**Experimental recording conditions**

During experiments the monkey was seated in a Plexiglas chair on a vestibular turntable. Each animal was trained to fixate two types of visual targets. Rewards were contingent on vergence angle and vertical eye position. In some experimental paradigms (Fig. 1A) the monkey fixated small targets that were projected onto a blank cylindrical screen 90 cm distant from the axis of rotation. In other paradigms the monkey looked through a door in the screen at an earth-stationary far target 1.52 m distant from the axis of rotation (Fig. 1B). All experiments were carried out in a dimly lit room (2 × 4 m in size) in which the monkey was isolated. Squirrel monkeys typically fixated targets for brief periods (0.5–2 min) on demand for periods of ≥6 h or more.

**Turntable rotation and head movement recording**

Passive head rotations in the plane of the horizontal semicircular canals (15° nose down from the stereotaxic plane) were produced with a position servo-control system whose output modified the command of a velocity servo turntable controller (Inland model 832). The axis of turntable rotation, which was parallel to earth-vertical, passed through the squirrel monkey’s midsagittal plane, where it intersects with the line connecting the two external auditory meatuses. The responses of single units were typically recorded during rotations at several different frequencies and velocities of table rotation (most commonly, 0.7 Hz, peak velocity 20–40°/s; 1.9 Hz, 10–20°/s and 4.0 Hz, 6–10°/s). Each stimulus frequency produced a slightly higher peak head acceleration (88, 119, and 150°/s² for the 0.7-, 1.9-, and 4.0-Hz stimuli, respectively) and a significant decrease in head displacement (4.55, 0.84, and 0.24° for the 0.7-, 1.9-, and 4.0-Hz stimuli, respectively). Angular table velocity was recorded with an angular velocity transducer (Watson Industries).

**Eye movement recording**

Eye movements were measured with a magnetic search-coil system (40 cm diam, Neurodata Instruments) that was mounted on a movably superstructure on top of the vestibular turntable. The system was linearly related to eye position throughout the monkey’s oculomotor range of ±20°. The root-mean-square (rms) noise level of the system was equivalent to ±0.2° (0–7 kHz). The gain of each eye coil was calibrated independently by assuming that the AVOR gain recorded in the light in response to a 1.9-Hz, 10°/s sinusoidal head rotation had a
Values are means ± SE. The numbers of each type of unit recorded and the fraction that received monosynaptic inputs from the ipsilateral vestibular nerve when tested (mono/tested) are shown in the left-hand columns. The mean static eye position sensitivity ($K_e$) and the mean gain and phase of unit responses re contralateral eye velocity during 0.7-Hz smooth pursuit eye movements are indicated in the middle columns. The columns on the right-hand side of the table list the mean gain and phase of unit responses re ipsilateral head velocity during 0.7-Hz VOR cancellation (VOR Gain, Phase) and during fixation of an earth-stationary far target (AVOR Gain, Phase). VOR, vestibuloocular reflex; AVOR, angular VOR; BP, burst position; VI, type I vestibular only; PVP, position-vestibular-pause; EHVII and EHVI, type II and type I eye-head-velocity, respectively; PV, position vestibular; EVII, eye movement–related type II; N/A, not applicable.

gain of unity. Zero horizontal eye position for each eye was defined as the position of the eye during fixation of a target 8 mm lateral to the midline (half of the interpupillary distance) when the other eye was patched. These calibrations were checked periodically by recording smooth pursuit eye movements evoked by a 0.7-Hz, 20°/s moving target. The vergence angle was obtained from the difference between right and left eye positions. Negative values indicate convergence.

Visual target characteristics and behavior control

Smooth pursuit eye movements were evoked with a laser projected onto a cylindrical screen 90 cm from the monkey with a pair of mirrors attached to position controlled galvanometers. Two retractable ceiling-mounted earth-stationary visual targets were used to study viewing distance–related changes in the VOR. Each target was a red light-emitting diode (LED) affixed to the tip of a motorized telescoping rod attached to the ceiling. The far and near LEDs were 5 mm (0.2°) and 1 mm (0.5°) in diameter, respectively. The far target was positioned on the midline 152 cm from the monkey. The near target was positioned on the midline 12.25 cm from the interaural plane (10 cm from a plane passing through the tip of the ears). A third head-stationary near target was attached to the monkey chair with a hinge that allowed it to be positioned 10 cm from the eyes. The monkey was intermittently rewarded for fixating a visual target with drops of vanilla-flavored milk. The milk rewards were typically given at a frequency of ~1/s when the horizontal and vertical position of both eyes converged within 1° of the target.

Single-unit recording techniques

The techniques used for obtaining single-unit recordings have been previously described (Chen-Huang et al. 1997; Cullen and McCrea 1993). Briefly, Epoxy-insulated tungsten microelectrodes (4–7 MΩ impedance) were advanced into the cerebellum with a manually operated micromanipulator. A hydraulic microdrive (Trent Wells) was then used to move the microelectrode into the vestibular nuclei. The location of each electrode probe into the brain was determined with respect to a skull-mounted reference point inside the recording chamber and verified by the location of vestibular field potentials evoked after electrical stimulation of the ipsilateral vestibular nerve (0.1-ms monophasic perilymphatic cathodal pulses, 200 μA). Single isolated vestibular unit potentials were conventionally amplified and discriminated. The output pulse of a window discriminator was used to trigger the event channel of a CED 1401 data acquisition system, which stored the time of the spike at a 100-μs resolution (see Data acquisition and data analysis). SYNAPTIC ACTIVATION OF VESTIBULAR UNITS FROM THE VESTIBULAR NERVE. For most of the units encountered, the ipsilateral vestibular nerve was electrically stimulated (0.1-ms pulses,

### Table 1. Eye and head movement sensitivity of different classes of vestibular neurons included in this study

<table>
<thead>
<tr>
<th>Unit Type</th>
<th>$n$</th>
<th>Mono (tested)</th>
<th>$K_e$, spikes/s/deg</th>
<th>Pursuit Gain, spikes/s/deg/s</th>
<th>Pursuit Phase, deg</th>
<th>VOR Gain, spikes/s/deg/s</th>
<th>VOR Phase, deg</th>
<th>AVOR Gain, spikes/s/deg/s</th>
<th>AVOR Phase, deg</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>10</td>
<td>1/4</td>
<td>11.1 ± 2.5</td>
<td>3.40 ± 0.55</td>
<td>136.5 ± 6.3</td>
<td>0.61 ± 0.15</td>
<td>179.2 ± 16.2</td>
<td>3.71 ± 0.62</td>
<td>140.8 ± 9.8</td>
</tr>
<tr>
<td>VI</td>
<td>10</td>
<td>8/8</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>1.08 ± 0.15</td>
<td>13.8 ± 6.4</td>
<td>0.96 ± 0.16</td>
<td>15.5 ± 6.0</td>
</tr>
<tr>
<td>PVP</td>
<td>47</td>
<td>38/40</td>
<td>4.1 ± 0.4</td>
<td>1.52 ± 0.15</td>
<td>−40.2 ± 5.6</td>
<td>0.88 ± 0.09</td>
<td>14.3 ± 3.1</td>
<td>1.71 ± 0.14</td>
<td>−13.0 ± 3.7</td>
</tr>
<tr>
<td>EHVII</td>
<td>12</td>
<td>9/10</td>
<td>1.8 ± 1.0</td>
<td>2.56 ± 0.25</td>
<td>21.6 ± 6.3</td>
<td>0.74 ± 0.09</td>
<td>202.0 ± 4.8</td>
<td>1.87 ± 0.21</td>
<td>25.9 ± 6.6</td>
</tr>
<tr>
<td>EVII</td>
<td>10</td>
<td>7/7</td>
<td>−1.1 ± 0.5</td>
<td>1.04 ± 0.28</td>
<td>174.4 ± 6.1</td>
<td>0.40 ± 0.08</td>
<td>17.5 ± 6.6</td>
<td>0.72 ± 0.17</td>
<td>131.8 ± 14.0</td>
</tr>
<tr>
<td>PV</td>
<td>6</td>
<td>5/6</td>
<td>1.9 ± 0.3</td>
<td>0.48 ± 0.12</td>
<td>−40.9 ± 7.2</td>
<td>0.89 ± 0.38</td>
<td>11.1 ± 11.5</td>
<td>1.38 ± 0.39</td>
<td>−5.5 ± 14.1</td>
</tr>
<tr>
<td>EVII</td>
<td>6</td>
<td>5/5</td>
<td>−1.5 ± 0.96</td>
<td>0.97 ± 0.27</td>
<td>148.8 ± 4.5</td>
<td>0.53 ± 0.13</td>
<td>177.2 ± 17.8</td>
<td>0.81 ± 0.18</td>
<td>146.0 ± 20.1</td>
</tr>
</tbody>
</table>

Mean responses during 0.7- and 1.9-Hz rotations and the ratio between the response obtained during near and far viewing (N/F) is also shown. Two sets of coefficients are listed for each condition. The coefficients in parentheses are corrected for eye position sensitivity (corr). For abbreviations, see Table 1.

### Table 2. Response gain and phase of different classes of secondary vestibular neurons during far ($G_F, P_F$) and near ($G_N, P_N$) target viewing

<table>
<thead>
<tr>
<th>Unit Class</th>
<th>$G_F$</th>
<th>$P_F$</th>
<th>$G_N$</th>
<th>$P_N$</th>
<th>N/F</th>
<th>$G_F$</th>
<th>$P_F$</th>
<th>$G_N$</th>
<th>$P_N$</th>
<th>N/F</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>3.7</td>
<td>141</td>
<td>4.4</td>
<td>134</td>
<td>1.23</td>
<td>2.8</td>
<td>166</td>
<td>3.7</td>
<td>162</td>
<td>1.23</td>
</tr>
<tr>
<td>(corr)</td>
<td>(2.9)</td>
<td>(202)</td>
<td>(3.8)</td>
<td>(202)</td>
<td>(1.26)</td>
<td>(2.8)</td>
<td>(200)</td>
<td>(3.6)</td>
<td>(200)</td>
<td>(1.21)</td>
</tr>
<tr>
<td>PVP</td>
<td>1.7</td>
<td>−13.0</td>
<td>2.0</td>
<td>−18</td>
<td>1.20</td>
<td>1.7</td>
<td>3.7</td>
<td>1.9</td>
<td>−9</td>
<td>1.21</td>
</tr>
<tr>
<td>(corr)</td>
<td>(1.7)</td>
<td>(23)</td>
<td>(1.9)</td>
<td>(16)</td>
<td>(1.11)</td>
<td>(1.8)</td>
<td>(15)</td>
<td>(1.9)</td>
<td>(3)</td>
<td>(1.21)</td>
</tr>
<tr>
<td>EHVII</td>
<td>1.9</td>
<td>26</td>
<td>2.6</td>
<td>−7</td>
<td>1.39</td>
<td>2.4</td>
<td>21</td>
<td>2.9</td>
<td>2</td>
<td>1.23</td>
</tr>
<tr>
<td>(corr)</td>
<td>(2.0)</td>
<td>(33)</td>
<td>(2.5)</td>
<td>(−7)</td>
<td>(1.25)</td>
<td>(2.5)</td>
<td>(24)</td>
<td>(2.8)</td>
<td>(4)</td>
<td>(1.16)</td>
</tr>
<tr>
<td>EVII</td>
<td>0.7</td>
<td>131</td>
<td>1.1</td>
<td>138</td>
<td>1.49</td>
<td>0.9</td>
<td>135</td>
<td>1.4</td>
<td>143</td>
<td>1.64</td>
</tr>
<tr>
<td>(corr)</td>
<td>(0.8)</td>
<td>(144)</td>
<td>(1.1)</td>
<td>(150)</td>
<td>(1.43)</td>
<td>(0.9)</td>
<td>(154)</td>
<td>(1.4)</td>
<td>(156)</td>
<td>(1.87)</td>
</tr>
<tr>
<td>PV</td>
<td>1.4</td>
<td>−6</td>
<td>1.7</td>
<td>2</td>
<td>1.41</td>
<td>1.6</td>
<td>13</td>
<td>1.8</td>
<td>14</td>
<td>1.29</td>
</tr>
<tr>
<td>(corr)</td>
<td>(1.5)</td>
<td>(33)</td>
<td>(1.8)</td>
<td>(27)</td>
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<td>(1.6)</td>
<td>(22)</td>
<td>(1.8)</td>
<td>(20)</td>
<td>(1.28)</td>
</tr>
<tr>
<td>VI</td>
<td>1.0</td>
<td>16</td>
<td>0.8</td>
<td>16</td>
<td>0.93</td>
<td>1.3</td>
<td>24</td>
<td>1.2</td>
<td>31</td>
<td>0.95</td>
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<tr>
<td>EVII</td>
<td>0.8</td>
<td>146</td>
<td>1.0</td>
<td>151</td>
<td>1.39</td>
<td>1.4</td>
<td>173</td>
<td>1.7</td>
<td>175</td>
<td>1.24</td>
</tr>
<tr>
<td>(corr)</td>
<td>(0.9)</td>
<td>(161)</td>
<td>(1.1)</td>
<td>(175)</td>
<td>(1.49)</td>
<td>(1.4)</td>
<td>(181)</td>
<td>(1.7)</td>
<td>(188)</td>
<td>(1.25)</td>
</tr>
</tbody>
</table>
50–300 μA) to determine the presence and latency of synaptically evoked responses. Units whose response latency was <1.4 ms were considered to have monosynaptic inputs from the vestibular nerve. The lack of a response to vestibular nerve stimulation was not considered to be strong negative evidence for the lack of direct inputs from the vestibular nerve. The maximum currents used were not sufficient to activate all of the fibers in the vestibular nerve, and, in a few cases (particularly with those units not tested) the unit potential was too small to recognize in the presence of the vestibular field that was concomitantly evoked by the stimulus.

Most of the cells included in this report were located in the vestibular nuclei, based on the latency and amplitude of the field potentials recorded in the vicinity of each unit. Estimates of the rostral, medial, and lateral borders of the nuclei were based on the amplitude of monosynaptic field potentials recorded following electrical stimulation of the vestibular nerve and the firing behavior of neurons in regions judged to be outside the borders of the vestibular nuclei.

ANTIDROMIC IDENTIFICATION OF ASCENDING TRACT OF DEITERS NEURONS. In one monkey, a concentric bipolar electrode (platinum-irridium; 150 μm OD, 75 μm ID; tip separation 300 μm) was placed ~400 μm dorsal to the left ascending tract of Deiters (ATD) after determining the location of the left medial longitudinal fasciculus at a rostrocaudal level that was 0.5 mm caudal to the caudal pole of the trochlear nucleus. Some of the data from these units have been presented in a preliminary report (Chen-Huang and McCrea 1998a).

**Single-unit recording protocol**

Only isolated units whose firing behavior was related to head rotation in the horizontal semicircular canal plane (either during fixation of an earth-stationary target or during fixation of a head-stationary target) were studied. Once encountered, horizontal semicircular canal–related units were tested for their responses following electrical stimulation of the ipsilateral vestibular nerve and/or the ipsilateral ATD (see ANTIDROMIC IDENTIFICATION OF ASCENDING TRACT OF DEITERS NEURONS). The response of each unit was then recorded in the following paradigms (~30–60 s per paradigm):  
1) AVOR during fixation of a far target (152 cm distant; 0.7 Hz, ±20°/s; 1.9 Hz, ±10°/s; 4.0 Hz, ±6°/s)  
2) AVOR during fixation of a near target (12.25 cm distant; 0.7, 1.9, and 4.0 Hz)  
3) Smooth pursuit of a moving target (90 cm distant; 0.7 Hz, ±20°/s)  
4) Cancellation of the AVOR; head stationary far target (90 cm distant; 0.7 Hz)

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**FIG. 4.** Firing behavior of an ipsilateral on-direction burst-position (BP) unit. Sample records are shown in A1–D1. Averaged, desaccaded responses are illustrated in A2–D2. Calibration and labeling conventions of different traces are the same as in Fig. 3. Note that the background firing rate in D (when the mean position of the contralateral eye was ipsilateral to center position) was slightly higher than in C (when the mean position of both eyes was near the center), which suggests that this unit was primarily related to the position of the contralateral eye.
UNIT STATIC EYE POSITION SENSITIVITY. Static eye position sensitivity was assessed from firing behavior generated during periods (500 ms to 2 s) of spontaneous fixation in the absence of a target. Multiple regression estimates of the sensitivity to horizontal and vertical eye position were computed from at least 20 stable eye positions (>100 ms after a saccade). For units that demonstrated nonlinearity in the rate-position relationship, multiple fits of firing rate were made to subranges of eye position where the relationship appeared to be linear by visual inspection. In some units whose firing rate approached zero during off-direction gaze, eye position coefficients were determined with an iterative fitting technique that used changes in residual variance to estimate the point at which the fit became nonlinear. Static eye position coefficients were used to correct records for eye position–associated signals.

Little effort was made to determine unit sensitivity to monocular eye position. A rough estimate of the ocular dominance of a unit’s eye position sensitivity (McConville et al. 1994) was made in most cells by comparing the discharge rate of cells during far target trials to that during near target trials. This two-point estimation did not allow us to quantify monocular eye position sensitivity or to eliminate the possibility that a vergence position signal contributed to the differences recorded.

ANALYSIS OF UNIT DATA OBTAINED WITH SINUSOIDAL STIMULI. All of the unit data related to smooth pursuit, VOR cancellation, and the VOR were obtained during sinusoidal target or turntable rotations. Typically, unit responses during two or more 30-s duration trials were obtained for each of the behavioral paradigms described above. Only cycles in which the positions of both eyes were within 2° of the target were included in the analysis. In the analysis of records related to the VOR, cycles were rejected if an appropriate vergence angle was not maintained during the cycle. The included cycles were concatenated, desaccaded, averaged, and fit with sinusoidal functions.

The desaccading, or “de-quickphase” algorithm involved a comparison of the estimated slow phase eye velocity to the actual eye velocity recorded from one eye in selected cycles (usually the right eye). When the recorded eye velocity deviated from estimated slow phase velocity by a criterion amount (usually 10–30°/s), the records were considered to be saccades or artifacts, and all of the data within a window around the saccade were eliminated from further analysis. The saccade window was usually 30 ms before the saccade and 40 ms after the end of the saccade. The window was adjusted so that saccade-related bursts and pauses in firing rate were eliminated from records. It was sometimes not feasible to entirely eliminate firing behavior related to postsaccadic “slide,” because its duration could be longer than the mean quick-phase interval. In these cases (particularly EHV omnibusters), the time constant of the saccade slide was estimated, and the saccade window was adjusted to extend at least one time constant after the end of the saccade. The efficacy of the desaccading algorithm was evaluated by careful visual comparison of desaccaded records to the original records.

Desaccaded, selected records were then duplicated. One set of records was averaged and fit with sinusoidal functions whose frequency was the same as the fundamental frequency of the stimulus. An iterative fitting technique was used to eliminate low firing frequency responses that deviated significantly from linearity during periods of inhibitory saturation (Chen-Huang et al. 1997). The second set of records was “corrected” for static eye position sensitivity, using the vertical and horizontal eye position coefficients obtained from the static eye position analysis described above. These “corrected” records were then averaged and fit with sinusoidal functions. Correction for eye position sensitivity was more problematic in units whose firing behavior was not linearly related to eye position; particularly EHV units. The eye position correction analysis of EHV units was...
sometimes complicated by nonlinear eye position firing rate relationship. In these units, two linear functions were used to characterize eye position sensitivity, and the coefficient used for correction at each point in a record was determined for the concurrent eye position.

**Methods for calculating viewing distance–related changes in unit and eye movement responses**

The rotational responses of many vestibular units were significantly different during near target viewing (AVOR_n) than during far target viewing (AVOR_f), but the phase of these responses did not change significantly as a function of viewing distance. It was convenient to define a scalar quantity (ΔGVD) that was an estimate of change in response gain (ΔG) related to viewing distance (VD) of a unit, based on the difference in responses recorded during AVOR_n and AVOR_f. A ratio of a unit’s VOR response gains during AVOR_n and AVOR_f (N/F ratio) was also calculated.

The vergence angles required to fixate the far and near targets were 0.2 and 9.6°, respectively. The ideal gain of the VOR required to maintain image stability during head rotation calculated from the equations described by Viirre et al. (1986) was 1.02 for the far target and 1.22 for the near target. Thus the change in VOR gain required by kinematic considerations was 0.20. In previous behavioral studies we found that the AVOR gain change was inversely related to the frequency of turntable rotation, and even at low stimulus frequencies it had a variable gain that rarely matches the kinematic requirement (Chen-Huang and McCrea 1998b). In the present study, the change in AVOR gain recorded concomitantly with unit recordings varied from 0.0 to 0.25. The mean gain change was 0.17 ± 0.02 (mean ± SE) during 0.7-Hz table rotations, 0.09 ± 0.01 during 1.9-Hz rotations, and 0.03 ± 0.01 during 4.0-Hz rotations (Fig. 1B). These values were
slightly smaller than those recorded in the absence of single-unit recordings. Because the target was located on the midline, very little change in the phase of the eye movements was predicted, or, on average, recorded (Fig. 1C). Variances in both eye movement and unit responses reported in figures, text, and tables are means ± SE.

RESULTS

Location and classification of vestibular units

The effects of viewing distance on the responses of 105 vestibular nucleus neurons to sinusoidal angular head rotation in the plane of the horizontal semicircular canals were recorded in two squirrel monkeys. Every unit encountered was classified on the basis of its firing behavior during 1) sinusoidal rotation while the monkey fixated an earth-stationary LED target, 2) VOR cancellation evoked by fixation of a head-stationary target projected onto a cylindrical screen, and 3) sinusoidal smooth pursuit of a target projected onto the screen. Units whose firing rate was not related to horizontal rotation or horizontal eye movements during pursuit were discarded. Most of the units tested (84/105) were activated at monosynaptic (77/84) or disynaptic (1/84) latencies following electrical stimulation of the ipsilateral vestibular nerve (Fig. 2, A and B).

Most of the units in this study were located in the rostral half of the vestibular nuclei. The estimated location of the tracks in one monkey is illustrated in a diagrammatic horizontal section through the vestibular nuclei in Fig. 2C. The diagram is a dorsal view of the vestibular nuclei constructed in a plane orthogonal to the orientation of electrode tracks. Reconstruction of electrode tracks suggested that neurons in the medial, superior, and lateral vestibular nucleus were included in our sample. The majority of horizontal canal–related secondary vestibular neurons were encountered in the ventral lateral vestibular nucleus and adjacent regions of the rostral medial vestibular nucleus. Four units recorded on one track were included in this study even though the reconstruction suggested they were located in the rostral prepositus nucleus [starred track at rostral end of nucleus prepositus hypoglossi (PH) in Fig. 2C]. These burst-position units were included because the synaptic field potential recorded in that track was large and because adjacent nonhorizontal canal–related units were activated at monosynaptic latencies following stimulation of the vestibular nerve.

Horizontal canal–related vestibular neurons were classified on the basis of their firing behavior during the VOR, VOR cancellation, smooth pursuit eye movements, fixation, and spontaneous saccadic eye movements. The classification scheme and nomenclature used here are similar to that previously described for segregating the responses of primate vestibular neurons (Cullen and McCrea 1993; Miles 1974; Scudder and Fuchs 1992; Tomlinson and Robinson 1984). These categories include the following:

1) Type I vestibular only (VI) units (n = 10)
2) Burst-position (BP) units (n = 10)
3) PVP units (n = 47)
4) EHV units (n = 22)
5) Position-vestibular (PV) units (n = 6)
6) Other eye movement–related vestibular units (n = 10)

All of the VI, PVP, and PV units, and the majority of the EHV units exhibited type I (ipsilateral on direction) rotational responses during fixation of earth-stationary targets. Most eye

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**FIG. 7.** Viewing distance–related changes in the sensitivity of PVP units to head rotation. A: PVP unit near target AVOR gain (AVOR) vs. far target AVOR gain (AVOR). Solid line indicates equal response gains during AVOR and AVOR. Dashed lines indicate near responses that deviated by >2 SE from the average AVOR response. B: distribution of ΔGVD in PVP units. Positive values are increases in rotational gain during near target viewing. C: distribution of N/F response ratio in PVP units (mean = 1.20).
movement–related BP units, some EHV units, and most of the unclassified units exhibited type II responses (see below). The predominance of PVP and EHV units in this sample was due to the fact that a concerted attempt was made to record from regions of the vestibular nuclei that contained horizontal canal–related secondary vestibular neurons that project to the abducens nucleus or the medial rectus subdivision of the oculomotor nucleus. Six EHV units and one PVP unit included in this study were antidromically activated following electrical stimulation of the ascending tract of Deiters. Some of the firing characteristics of these cells have been described in a preliminary communication (Chen-Huang and McCrea 1998a). In the following paragraphs the characteristic firing behavior of each class of units will be described.

Vestibular-only units

The firing behavior of a typical VI unit during smooth pursuit, VOR cancellation and during the VOR evoked while fixating the far target (AVORf) and while fixating the near target (AVORn) is illustrated in Fig. 3. In each paradigm the stimulus frequency was 0.7 Hz and the peak velocity was 20°/s. Figure 3, A1–D1, shows sample records obtained from the cell. Figure 3, A2–D2, shows the averaged, desaccaded responses of the same cell. The polar plots in A3–D3 show the gain and phase of the responses of all of the EHV units tested at 0.7 Hz in each paradigm. Open squares are EHVII units, crosses are EHVI units. A gain of 2 spikes/s/deg/s is indicated by the circle in each plot. Conventions for illustration are the same as Fig. 6.
s/deg/s at 0.7 Hz). Their head velocity sensitivity was slightly higher during VOR cancellation (mean gain = 1.08 ± 0.15 spikes/s/deg/s). VI units were not sensitive to eye position and saccades and were not modulated during sinusoidal smooth pursuit eye movements.

The rotational responses of most VI units were not significantly affected by viewing distance. One VI unit’s response gain decreased from 1.4 spikes/s/deg/s to 1.1 spikes/s/deg/s during near target viewing (22% decrease in gain). This exceptional unit caused the average rotational responses of VI units to be slightly lower during near target viewing (Table 2).

**Burst position units**

Burst-position units were usually encountered in probes through the medial aspect of the rostral medial vestibular nucleus. They are included in this study because most of them were located in the vestibular nucleus and because they presumably could be involved in producing the AVOR. BP units were sensitive to horizontal eye position, usually (9/10) in the ipsilateral direction, and generated bursts of spikes during on-direction saccades. Only the contralateral eye movement–related BP unit could be monosynaptically activated following electrical stimulation of the ipsilateral vestibular nerve. Sample records and averaged responses of an ipsilateral BP unit are illustrated in Fig. 4. The firing characteristics of ipsilateral on direction BP units are summarized in Table 1.

BP units were sensitive to eye position during steady fixation (static eye position sensitivity, $K_s = 2.6–32$; median $K_s = 11.1$ spikes/s/deg; Fig. 5A) and to eye velocity during pursuit. In six units the background firing rate during near and far target viewing was significantly different, which presumably reflected the preference of the units to the position of one eye. The eye position signals appeared to be related primarily to the contralateral eye in three units and to the ipsilateral eye in three other units. In the remaining four units, the difference in background firing rate was too small, or inconsistent, to allow an estimate of ocular dominance. BP units were typically more sensitive to on-direction saccades than to off-direction saccades. The unit illustrated in Fig. 4, A1–D1, for example, generated bursts of spikes during leftward, on-direction saccades and quick phases of nystagmus, but was weakly, and inconsistently inhibited during off-direction saccades.

BP units were strongly modulated during sinusoidal smooth pursuit eye movements (Fig. 4, A1 and A2). Their mean sensitivity to eye velocity was 3.40 ± 0.55 spikes/s/deg/s; $n = 10$). During VOR cancellation (Fig. 4, B1 and B2) the rotational response of these cells was related primarily to the small, unsuppressed eye velocity.

The rotational responses of most (9/10) BP units increased as a function of inverse viewing distance (e.g., $\Delta GVD$ ranged from 0.3 to 1.4 spikes/s/deg/s at 1.9 Hz). One unit exhibited a significant decrease in rotational gain during AVOR$_s$ ($\Delta GVD = -0.5$ spikes/s/deg/s). On average, BP unit sensitivity to head velocity increased from 2.82 sp/s/deg/s during far target viewing to 3.68 spikes/s/deg/s during near target viewing at 1.9 Hz, whereas response phase was not significantly affected by viewing distance. A large increase in the mean BP unit response gain was also observed during 0.7-Hz rotation.

These rotational responses are summarized in Table 2. The mean BP $\Delta GVD$, of 0.71 and 0.86 spikes/s/deg/s at 0.7 and 1.9 Hz, corresponded to an average increase in modulation of 23%. The 23% increase in modulation was larger than 17% increase in VOR gain recorded concomitantly, and the average increase in modulation ($\approx 16$ spikes/s) was larger than the increase that would be predicted from their sensitivity to pursuit eye velocity and static eye position ($\approx 12$ spikes/s).

**Position-vestibular-pause units**

The effects of viewing distance on the AVOR responses were recorded for 47 PVP units that were sensitive to ipsilateral head rotation in the plane of the horizontal semicircular canal. One unit was antidromically activated following electrical stimulation of the ipsilateral ATD, and thus presumably projected to the medial rectus subdivision of the ipsilateral oculomotor nucleus. Sample records and averaged responses of one PVP unit are illustrated in the left and middle columns of Fig. 6. The gain and phase of the responses of each PVP unit during 0.7-Hz smooth pursuit eye movements, VOR cancella-
tion, and the VOR are illustrated in the polar plots in Fig. 6, A3–D3. The firing characteristics of PVP units are summarized in Table 1.

Most of the PVPs tested (38/40) were activated at a monosynaptic latency following electrical stimulation of the ipsilateral vestibular nerve (Fig. 2A). During steady fixation in the absence of a target, the tonic firing rate of these units was related to eye position (range 0.4–10.6 spikes/s/deg; mean $K = 4.1 \pm 0.4$ spikes/s/deg). The spatial tuning of this static position signal calculated from multiple regression analysis varied considerably from cell to cell (see Fig. 5B) but was always contralateral. On the basis of their background firing rate during near and far target viewing, the eye position sensitivity of most PVPs (23/25) appeared to be better related to the position of the contralateral eye than to the ipsilateral eye (Fig. 5B). PVPs were either inhibited or stopped firing altogether during saccades; although the inhibition was typically weak or nonexistent when the saccade was in the unit’s on-direction. A few (6) PVP units generated bursts of spikes during on-direction saccades.

PVP units tended to be more sensitive to head rotation during the VOR than to eye movements alone. The modulation in their firing rate during horizontal smooth pursuit of a moving visual target (0.7 Hz, 20°/s) was $1.52 \pm 0.15$ spikes/s/deg/s re eye velocity (Fig. 6, A1–A3). This pursuit response phase-lagged contralateral eye velocity by an average of $40.2 \pm 5.6°$. When the monkey fixated a far, earth-stationary target, rotation of the turntable at the same velocity and frequency evoked a response whose gain was $1.71 \pm 0.14$ spikes/s/deg/s re ipsilateral head velocity and contralateral eye velocity during the VOR. The PVP unit responses phase-lagged head and eye velocity by 13 and 12°, respectively. During VOR cancellation (Fig. 6, B1–B3) the gain of PVP rotational responses decreased by $\sim 50\%$ (mean gain re head velocity = $0.88 \pm 0.09$ spikes/s/deg/s; see Table 1).

The rotational responses of most (34/47) PVP units increased as a function of viewing distance (Fig. 7), although in several of those units (9/34) the increase was small and statistically insignificant. On average, PVP head velocity sensitivity increased from $1.73$ spikes/s/deg/s during far target viewing to $2.04$ spikes/s/deg/s during near viewing at 0.7 Hz (see Table 2 and Fig. 7). The average PVP $\Delta GVD$ was $0.28 \pm 0.08$ spikes/s/deg/s; a 20% change in gain that was comparable with the concomitantly recorded VOR gain change of 17%. This small unit gain change was statistically significant ($P < 0.01$; t-test for paired observations) in spite of the fact that the viewing distance-related changes in response gain of individual PVP units varied considerably (Fig. 7, B and C). Thus the change in PVP unit population response was appropriate for generating the viewing distance-related changes in the AVOR that was concomitantly recorded, even though a significant fraction of units of this type were insensitive to viewing distance.

**Eye-head-velocity units**

The effects of viewing distance on the AVOR responses of 22 EHV units that were sensitive to head rotation in the plane of the horizontal semicircular canal were recorded. Six of these units were antidromically activated following electrical stimulation of the ipsilateral ATD. Sample records and averaged
responses of one EHV unit are illustrated in the left and middle columns of Fig. 8. The gain and phase of the responses of each EHV unit during 0.7-Hz smooth pursuit eye movements, VOR cancellation, and the VOR are illustrated in the polar plots in Fig. 8, A3–D3. The firing characteristics of EHV units are summarized in Tables 1 and 2. During VOR cancellation, 10 EHV units increased their firing rate during ipsilateral head rotation (EHVI units, + in Fig. 8, A3–D3), and 12 units increased their firing rate during contralateral rotations (EHVII units, square symbols in Fig. 8, A3–D3). Most of the EHVI and EHVII cells that were tested were activated at monosynaptic latencies following electrical stimulation of the ipsilateral vestibular nerve (16/17 tested; Fig. 2B).

Most EHV units were sensitive to eye position during steady fixation, but the preference on direction and ocular preference were idiosyncratic for each cell (Fig. 5C). Several EHVI units were more sensitive to vertical eye position and vertical smooth pursuit eye movements than to horizontal eye movements and position. EHVI units tended to be weakly related to ipsilateral conjugate horizontal eye position (mean gain = 1.1 ± 0.5 spikes/deg), whereas EHVII units were related to contralateral conjugate horizontal eye position (mean gain = 1.8 ± 1.0 spikes/deg). The ocular preference of 3 EHV units was for the ipsilateral eye, and for 10 other EHV units the contralateral eye. The tonic firing rate of EHV units that projected into the ATD, and presumably to the ipsilateral medial rectus subdivision of the oculomotor nucleus, was not necessarily related to the ipsilateral eye. The ocular preference of two ATD units was for the ipsilateral eye, and four others were related primarily to the contralateral eye (Chen-Huang and McCrea 1998a). EHV units also exhibited idiosyncratic responses during saccades. Most units (13/22) were either inhibited or stopped firing altogether during saccades in the oculomotor off-direction. Four EHV units generated bursts of spikes during on-direction saccades, and the firing rate of six others was not related to saccades in any direction. EHV units tended to be more sensitive to smooth pursuit eye movements (mean gain = 1.94 spikes/deg/s eye velocity) than to head velocity during the VOR (mean gain = 1.35 ± 0.18). The head movement on direction of this class of cells reversed during VOR cancellation, although the gain re head velocity during cancellation was, on average, less than half that recorded during the VOR evoked by fixation of an earth-stationary target (mean gain = 0.60 ± 0.08).

Two peculiar features of many EHV units were that their sensitivity to static eye position was frequently nonlinear, and that the eye movement–related signals exhibited during smooth pursuit eye movements were often different from the signals generated during steady fixation. Figure 9 illustrates an analysis of the firing behavior of three ATD EHV units in the left vestibular nucleus during steady fixation (top panels) and during smooth pursuit eye movements (bottom panels). Note that the modulated discharge of all three units led contralateral eye velocity during 0.7-Hz ocular pursuit, but the signals generated by each cell during steady fixation (between saccades) were idiosyncratic. The firing rate of the cell in Fig. 9A was correlated with ipsiversive eye position, the cell in B was related to contraversive eye position, whereas the cell in C was related to both ipsi- and contraversive eye position. Similar observations have been reported for secondary vestibular units that receive inputs from the ipsilateral flocculus (Lisberger et al. 1994).

The effects of viewing distance on the AVOR responses of EHV units were not uniform. The response gain of most (13/22) EHV units increased during near target viewing (Figs. 8 and 10, Table 2). However, near viewing had no significant effect on the AVOR response of five units, produced a decrease in gain in three units, and a reversal of response phase in the response of another. On average, the ΔGVD of EHVII units was 0.75 ± 0.24 spikes/s/deg/s at 0.7 Hz, which corresponded to a gain increase of 38%. EHV units tended to be less sensitive to head velocity during the VOR, and consequently their ΔGVD was smaller on average (0.41 ± 0.20 spikes/s/ deg/s at 0.7 Hz). However, the average increase in rotational gain of EHV units was 49%. On the whole, the viewing distance–related changes in firing rate was significantly larger for EHV units than for PVPs (t-test, P < 0.01).

Viewing distance significantly affected the response phase of most EHV units. In most cases the near response significantly phase lagged the response recorded during far target viewing. At 0.7 Hz, the AVORr response phase lagged the AVORf response by 34 ± 6° in the nine EHVII units that exhibited a significant ΔGVD increase. The response phase of EHV units was more mixed. The AVORr responses of most of these units also lagged the AVORf responses. However, the AVORr response of four EHV units phase led their AVORf responses by 11–54°. All of those EHVI units were sensitive to vertical eye movements and had AVORr responses that lagged contralateral head velocity by >70°.

In sum, viewing distance–related changes in the AVOR response gain of EHV units were, on average, larger than the change in response recorded from any other class of vestibular unit, and more than twice as large as the concomitant changes in eye movements.

**Position vestibular units**

PV units were encountered in the same region where PVP units were found. Most of the PV units (5/6) were activated at a monosynaptic latency following electrical stimulation of the ipsilateral vestibular nerve. The firing rate of these cells was correlated with static eye position but was unaffected during saccadic eye movements. The responses of PV units during smooth pursuit, VOR cancellation, and the far or near viewing VOR are summarized in Tables 1 and 2. In general, the smooth pursuit eye movement sensitivity of PV units was much lower than the sensitivity to static eye position.
than PVP and EHV units. The gain and phase of the rotational responses of PV units were similar to PVPs when the animal was fixating an earth-stationary target or was canceling its VOR (Table 1). On average, PV units were more sensitive to changes in viewing distance than PVPs. Their mean D\text{GVD} was $0.38 \pm 0.53$ (spikes/s/deg/s), and their N/F ratio was $1.41 \pm 0.25$ during 0.7-Hz rotation. In sum, PV units were not as sensitive to eye movements as EHV and PV units, but the sensitivity of their rotational responses to viewing distance was quite high.

Other vestibular units

Ten units were encountered in the vestibular nuclei that could not be readily classified in one of the five cell classes described above. Nine of the units were activated at monosynaptic latencies following electrical stimulation of the vestibular nerve. Six of the cells were eye movement–related type II neurons (EVII units), whose firing rate was modulated in phase with contralateral head velocity during rotation and with ipsilateral eye velocity during smooth pursuit. The rotational responses of EVII units were also sensitive to viewing distance. During 1.9-Hz table rotations, where the result for every unit was available, the mean D\text{GVD} increased 24.0% to 19.0%. The responses of EVII units are summarized in Tables 1 and 2.

Two monosynaptically activated units were related to contralateral smooth pursuit eye velocity but not to static eye position. One monosynaptically activated unit did not respond to any type of eye movements but showed large differences between VOR cancellation and AVORf. One other unit paused during saccades but exhibited no other signals related to eye movements. The rotational responses of the pausing neuron and one of the two pursuit units increased during near viewing. The rotational responses of the other two units were unaffected by viewing distance.

Effects of stimulus frequency on AVOR D\text{GVD}

Most of the results described above were obtained with a sinusoidal vestibular stimulus ($0.7$ Hz, 20°/s) that matched the stimulus frequencies used to evoke smooth pursuit eye movements and to evaluate the responses of units during VOR cancellation. The AVOR responses were also recorded during higher frequencies of vestibular stimulation in many (87/105) units. Figure 11 illustrates examples of the averaged AVOR responses of a PVP unit and an EVII unit evoked at three rotation frequencies ($0.7$ Hz, 20°/s; $1.9$ Hz, 10°/s, and/or $4.0$ Hz, 6°/s).

Graphic summaries of the responses of 13 PVP and 7 EVII units that were tested at all 3 frequencies of rotation are illustrated in Fig. 11, D1 and D2. The average response gain and phase of both classes of cells tended to increase slightly during far target viewing as stimulus frequency increased, although there was considerable individual variability within each class. During near target viewing the average PVP unit gain decreased slightly from $2.04 \pm 0.18$ spikes/s/deg/s at $0.7$ Hz to $1.53 \pm 0.24$ spikes/s/deg/s at $4$ Hz, whereas the average EVII unit gains were nearly the same at each stimulus frequency. Viewing distance had larger, significant effects on the average response gain of the 32 vestibular units tested at 0.7 Hz, but it did not affect the average response gain of these vestibular neurons recorded at $4$ Hz, which corresponded to the changes in AVOR gain as a function of viewing distance at these frequencies.

The AVOR near target responses of both PVP and EVII units slightly phase lagged their responses during far target viewing at every frequency (Fig. 11, D1 and D2). The phase lag in unit response produced by a reduction in viewing distance was remarkably constant at different frequencies.

Firing behavior of vestibular units during voluntary cancellation of AVORf

The responses of 16 vestibular units, including 5 EVII and 11 PVP units, were recorded during cancellation of a head-stationary target located on the midline 10 cm from the monkey’s eyes. The firing behavior of vestibular neurons during 0.7-Hz VOR cancellation in the presence of near (VORc\text{n}) and far (VORc\text{f}) head-stationary targets was compared, and the effects of viewing distance on rotational gains were assessed.

Cancellation of the VOR during fixation of a head-stationary near target (mean VOR gain = $0.07 \pm 0.01$ re head velocity) was equal or better than VOR cancellation during fixation of a head-stationary far target (mean gain = $0.14 \pm 0.01$). EHV units, as noted above, characteristically reversed the direction of their response during VOR cancellation, whereas PVP units characteristically exhibited a reduction in their rotational response. The response of one unit during cancellation of AVOR\text{n} and AVOR\text{f} is illustrated in Fig. 12A. The filled, shaded histogram is the averaged response recorded during fixation of an earth-stationary near target, whereas the superimposed thin and thick traces show the averaged responses recorded during fixation of head-stationary far and near targets, respectively. The unit’s modulation during VORc\text{n} and VORc\text{f} was similar, regardless of the distance of the target, although there was a small decrease in the background firing rate during near target viewing that was related to the more medial deviation of the contralateral eye during VORc\text{n}.

In each of the PVP and EVII units tested, the response recorded during VORc\text{n} was comparable with the response recorded during VORc\text{f} cancellation (Fig. 12B). During VORc\text{n} the average gain of PVP responses was $0.8 \pm 0.2$ spikes/s/deg/s compared with $1.0 \pm 0.2$ spikes/s/deg/s during VORc\text{f}. The mean response phase led head velocity by 45° during VORc\text{n} and 17° during VORc\text{f}. The small differences in response gain recorded during AVOR\text{n} cancellation and AVOR\text{f} cancellation (VOR D\text{GVD}; Fig. 12C) were not correlated with viewing distance sensitivity. The comparability of the single-unit responses during VOR cancellation might be interpreted to be a reflection of the fact that the D\text{GVD} signals on secondary VOR neurons were related to their eye movement sensitivity. An alternative possibility is that the inputs that reduce or modify the rotational responses of PVP and EVII units during VORc are also modified as a function of viewing distance. The comparison of unit viewing distance sensitivity and eye movement sensitivity in the next section suggests that the latter interpretation is more likely.
Relationship of AVOR DGVD to pursuit eye velocity sensitivity and static eye position sensitivity

The viewing distance–related changes in AVOR responses of central vestibular neurons were not well correlated with their sensitivity to eye velocity during smooth pursuit. The correlation coefficient of the linear relationship between smooth pursuit eye velocity sensitivity and AVOR DGVD was 0.51 for PVP units and 0.05 for EHVII units. On the other hand, unit sensitivity to viewing distance was better correlated with their static eye position sensitivity. The correlation between eye position sensitivity and the increase in unit modulation during near target viewing was 0.76 for EHVII units, 1.00 for EHVII units, and 0.58 for PVP units (Fig. 13).

The positive correlation between AVOR DGVD and static eye position sensitivity was not readily attributable to the increase in the amplitude of eye movements during near viewing per se. The slopes of the regressions illustrated in Fig. 13 suggest that PVP and EHVII units tended to be three times more sensitive to the increased amplitude of eye movements during near viewing than their static eye position sensitivity coefficients would predict. Subtraction of static eye position signals from vestibular unit rotational responses affected both their gain and phase, particularly at the 0.7-Hz stimulus frequency, but on average this removal had little effect on the increase in response gain recorded during near viewing. The effects of subtraction of eye position signals on the average responses of representative BP, PVP, and EHVII units during 1.9-Hz rota-

FIG. 13. Vestibular unit change in firing rate related to viewing distance vs. static eye position sensitivity (Ks). Values on both the ordinate and abscissa are expressed in terms of unit sensitivity to eye position (spikes/s/deg). Red solid line is the regression related to EHVII units (red squares), and the dashed line is the regression related to PVP units (green squares). The viewing distance sensitivity of EHVII units was strongly correlated with their sensitivity to eye position (R = 0.82), although the slope of the regression (3.4) was greater than 3 times that expected if eye position signals alone had caused the change in unit gain during AVORc (thin black dotted line). The relationship between viewing distance sensitivity and eye position sensitivity was weaker for PVP units (R = 0.46), but the slope of the regression was similar.

Relationship of AVOR ΔGVD to pursuit eye velocity sensitivity and static eye position sensitivity

The viewing distance–related changes in AVOR responses of central vestibular neurons were not well correlated with their sensitivity to eye velocity during smooth pursuit. The correlation coefficient of the linear relationship between smooth pursuit eye velocity sensitivity and AVOR ΔGVD was 0.51 for PVP units and 0.05 for EHVII units. On the other hand, unit sensitivity to viewing distance was better correlated with their static eye position sensitivity. The correlation between eye position sensitivity and the increase in unit modulation during near target viewing was −0.76 for EHVII units, +0.85 for EHVII units, and +0.58 for PVP units (Fig. 13).

The positive correlation between AVOR ΔGVD and static eye position sensitivity was not readily attributable to the increase in the amplitude of eye movements during near viewing per se. The slopes of the regressions illustrated in Fig. 13 suggest that PVP and EHVII units tended to be three times more sensitive to the increased amplitude of eye movements during near viewing than their static eye position sensitivity coefficients would predict. Subtraction of static eye position signals from vestibular unit rotational responses affected both their gain and phase, particularly at the 0.7-Hz stimulus frequency, but on average this removal had little effect on the increase in response gain recorded during near viewing. The effects of subtraction of eye position signals on the average responses of representative BP, PVP, and EHVII units during 1.9-Hz rota-
The mean eye position-corrected 1.9-Hz AVOR responses of BP, PVP, and EHVII units are summarized in the polar plots at the bottom of Fig. 14 (see also Table 2). Subtraction of static eye position signals produced a phase lead in both BP and PVP unit responses (Fig. 14, A and B), but the gain change produced by changing viewing distance was not significantly affected. Subtraction of static eye position signals had little effect on the gain or phase of EHVII unit rotational responses during near and far target viewing (Fig. 14C). Subtraction of static eye position signals...
FIG. 15. Polar plot of the viewing distance–related rotational responses of different classes of vestibular neurons and the concomitant change in eye movements at 1.9 Hz. The vector difference in the response of PVP, EHV, and BP units recorded during near and far target viewing is plotted as phasors P, E, and B, respectively. PVP and EHV viewing distance–related signals phase lag ipsilateral head velocity, and lag the signals generated by ipsilateral irregular afferents by ∼90°. EHV units are approximately twice as sensitive to viewing distance as PVP units. Viewing distance–related signals carried by most BP units phase lag ipsilateral eye velocity. The phasor sum of the viewing distance–related response of all 3 classes of cells at 1.9 Hz is similar in phase to the vector difference in eye movements evoked by rotation during near and far target viewing (Eye), if it is assumed that the signals carried by PVPs or EHVs are anatomically inverted while ipsilateral BP units are not. These observations suggest that the viewing distance changes in central vestibular neurons are sufficient to produce the changes in eye movement observed.

also did not account for viewing distance–related unit responses during 0.7-Hz rotation (Table 2), although the eye position changes evoked at that frequency were larger.

In sum, static eye position sensitivity and viewing distance sensitivity were correlated, but the increase in rotational gain of eye movement–related central vestibular units during near target viewing was not directly attributable to eye position and eye velocity oculomotor signals they carry.

Relationship between vestibular nerve signals and viewing distance related unit and eye movement signals

In a previous study we showed that viewing distance–related changes in the AVOR were significantly reduced when irregular afferents were selectively silenced by bilateral application of galvanic anodal currents (Chen-Huang and McCrea 1998b), whereas the AVORf was unaffected. We suggested that the central AVOR AGVD signals might reflect the addition of an irregular afferent input to secondary AVOR pathways. Figure 15 is a polar plot of the difference between the AVORn and AVORf uncorrected 1.9-Hz responses of the three classes of neurons illustrated in Fig. 14. These phasors represent the signals added to each unit class during near target viewing. The dashed arrow corresponds to the phase of the vector difference between AVORn and AVORf eye movements. The shaded sector of the polar plot represents the response phase of squirrel monkey vestibular nerve irregular afferents at 2 Hz (Lysakowski et al. 1995). Considered together, PVP and EHV viewing distance signals lagged the mean vestibular nerve irregular afferent signals by ∼91°. On the other hand the viewing distance signals generated by BP units had nearly the same phase as the viewing distance–related eye movements.

In Fig. 15, the phasor sum of the viewing distance signals related to BP, PVP, and EHV units (labeled B, P, and E, respectively) was 134° when the signals related to BP and PVP units were inverted. The inversion could occur anatomically in the case of units that project to the contralateral abducens nucleus or ipsilateral medial rectus subdivision of the oculomotor nucleus. The sum of the inverted EHV and PVP signals with BP signals had nearly the same phase as the viewing distance–related eye movement at 1.9 Hz. This suggests that inputs from these three groups of units to the oculomotor plant are sufficient to produce AVORn at 1.9 Hz.

Discussion

The gain of the VOR evoked by semicircular canal stimulation needs to increase as an inverse function of viewing distance to compensate for the differences in the axis of rotation of the eyes and head (Virsch et al. 1986). In the squirrel monkey the rotational sensitivity of many secondary vestibular neurons is altered as a function of viewing distance. These changes in sensitivity related to viewing distance, ΔGVD, were found only in classes of vestibular neurons that have been shown in previous studies to project to the extraocular motor nuclei. Although putative secondary VOR neurons varied considerably in their sensitivity to viewing distance; on the whole, our results suggest that the signals carried by direct VOR pathways from the vestibular nuclei to extraocular motoneurons are sufficient to generate the behavioral changes in VOR gain that were observed.

Two notable features of the central signals related to AVOR ΔGVD in putative secondary VOR neurons were that they phase lagged head velocity by nearly 90° and were correlated with static eye position sensitivity. These observations, together with the low band-pass characteristic of the viewing distance AVOR gain adjustment in squirrel monkeys suggest that viewing distance–related changes in the AVOR utilize central circuits that are involved in temporal integration of the AVOR. In the following discussion, we will first address certain technical issues related to the variability in the AVOR ΔGVD. We will then briefly discuss our neurophysiological observations in light of previously described anatomy and physiology of central horizontal canal–related AVOR pathways. Finally, we will discuss the nature of the neural mechanisms related to viewing distance–related changes in the AVOR and advance a specific hypothesis for how AVOR signals are modified as a function of viewing distance.

Technical issues related to viewing distance–related changes in the AVOR

We previously reported that viewing distance–related gain changes in the squirrel monkey AVOR are variable both within and between animals (Chen-Huang and McCrea 1998b). The distance of the squirrel monkey’s interaural axis from the center of the eye is ∼2.25 cm, and in our experiments the gain of the VOR needed to be increased by ∼20% to stabilize a visual target on the retina that was 12.25 cm in front of the axis of head rotation. Indeed, each of the squirrel monkeys in this
study increased the gain of their AVOR, although, on average, the increase was 17% at the lowest stimulus frequencies used. This mean increase in AVOR gain while viewing a near target was slightly higher than the change in gain we previously reported in a study of the effects of functional ablation of irregular afferents on the AVOR \(_{\text{r}}\) (Chen-Huang and McCrea 1998b). One problem in estimating single-unit AVOR \(\Delta \text{GVD}\) values was that a 17% gain change represented a relatively small change in firing rate (%6 spikes/s at 0.7 Hz, %3 spikes/s at 1.9 Hz) in most cells, even though unit \(\Delta \text{GVD}\) was slightly larger on average than the change in eye movement. The small changes in firing rate and variable changes in AVOR \(_{\text{r}}\) gain may partially explain why the responses of many putative secondary VOR units were not significantly affected by viewing distance, and why these changes in response gain were not observed in a previous study by McConville et al. (1996).

A second behavioral issue relates to the relationship between vergence angle and AVOR gain. Although vergence angle and AVOR gain were positively correlated, the correlation was not perfect. AVOR \(_{\text{r}}\) gain varied from trial to trial, but the vergence angle generated by a particular monkey was relatively stereotyped and fixed. The fluctuation in AVOR \(_{\text{r}}\) gain was thus not attributable to fluctuations in vergence angle. Other factors may be involved in triggering viewing distance–related gain changes in the AVOR. Unfortunately vergence angle is the only variable we measured or controlled.

Neural substrate for generating viewing distance–related gain changes in the AVOR

The results of a number of anatomic and electrophysiologic studies suggest that the immediate premotor substrate for generating the horizontal canal–related AVOR includes direct secondary VOR pathways made up of PV, PVP, and EHV units and more indirect pathways from the vestibular nerve to medial and lateral rectus motoneurons (Cullen and McCrea 1993; McCrea et al. 1980, 1987; Reisine and Highstein 1979; Scudder and Fuchs 1992). The latter include primarily pathways from BP units in the rostral prepositus and adjacent regions of the medial vestibular nucleus (Cullen et al. 1993; McFarland and Fuchs 1992; Scudder and Fuchs 1992). The identification of putative VOR-related vestibular units in this study depends primarily on observations made in those previous studies, although a few units that projected into the ascending tract of Deiters were identified by antidromic activation following stimulation of that tract (see Chen-Huang and McCrea 1998a).

McConville et al. (1996) reported that vestibular units whose rotational responses were modified by viewing distance also carried signals related to eye movements. In this study, we also found that all of the vestibular units whose firing behavior was related to viewing distance were eye movement–related cells. Although it has certainly not been demonstrated that all eye movement–related vestibular neurons are premotor neurons, there is evidence that virtually every major subcategory of eye movement–related secondary vestibular neurons projects to one or more of the extracocular motor nuclei. In any case, it is not unreasonable to suggest that most eye movement–related extracocular vestibular units whose firing rates are modulated during the VOR are probably involved, in some way, in controlling or generating the VOR. This assumption seems particularly valid for the four classes of eye movement–related units described in this study: PVP units, EHV units, BP units, and PV units.

The change in gain of the signals carried to the extraocular motor nuclei by direct central secondary VOR pathways appears to be at least as large as the concomitant change in eye movement gain. The average increase in rotational gain exhibited by PVP and BP units during near target viewing was larger than the concomitant increase in eye movements, and the signals generated by EHV and PV units increased in gain more than twice as much as the eye movements. It seems likely that the AVOR-related signals carried by the other, indirect, central pathways are similarly affected by viewing distance. For example, all except one of the vestibular BP units described in this study were activated at disynaptic latencies following electrical stimulation of the vestibular nerve and were more likely related to these indirect AVOR pathways. Because the firing behavior of many EHV cells and BP cells is similar to that described for vestibular units that are inhibited following electrical stimulation of the cerebellar flocculus (Lisberger et al. 1994), it seems likely that the AVOR pathways receiving inputs from the flocculus are particularly sensitive to viewing distance.

Viewing distance adjustments in vestibular sensitivity make sense for neurons involved in controlling the VOR, but the function of this adjustment for cells involved in other vestibular functions, for example vestibulospinal pathways, is not obvious. Tomlinson et al. (1996) found that viewing distance did not modify the rotational responses of vestibular units whose firing behavior was not related to eye movements. We also were unable to observe viewing distance–related changes in the angular rotational responses of most VI units. Currently there is no evidence that vestibular neurons of this type project to the extraocular motor nuclei in the primate.

Contribution of smooth pursuit and visual feedback signals to vestibular unit viewing distance sensitivity

The AVOR responses of EHV and BP units were on average more than twice as sensitive to viewing distance as PVP units. EHV and BP vestibular units also tended to be more sensitive to smooth pursuit eye movements than PVP units. These observations, together with the low-pass filtered characteristic of the viewing distance–related changes in the squirrel monkey AVOR, raise the question of whether viewing distance–related changes in the AVOR are mediated by a visual feedback or smooth pursuit mechanism.

There are several reasons why it is unlikely that ocular pursuit and/or visual feedback are essential for viewing distance gain changes in the AVOR:

1) AVOR \(\Delta \text{GVD}\) is evident before the onset of the eye movement that brings a near target on the fovea (Snyder et al. 1992).

2) AVOR \(\Delta \text{GVD}\) persists in the dark for many seconds (Hine and Thorn 1987; Viirre et al. 1986).

3) The latency of AVOR \(\Delta \text{GVD}\) responses evoked by steps in head acceleration is shorter than the shortest latency visual feedback signals that have been described (Chen-Huang and McCrea 1998b; Snyder and King 1992; Snyder et al. 1992).

4) The magnitude of AVOR \(\Delta \text{GVD}\) signals on individual
secondary vestibular neurons is not well correlated with their smooth pursuit eye velocity sensitivity.

5) And, finally, functional ablation of vestibular nerve irregular afferents has no effect on smooth pursuit eye movements, but seriously compromises AVOR ΔGVD (Chen-Huang and McCrea 1998b).

In sum, visual feedback mechanisms may supplement or add to the viewing distance adjusted signals carried by central AVOR pathways, but the existing evidence suggests that these inputs are neither necessary nor sufficient to change the gain of the AVOR as a function of viewing distance.

It is reasonable to assume that viewing distance–related changes in the gain of the AVOR are usually, if not always, triggered by a visual estimate of target distance. It also appears that the central premotor pathways that are most important for generating these gain changes in the AVOR are those that are most sensitive to visual feedback. However, the weight of evidence suggests that the change in gain of the AVOR is accomplished by multiplication of a vestibular signal that is then added onto secondary VOR pathways rather than an addition of a visual signal.

**Relationship between viewing distance–related changes in the AVOR and AVOR cancellation**

Squirrel monkeys were at least as capable of suppressing or canceling near viewing VOR as they were the VOR evoked during far target viewing. VOR cancellation tended to be more complete when a near LED target was fixated than when the 90-cm distant head-stationary laser spot was fixated. The improvement in VOR cancellation may have been related to the fact that the visual angle subtended by the near target was larger than that subtended by the far target. However, it is clear that the mechanisms involved in AVOR cancellation are capable of canceling the viewing distance–enhanced AVOR as well.

One possible interpretation of these results is that the “vestibular” signals carried by secondary vestibular EHV and PVP neurons were largely unaffected by viewing distance. The problem with this idea is that many vestibular neurons may receive inputs that are specifically related to cancellation of the VOR and function to cancel, or reduce vestibular signals on VOR pathways during fixation of head-stationary targets. For example, EHV units may receive modulated inputs from gaze velocity cerebellar flocculus Purkinje cells during VOR cancellation but not during the VOR. A more plausible explanation of the VOR cancellation results is that the central signals that modify signal processing in VOR pathways during VOR cancellation are sensitive to viewing distance.

**Relationship between vestibular unit static eye position signals and viewing distance sensitivity**

The AVOR viewing distance sensitivity of central vestibular units was weakly but positively correlated with their sensitivity to eye position. The most striking correlation between static eye position sensitivity and AVOR ΔGVD was in EHV units, but the smaller response changes observed in PVP units were also positively correlated with their static eye position sensitivity. This correlation, together with the observation that ΔGVD signals tend to phase lag head velocity, suggests that ΔGVD signals might be related to the central AVOR velocity-position integrator.

The static eye position signals on secondary VOR neurons are clearly not, by themselves, sufficient to cause the changes in rotational response observed during near target viewing. Subtraction of static eye position signals from unit rotational responses only reduced the mean AVOR ΔGVD signal generated by PVP units by half and had virtually no effect on the mean response of EHV units. The latter observation is consistent with the idea presented below that the input to the AVOR integrator is multiplied to produce the enhanced signal on secondary VOR neurons during near viewing.

If the ΔGVD signals generated by secondary VOR neurons are related to pathways that feed back integrated head velocity signals to secondary VOR neurons (see below), it is reasonable to expect that these signals would be mixed with static eye position signals. No central neuron has been identified whose firing behavior is specifically related to the AVOR integrator, but there are many neurons in the vicinity of the vestibular nuclei, particularly in the rostral medial vestibular nucleus and the prepositus nucleus, that receive disynaptic inputs from the vestibular nerves and generate signals that phase lag head velocity by 40–90° (Escudero et al. 1992; Lopez-Barneo et al. 1982). These integrated vestibular signals usually coexist with static eye position signals and dynamic eye position signals related to ocular pursuit, although the latter two signals may differ significantly in various types of VOR neurons (Cullen et al. 1993; Tomlinson and Robinson 1984).

**Monocular static eye position signals on vestibular neurons**

The eye position signals generated by most, if not all, of the neurons in the regions of the brain that are likely to be immediately involved in eye velocity-position integration are monocular (McConville et al. 1994; Zhou and King 1996, 1997). Although this issue was not studied systematically, we also found that most of the units in this study were dominantly related to the movements of one eye. Presumably, the difference between the signals generated by the neural integrators related to each eye could represent an internal estimate of viewing distance. This viewing distance estimate could then be used to amplify vestibular afferent inputs to VOR pathways.

**Scheme for amplifying vestibular signals by viewing distance**

The polar plot in Fig. 15 illustrates that the change in the signals produced by secondary vestibular neurons during near target viewing is comparable with the change in eye movement gain. The phase of the viewing distance–related signals lags vestibular irregular afferent signals by an average of ~90°. We suggest that this viewing distance–related change in the responses of secondary vestibular pathways is produced primarily by the addition of multiplied, temporally integrated vestibular irregular afferent inputs.

Most secondary vestibular neurons receive inputs from a combination of regular and irregular vestibular nerve fibers (Goldberg et al. 1987). The latter inputs can be selectively silenced by application of galvanic currents, which reveals that most central neurons receive not only direct excitatory inputs...
neurons; NI, neural integrator. Relayed in part through secondary VOR pathways. MNS, extraocular motor neurons. In this case mediated by the central AVOR neural integrator, receives viewing integrated irregular afferent vestibular input to secondary AVOR pathways (in this study assumed by the observation that functional ablation of irregular afferents dramatically reduces or abolishes viewing distance–related gain changes in the AVOR. In A, the gain of an inhibitory irregular vestibular afferent input to secondary VOR pathways is adjusted by viewing distance. During far target viewing, the inhibitory irregular afferent input equals the direct excitatory input, and the VOR is driven by regular vestibular afferent inputs. During near target viewing, a central estimate of viewing distance (in this case generated by comparing monocular eye position signals related to the ipsilateral and contralateral eyes) adjusts the threshold of interneurons that mediate the inhibitory irregular afferent input to secondary VOR pathways so that progressively more interneurons are silenced as the 2 eyes converge. The reduction of inhibitory inputs results in an increased AVOR gain. The more complex scheme illustrated in B is motivated by the observation that the gain increase in eye movements and secondary vestibular responses during near viewing appear to be due to the addition of integrated vestibular signals. Therefore an additional integrated irregular afferent vestibular input to secondary AVOR pathways (in this case mediated by the central AVOR neural integrator) receives viewing distance multiplied vestibular afferent inputs. The output of the integrator is relayed in part through secondary VOR pathways. MNS, extraocular motor neurons; NI, neural integrator.

One way the gain of the AVOR could be adjusted as a function of viewing distance is illustrated in Fig. 16A. In this scheme, the inhibitory irregular afferent inputs to secondary VOR neurons are relayed through a set of inhibitory interneurons that receive inputs from irregular vestibular nerve afferents whose threshold is set by another inhibitory input whose gain is inversely related to viewing distance. Individual elements of this viewing distance multiplier could code viewing distance by receiving excitatory inputs related to the position of one eye and inhibitory inputs related to the position of the other. The difference between the two inputs would be equal to a central estimate of viewing distance. When the two inputs are equal, viewing distance would be infinite, and the inhibitory feedback pathway could cancel irregular afferent inputs to secondary VOR neurons. As the two eyes converge on a near target, the inhibitory eye position signal would drive progressively more interneurons into inhibitory saturation and effectively increase the irregular afferent input to secondary VOR pathways. Alternatively, the viewing distance signal could be a signal related to vergence angle.

Although the direct parametric adjustment in irregular afferent inputs to secondary VOR pathways would be an elegant way to modify the gain of the AVOR as a function of viewing distance, several observations suggest that the viewing distance multiplied inputs to secondary VOR pathways are low-pass filtered. First of all, if irregular afferent inputs to AVOR pathways were gated in a manner similar to that shown in Fig. 16A, their AVOR, signals would phase lead, rather than phase lag their AVORn responses. Second, the gain of the near viewing AVOR would be expected to progressively increase as a function of stimulus frequency rather than decrease (Fig. 1B). Because the viewing distance–related rotational signals generated by EHV and PVP units typically phase lag irregular afferent signals by $-90^\circ$ (Fig. 15), it seems likely that a large fraction of the viewing distance multiplied irregular afferent signal input to secondary VOR neurons is low-pass filtered or integrated. One place this could occur is in the AVOR velocity-position integrator itself, as illustrated in Fig. 16B. The correlation between the viewing distance sensitivity of EHV and PVP units and contralateral static eye position sensitivity shown in Fig. 13 suggests that the central pathways involved in changing the gain of the AVOR as a function of viewing distance also transmit contralateral static eye position signals to secondary VOR pathways. The idea is attractive in part because the sensitivity of secondary vestibular units to viewing distance was correlated with their eye position sensitivity, and in part because it relieves the necessity to postulate the existence of a separate neural integrator specifically related to viewing distance changes in the AVOR. Clearly additional study is required to determine whether regions that are considered to be related to central integration of the AVOR, such as the prepositus nucleus, generate signals that are necessary or sufficient to produce viewing distance–related changes in eye movements or secondary VOR pathways that have been observed in this study.

**Conclusion**

The results of this study suggest that the central premotor pathways that mediate the horizontal canal–related AVOR also

![Figure 16](image)
mediate viewing distance–related changes in the reflex. We suggest that a central estimate of viewing distance modifies the gain of indirect, centrally integrated, vestibular semicircular canal irregular afferent inputs to secondary neurons to produce the change in gain in the AVOR observed.

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Present address of C. Chen-Huang: Dept. of Physiology, Northwestern University, Chicago, IL 60611.

Address for reprint requests: R. A. McCrea, Dept. of Neurobiology, Pharmacology and Physiology, 947 E. 58th St. (MC 0926), University of Chicago, Chicago, IL 60637.

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