Tests of Linearity in the Responses of Eye-Movement-Sensitive Vestibular
Neurons to Sinusoidal Yaw Rotation

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Author Contributions:
Shawn D. Newlands – Designed experiments, analyzed data, wrote paper
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PVP and EHV dynamics
ABSTRACT:
The rotational vestibulo-ocular reflex in primates is linear and stabilizes gaze in space over a large range of head movements. Best evidence suggests that position-vestibular-pause (PVP) and eye-head velocity (EHV) neurons in the vestibular nuclei are the primary mediators of vestibulo-ocular reflexes for rotational head movements, yet the linearity of these neurons has not been extensively tested. The current study was undertaken to understand how varying magnitudes of yaw rotation are coded in these neurons. 66 PVP and 41 EHV neurons in the rostral vestibular nuclei of 7 awake rhesus macaques were recorded over a range of frequencies (0.1 to 2 Hz) and peak velocities (7.5 to 210 °/s at 0.5 Hz). The sensitivity (gain) of the neurons decreased with increasing peak velocity of rotation for all PVP neurons and EHV neurons sensitive to ipsilateral rotation (type I). The sensitivity of contralateral rotation sensitive (type II) EHV neurons did not significantly decrease with increasing peak velocity. These data show that, like non-eye-movement-related vestibular nuclear neurons that are believed to mediate nonlinear vestibular functions, PVP neurons involved in the linear vestibulo-ocular reflex also behave in a nonlinear fashion. Similar to other sensory nuclei, the magnitude of the vestibular stimulus is not linearly coded by the responses of vestibular neurons; rather amplitude compression extends the dynamic range of PVP and type I EHV vestibular neurons.

Keywords – Position-vestibular-pause, eye-head velocity, gaze velocity, vestibular nuclei, vestibulo-ocular reflex
INTRODUCTION:

One of the fundamental problems in sensory physiology is how the nervous system, constrained by the physical properties of sensory receptors and neurons, conveys signals from a complex world into the brain. In the vestibular system, a classic question is how this system transforms inherently nonlinear signals in the vestibular nerve, which are silent with strong inhibitory stimulation, into reflexive eye movements that are symmetrical, linear and robust over a very wide range of frequencies and velocities (Huterer and Cullen, 2002; Ramachandran and Lisberger, 2005; Tomlinson, 1990). Conceptually, this problem can be solved by the combination of signals from both vestibular nerves, which are complementary in that the excitatory phase in one vestibular nerve will provide information centrally that is lost during silencing in the opposing nerve (Abend, 1978). Presumably, such an interaction could occur in the vestibular nuclei, where a substantial commissural system interconnects the bilateral vestibular nuclei, and in the vestibulocerebellum, which also has extensive interconnection to the vestibular nuclei.

The vestibular system is often labeled sensorimotor because of convergence of vestibular, optokinetic, somatosensory and motor-related signals in the vestibular nuclei. Neurons in the vestibular nuclei are proposed to coordinate both sensory inputs and motor signals to deliver an inversely engineered signal to the motor nuclei. The inherent attributes of the motor system then compensate for the limitations of the signal from the vestibular system. This concept is best developed in the direct rotational VOR system,
where a combination of data and models has allowed assignment of particular
roles in sensorimotor transformation to particular neuronal types in the vestibular
nucleus. While these models are compelling, they have largely been tested only
in limited stimulus ranges of frequency and velocity of rotation, and start with an
assumption of linearity, at least over a physiologic range of natural movements
(Green and Angelaki, 2010 for review).

Other data have challenged the concept that vestibular signals in the VN
are indeed linear (Mussalum and Tomlinson, 2001, 2002; Newlands et al., 2009).
Linearity requires both homogeneity, meaning that the output of the system
increases linearly with the input, and additivity, meaning that given two inputs
that the output of the system is the linear summation of the outputs that would be
seen for each input applied independently. Neurons in these studies
demonstrated nonlinearity; primarily in the form of lacking scalability
(inhomogeneity), meaning that the output of the system does not increase
linearly with the input. These earlier studies reported on neurons without eye
movement sensitivity. These non-eye-movement vestibular neurons (NEM),
which are also known as vestibular-only (VO) neurons (Fuchs and Kimm, 1975)
are not directly implicated in direct VOR pathways (Scudder and Fuchs, 1992)
and probably are a collection of diverse neurons serving a number of purposes in
vestibular function (Boyle 1993; Cheron et al 1996). Neurons that are directly
involved in the VOR include Position-Vestibular-Pause (PVP) and Eye-Head
Velocity (EHV) (Scudder and Fuchs, 1992; Cullen and McCrea, 1993; McCrea et
al., 1980, 1987). According to prevailing models, these neurons would be
expected to display linear behavior, at least over a physiological range of motion
stimuli (Green and Angelaki, 2010). However, the linearity of these neurons has
not been directly tested over a wide range of stimuli.

The current study examines the responses of the two major identified
neuronal types in the vestibular nuclei that have been demonstrated to have
sensitivity to yaw rotation and to eye movements to a range of peak velocities
while holding the frequency of the rotation stable. We demonstrate that PVP and
EHV cells, like NEM neurons, do not scale their firing responses to vestibular
input linearly, even at relatively low levels of stimulation. This nonlinearity is
consistent with a strategy that trades off linearity for the extension of dynamic
range that is required for sensory systems, which have wide dynamic ranges
despite having discharge rates that are limited by rectification and saturation.
METHODS:

Animal preparation:

Single neurons in the vestibular nuclei were recorded from seven juvenile rhesus monkeys (Macaca mulatta) of both sexes with weights ranging from four to seven kg. Five of these animals were included in a previously reported study (Newlands et al., 2009). The surgical techniques have been described previously (Newlands et al., 2009). In short, animals were implanted with dental acrylic head caps for head immobilization in a primate chair and with a stereotaxically placed recording chamber for bilateral access to the abducens nucleus and rostral vestibular nuclei. Eye coils (three turn, 15.5 mm) were attached to the sclera of one eye with 7-0 prolene sutures in a second procedure. Some animals had bilateral eye coil placement. After surgery the animals were treated with postoperative antibiotics for 7 days and intramuscular buprenorphine (0.01 mg/kg) for pain control twice daily for 3 days. All surgical procedures were performed in accordance with institutional and National Institutes of Health guidelines and under a protocol approved by the appropriate Institutional Animal Care and Use Committees at the University of Texas Medical Branch and at the University of Rochester.

Experimental setup and recording:

This experimental setup has also been previously described in detail (Newlands et al., 2009). In short, the animal was head-restrained and the head is immobilized pitched 20° nose-down in primate chair, which was mounted on a motorized turntable. A two-field magnetic search coil system (C-N-C Engineering
Systems, Seattle, WA) was used to record monocular eye position. The chair could be pitched/rolled (up to ± 24°) to confirm that the neurons did not have dynamic pitch/roll sensitivity. All of the motors were controlled by custom-written software (LabVIEW, National Instruments, Austin, TX). Visual targets were presented using a laser reflected by an x-y mirror galvanometer system (General Scanning, Billerica, MA) onto a target screen for eye movement tasks (targeted saccades or pursuit) or head-fixed lasers projected onto a screen attached to the primate chair for VOR suppression. Animals were trained to fixate targets with accuracy windows set at 1-2° in both the x and y direction for liquid rewards. All of the visual targets were also controlled by custom software written in LabVIEW. Eye coils were calibrated during the smooth pursuit task in each recording session.

Extracellular recordings were carried out using epoxy-coated tungsten microelectrodes (2-10 Mohm impedance, FHC, Bowdointam, ME) with techniques previously described (Newlands et al., 2009). Recordings were concentrated in the rostral vestibular nuclei, 1-5 mm posterior and 0-4 mm lateral to the abducens nuclei on either side. Histological confirmation of recording sites is available in 5 monkeys; histology was lost for one animal, and one monkey is still alive.

Individual recording sessions were performed by placing the microelectrode, sheathed in a 26 gauge cannula, roughly 5-7 mm above the area of interest and then driving the microelectrode using a remote-controlled hydraulic microdrive through the vestibular nuclei. The search stimulus was 0.5
Hz sinusoidal yaw rotation at 30 or 60 °/s peak velocity with a head-fixed target illuminated. After a candidate neuron was identified, based on auditory detection of modulation of the discharge rate with rotation, the response of the neuron to rotation while the animal fixated a head-fixed target was recorded at 0.5 Hz, 60 °/s peak velocity. Next, the neuron was recorded during untargeted saccades, during eye movements to horizontal targets (spaced every 5°, for ± 20°), and during pursuit to horizontal targets moving sinusoidally at 0.2 Hz (±23°) or 0.5 Hz (±13°). Dynamic sensitivity in the pitch plane was tested, and responsive neurons were excluded. Yaw and eye-movement sensitive neurons were tested using two series of rotational stimuli, all with head-fixed spatial targets.

Responses were recorded to stimuli with peak velocity of 60°/s at 0.5, 1.0, 2.0, 0.1, 0.2, 0.3, 0.8 and 1.5 Hz, in that order. An escalating velocity series of rotations was performed at a frequency of 0.5 Hz starting with a peak velocity of 7.5 or 15 °/s, and then increasing the peak velocity to 15, 30, 60, 90, 120, 150, 180, and 210°/s. For all of the rotation trials, 20 to 60 seconds of data were collected. All recordings were performed in a dark room with a head-fixed target moving with the animal to suppress the VOR, and the monkey was rewarded for maintaining fixation of the target.

Data analysis:

Many of the details of the analysis techniques have also been reported previously (Newlands et al., 2009). All data were analyzed off-line using a suite of custom programs written in LabVIEW. Of particular interest for this study are
additional techniques used to account for eye-movement sensitivity in analyzing vestibular sensitivity in PVP and EHV neurons. The raw neuronal data, which was recorded and stored at 40 kHz, was converted to discharge trains using a combination of feature analysis and time-amplitude window discrimination, as previously reported (Newlands et al., 2009). The resulting discharge train was converted into a digital instantaneous firing rate (IFR) trace. At each time point (every msec), the value of the IFR trace was assigned as the inverse of interspike interval between the spike immediately before and immediately after that particular time point. Digital differentiation of the eye position trace was used to generate eye velocity and subsequently eye acceleration. An eye acceleration filter was used to desaccade the eye traces. All data collected during saccades was ignored for analysis. For eye position data collected during eye movement saccades between horizontal targets, eye position was plotted against IFR. These points were then fit with a least-squares linear fit to determine the eye position sensitivity of the neuron. To determine eye velocity sensitivity, eye velocity was plotted as a function of the instantaneous firing rate during smooth pursuit of a target, but only after subtracting the eye position influence from the IFR trace based on the sensitivity to eye position calculated using the eye fixation data (Tomlinson and Robinson, 1984). In this way, both eye position and eye velocity sensitivity for each of the eye-movement-sensitive neurons was determined. Neurons that did not show reproducible oculomotor sensitivity were not included in the results.
To determine head velocity sensitivity, the data was first desaccaded, eliminating the influence of high velocity corrective saccades due to the VOR. When possible, only portions of data in which the animal was suppressing the VOR by following the head-fixed target during rotation were considered. Adequate fixation was defined as keeping the eye within 5° of the target and the eye velocity under 33% of the head velocity. These definitions of suppression were somewhat loose, but fixation on the target was difficult to impossible for the animals at peak velocities above 120°/s and at 2 Hz frequency, and excellent suppression at peak velocities above 60°/s and at frequencies above 0.5 Hz were rarely obtained in our animals. As described above, the portion of the response that was presumed to be responses to the eye position and velocity was subtracted using the sensitivities determined from the eye fixation and pursuit data. These adjustments assumed linear combination of eye and head movement sensitivity, as has been done by other investigators (Tomlinson and Robinson, 1984; Scudder and Fuchs, 1992; McConville et al., 1996). The resulting spike train was averaged over all of the collected rotational cycles and fit using a Levenberg-Marquardt least-squares algorithm. The vestibular sensitivity (gain) and the phase of the response were determined by comparing the amplitude and timing of the peaks of the fitted response curve and stimulus sinusoid. The peak of the response was defined as the peak of the curve fitting the response. Inhibitory and excitatory half-cycle sensitivities (gains) were also determined. This calculation was performed by first using the phase relationship
between the stimulus and response that were determined using the whole
sinusoid fits and adjusting the timing of the response such that the peak
response and peak stimulus were temporally aligned. The stimulus and
response were then plotted against one another on an x-y plot, such that the
excitatory direction was positive and the inhibitory direction was negative. The
excitatory and inhibitory directions were then fit with independent regression lines
that were forced to intersect the y-axis at the same value. The intersection value
is the firing rate around which the cell’s activity was modulated and is termed the
bias firing rate.

For all neurons, it was determined that the firing response was correlated
with the stimulus by calculating the Rayleigh coefficient, a measure of correlation
between the stimulus and response, and only data for which the calculated
Rayleigh coefficient was statistically significant at the p<0.05 level (Mardia, 1972)
was used. Other statistical comparisons also utilized a p<0.05 level for
significance (SPSS 14.0 for windows).
RESULTS:

Data reported here were derived from recordings of 66 PVP and 41 EHV neurons. Eight PVP and six EHV neurons were recorded at only one frequency and peak velocity (0.5 Hz and 60°/s); the other 93 cells were studied using at least one additional frequency at 60°/s or additional peak velocity at 0.5 Hz. For three PVP and six EHV neurons, the eye velocity and position were not well characterized, either due to apparently very nonlinear relationships between eye position or velocity and firing rate or due to poor behavior during visual tasks. The eye movement characteristics of the remaining neurons are summarized in Table I. All of the PVP neurons had eye position sensitivity in the opposite direction to the head velocity sensitivity and all of the EHV neurons had eye velocity sensitivity in the same direction as the head velocity sensitivity. Some PVP neurons also demonstrated eye velocity sensitivity in the same direction as the eye position sensitivity and some EHV neurons also had eye position sensitivity in the same direction as the eye velocity sensitivity.

Examples of a PVP and an EHV neuron are shown in Figure 1. The left column demonstrates a PVP neuron that has eye position sensitivity to the right but does not completely pause for saccades. This neuron was not eye velocity sensitive; the response is in phase with position during the smooth pursuit task. This neuron responded in phase with head (chair) movement to the left. In contrast, the right column of the figure shows responses of an EHV neuron that had no consistent eye position sensitivity but did have eye velocity sensitivity to
the left. This neuron also responded to yaw rotation to the left when the eye movement was suppressed and thus the VOR cancelled.

Eye velocity related discharge greatly influences the response of EHV neurons to rotation. For example, for a typical type II EHV cell recorded when the animal does not suppress the VOR, the influence of the eye movement is often greater than the drive from the labyrinth (Scudder and Fuchs, 1992; McFarland and Fuchs, 1992). In calculating the sensitivity to head movement, the eye velocity correction factor could dominate the actual vestibular response if the VOR were not suppressed. For neurons with eye position sensitivity, however, arithmetic correction for trials with poor VOR suppression does not have much impact on the analysis used here, because the eye position generally stays within 20° of the midline. For consistency, the sensitivity of all 86 neurons in this study for which the eye movement sensitivities were known was determined after correcting for those eye position and/or velocity sensitivities.

The sensitivity reported in this study is that attributable to head movement after removing the calculated estimate of eye movement influence on firing rate. For data periods with good behavior, the adjustment for eye movement contributions to firing is small. For the 9 neurons in which adequate eye movement data was not collected, only trials with well suppressed VOR were included and no eye correction was performed. In some trials, the animal alternated between suppression and no suppression. One such example is shown in Figure 2, in which suppression was briefly lost then regained in one trial. The correction for eye velocity, as seen in this example, gives a more consistent estimate of the
head velocity sensitivity neuron than would be obtained without correcting for the
eye velocity. In several cases, using adjustments for eye position and/or eye
velocity, it was possible to compare the well-suppressed data to poorly
suppressed data within single trials. In all such cases for which there was a
combination of well suppressed and poorly suppressed data in one trial, the
gains calculated for well-suppressed data and poorly suppressed portions were
always within 20%. Whenever possible, only well-suppressed portions of the
data were utilized in calculating the gains and phases.

Figure 3 demonstrates the responses of the population of neurons as a
function of frequency for a constant peak velocity of 60°/sec. Figure 3A
demonstrates Bode plots for type I (head velocity sensitivity in phase with
ipsilateral head rotation) and type II (head velocity sensitivity in phase with
contralateral head rotation) PVP neurons. For reference, the average responses
of NEM cells from Newlands et al. (2009) are shown. Type I PVP neurons were
more sensitive to head rotation than were type I NEM neurons (ANOVA,
\(F_{(1,956)}=30.9, p <0.000001\)). Type II PVP neurons were also more sensitive than
type II NEM neurons (ANOVA, \(F_{(1,488)}=157 p <0.000001\)). Unlike NEM neurons,
in which type I neurons were more sensitive than type II neurons, the population
of type I and type II PVP neurons had similar sensitivities (ANOVA, \(F_{(1,387)}=1.3, p
= 0.51\)). Type II PVP neurons showed high-pass dynamics with higher gains at
higher frequencies (ANOVA, \(F_{(7,187)}=2.38, p = .024\), whereas for type I PVP
neurons the change in gain with increasing frequency was not significant
(ANOVA, \(F_{(7,193)}=0.73, p = 0.64\)). The sensitivity for type I PVP neurons at 2 Hz
was 0.99 ± 1.02 (S.D.) sp/s°/s and at 0.1 Hz was 0.64 ± 0.56 (S.D.) sp/s°/s. For type II PVP neurons tested at 2 Hz the sensitivity was 0.95 ± 0.66 (S.D.) sp/s°/s, and at 0.1 Hz the sensitivity was 0.52 ± 0.27 (S.D.) sp/s°/s.

Figure 3B demonstrates Bode plots for EHV cells. In these plots, trials in which VOR suppression was adequate are distinguished by triangular symbols. In these trials, only portions of data for which the eye position stayed within 5° of the target and the eye velocity was <30% of head velocity were included. The sensitivity of type II (also known as eye-contra) EHV neurons was greater than the sensitivity of type I (also known as eye-ipsi) EHV neurons at all frequencies (ANOVA, \( F_{(1,207)} = 42, p < 0.000001 \)). Unlike the PVP neurons or NEM neurons, the sensitivity of EHV neurons did not appear to change with frequency (Type I ANOVA, \( F_{(7,115)} = 1.09, p = 0.38 \); Type II ANOVA, \( F_{(7,76)} = 0.22 p = 0.98 \)), though it is possible that the method used to calculate head sensitivity influenced this finding. Above 1 Hz, the animals were unable to consistently suppress their VOR, so it is possible that the correction for eye velocity served to flatten the calculated sensitivity of the neurons to head movements.

Of most interest for the purpose of this study was the effect of increasing velocity while holding frequency constant. Figure 4 demonstrates the responses of our population of neurons at varying peak velocities from 7.5 to 210°/s with the frequency of rotation fixed at 0.5 Hz. In particular, this analysis addresses the scalability, or homogeneity, of the responses. Linear responses are linearly scalable, such that when the input is increased or decreased, the output changes proportionally. Stated differently, the sensitivity (input/output) of the system is
constant for a homogeneous system. Figure 4A shows the sensitivity to yaw rotation for the PVP neurons, with type I and type II neurons displayed separately. The average responses of NEM neurons (Newlands et al., 2009) are again shown for reference. As with the NEM neurons, the sensitivity of the cells decreased with increasing peak velocity of rotation both for type I (ANOVA, $F_{(8,131)}=3.59$, $p = 0.0009$) and type II PVP neurons (ANOVA, $F_{(8,151)}=12.4$, $p <0.00001$). So, as was the case for the NEM neurons, the PVP responses to yaw rotation were not linearly scalable. Figure 4B shows the responses of the EHV neurons with increasing peak velocities, and the average responses of NEM neurons under the same conditions are shown for comparison. As in Figure 3, responses of EHV cells that were not well suppressed are distinguished from those for which suppression was present. In comparison to the NEM and PVP neurons, EHV neurons demonstrated less of a decrement in sensitivity to yaw rotation with increasing peak velocity of rotation. However, as with PVP and NEM neurons, there was a statistically significant decrease in response sensitivity with increasing rotational velocity for type I (ANOVA $F_{(8,97)}=3.43$, $p = 0.0016$) but not type II (ANOVA, $F_{(8,60)}=1.26$, $p = 0.28$) EHV neurons.

In addition to the analysis described above, the responses of PVP and EHV neurons were compared across peak velocities at 0.5 Hz without taking into account eye movement contributions to firing. As when the influence of eye movements where accounted for, PVP responses in this analysis also showed decreasing gains with increasing peak velocities (type I - ANOVA, $F_{(8,131)}=3.94$, $p = 0.0003$ and type II - ANOVA, $F_{(8,151)}=4.34$, $p = 0.0001$). The results reported
for EHV neurons were also similar, even if we did not account for eye movement contributions to firing rates. For type I EHV neurons, whether all trials were used in the analysis (ANOVA, $F(8, 97) = 7.07$ $p = 0.000003$) or only those trials with VOR suppression (ANOVA, $F(5, 59) = 4.18$ $p = 0.03$ (no data at 150, 180 or 210 °/s included)), the sensitivity to yaw rotation decreases with increasing velocities at 0.5 Hz. For type II EHV neurons, whether all trials were considered (ANOVA, $F(8, 60) = 0.60$ $p = 0.77$) or just those with VOR suppression (ANOVA, $F(5, 42) = 0.68$ $p = 0.64$), sensitivity was not significantly different across the range of peak velocities tested.

Other nonlinear response types that have been described in the vestibular system include rectification (silencing during the inhibitory portion of the cycle), saturation (a ceiling effect where the firing rate of the neuron will not exceed a certain level), skew deviation (an asymmetry in the distribution of spikes during a cycle, usually such that spikes arise earlier during excitatory portion of the cycle) and directional asymmetry (in which the neuron’s sensitivity is greater in one direction – generally the excitatory direction – than in the other direction). An example of a silenced, or rectified, response is demonstrated in Figure 5A. This particular neuron, tested with a peak velocity of 180 °/s, was silenced for about half of the cycle. For the portion of the cycle when the cell was firing, the firing rate was slightly skewed. Some degree of skewing was present in many of the neurons.

Table II and III display the proportion of silenced (rectified) PVP cells at each frequency and peak velocity. Neurons with higher gains and lower baseline
firing rates are more likely to silence during a portion of the cycle. The percentage of neurons that showed rectification was not a function of frequency or peak velocity for either type I or type II PVP neurons (Chi square, p>0.05), although at rotational velocities below 15°/s, no rectification was seen. Thus, an important consequence of lowered sensitivity of PVP responses at high velocities of rotation is avoidance of rectification. Rectification of EHV neurons probably rarely occurs in the absence of any suppression of the VOR, because the eye velocity sensitivity in the same direction of head sensitivity. However, we did not record the responses of cells in the absence of a target, so the existence of rectification in EHV neurons was not appropriately explored.

A possible natural solution to reduce silencing at higher peak velocities would be to increase the baseline firing rate around which the firing is modulated. Such a relationship has been reported in the vestibular nuclei of cats after unilateral labyrinthectomy (Heskin-Sweezie et al., 2007). As previously reported for NEM neurons (Newlands et al., 2009), the relationship between the mean firing rates during stimuli at different peak velocities and frequencies of PVP (values in Tables II and III) and EHV (Table IV) neurons was explored. There were no statistically significant differences in bias firing rates with changes in peak velocity for any of the cell types (type I PVP - ANOVA, F(8,131)=0.16, p = 0.99; type II PVP- ANOVA, F(8,151)=0.39, p = 0.93; type I EHV - ANOVA F(8,97)= 0.14 p = 0.99; type II EHV - ANOVA, F(8,60)=0.63 p = 0.75).

All neuronal responses were examined for evidence of saturation. A saturated response is illustrated in Figure 5B. This neuron’s response saturated
at a level near 110 sp/s°/sec through much of the excitatory portion of the cycle; this trial had a peak velocity of 180 °/s. Of the PVP neurons tested at 180 and/or 210°/sec, only the one in Figure 5B showed such saturation. For this neuron, saturation is reflected in the phase adjusted plot of IFR vs. head velocity shown in the right hand column of Figure 5B. For head velocities > 50 °/s, the firing rate did not increase.

Ewald’s second law states that vestibular responses are greater in the excitatory direction than in the inhibitory direction (Ewald, 1892). While Ewald originally stated this law in the context of vestibular responses (the VOR), subsequent investigation has found that the excitation caused by movement in the excitatory direction has a greater impact on vestibular nerve firing than does inhibition caused by movement in the inhibitory direction (Fernandez and Goldberg, 1971; Goldberg and Fernandez, 1971). A potential confounding variable in the results reported above is the possibility that by fitting the entire cycle, we might conclude that the sensitivity of the neuron generally declines with increasing peak velocity, when in fact the effect is a result of a limitation in the neuron’s response in one direction (potentially the inhibitory direction). To investigate the possibility that the relationship described here between sensitivity and peak stimulus velocity was primarily a result of changes in either the excitatory or inhibitory direction, the sensitivity was also calculated independently for each direction. For PVP neurons, the relationship between peak velocity and sensitivity in the excitatory direction and the inhibitory direction is shown independently in Figure 6. As shown in Figure 6A, sensitivity in the excitatory
direction is generally greater than in the inhibitory direction for both type I and type II neurons. As peak velocity increases, both the inhibitory and excitatory sensitivities decrease. In Figure 6B, the relationship between the excitatory and inhibitory sensitivities at each peak velocity are plotted against one another independently for type I and type II neurons. The slope of the line fitting the data points for type I neurons is 1.27 and for type II neurons is 1.23 and the relationship between excitatory and inhibitory gains lie along this line for each velocity tested. These data demonstrate that for PVP neurons, the excitatory direction input elicits a 23-27% larger response than does the inhibitory direction input, and the relationship between inhibitory and excitatory sensitivities is independent of the peak velocity over the population of neurons recorded.

There are a number of alternative relationships between input to and output from a system other than a linear relationship. One such relationship is a power-law relationship such that the output of the system (y) is related to the input of the system (x) by a relationship such as $y = bx^a$. Power law relationships are commonly evoked in perceptual relationships, including vestibular perception (Mallery et al., 2010). For this reason, the power law fit to the data was explored. If the output of the system, defined as the number of spikes generated at the peak of the excitatory response (calculated as peak firing rate = peak velocity * excitatory direction sensitivity to yaw rotation + bias) for each cell, was plotted as a function of the peak velocity of the stimulus, a power relationship would be represented on a log-log plot by a straight line with the slope of $a$. Figure 7 demonstrates such plots for PVP, EHV, and NEM neurons for the data presented.
in this paper and in Newlands et al. (2009). As demonstrated by Figure 7, the relationship between peak velocity of the stimulus and the output of the neurons is well described by a power-law relationship.
DISCUSSION:

This is the first study to systematically investigate the linearity of the relationship between vestibular stimulation and discharge rate in eye-movement-related vestibular neurons in the vestibular nuclei. In these experiments, it was demonstrated that PVP neurons and type I EHV neurons, like NEM neurons, have lower sensitivities to sinusoidal yaw rotation at higher peak velocities of stimulation. For type II EHV neurons, there is no statistically significant relationship between peak velocity and sensitivity. Other nonlinearities, such as skew, rectification, saturation, and excitatory-inhibitory asymmetries, are not significantly impacted by peak velocity.

Comparison to other studies of PVP and EHV neurons:

A number of authors have reported on the properties of PVP and EHV neurons over the last 40 years (Fuchs and Kimm, 1975; Scudder and Fuchs, 1992; Chubb et al., 1984; Keller and Kamath, 1975; King et al., 1976; Pola and Robinson, 1978; McCrea et al., 1980; Miles 1974; Keller and Daniels 1975; Fuchs et al., 2005; Roy and Cullen, 2003; Chen-Huang and McCrea, 1999a,b; McFarland and Fuchs, 1992). Additionally, Lisberger’s lab has extensively and elegantly studied floccular target neurons (Lisberger et al., 1994a,b; Ramachandran and Lisberger, 2008; Lisberger and Pavelko, 1988; Broussard and Lisberger, 1992), which are likely the same as, or at least an overlapping population with, EHV cells (Lisberger et al., 1994b). Comparable measurements to some of the results presented here have been published in macaques for eye-movement-related horizontal-rotation-sensitive neurons, which permits
comparison with previous studies. Scudder and Fuchs found average eye position and eye velocity sensitivities of PVP neurons during pursuit to be $1.73 \pm 0.93$ sp/s/° and $0.53 \pm 0.38$ sp/s/°/s, respectively. Eye position sensitivity during saccades was found to be $8.2\%$ greater than found during pursuit. The head sensitivity for PVP neurons in their study was $1.04 \pm 0.85$ sp/s/°/s with a phase of $10.7 \pm 10.4$ ° at 0.5 Hz and ~ $30$ °/s peak velocity, which is similar to the findings reported here at 0.5 Hz and 30 °/s peak velocity for all neurons (type I and type II) of $0.93 \pm 0.54$ sp/s/°/s and $19 \pm 10.4$ °. McConville et al. (1996) report a sensitivity in 24 PVP neurons of $1.03 \pm 0.01$ (SE) in their on-axis, far-target group, which was also tested with 0.5 Hz, ±10° excursion (31.4 °/s peak velocity). Fuchs et al. (2005) tested 19 PVP neurons at 0.4 to 0.5 Hz and peak velocities of 25.1-34.4 °/s and found sensitivities of $0.80 \pm 0.39$ (S.D.) sp/s/°/s with a phase of $10.6 \pm 7.9$ (S.D.) °. The population of PVP neurons described here appears similar, therefore, to those in the published literature.

For EHV cells, Scudder and Fuchs (1992) reported an average eye velocity sensitivity of $2.2 \pm 2.1$ sp/s/°/s (range of 0.64 to 12.7 sp/s/°/s) and average position sensitivity of $2 \pm 2$ sp/s/° with a range of -2 to 7.2 sp/s/°. These sensitivities are higher than observed in the current study. In Scudder and Fuchs (1992), the average head velocity sensitivity of the neurons at 0.5 Hz and 31.4 °/s peak velocity was $1.06 \pm 0.88$ sp/s/°/s, which was greater than the sensitivity of $0.63\pm 0.35$ sp/s/°/s described here. In the medial vestibular nucleus and NPH, McFarland and Fuchs (1992) noted eye velocity sensitivity of EHV neurons $1.3 \pm 0.76$ sp/s/°/s. In that study, the authors noted little difference between the eye
position sensitivity to static targets \((1.5 \pm 1.1 \text{ sp/s/°})\) and eye position sensitivity during pursuit \((1.7 \pm 1.1 \text{ sp/s/°})\). McFarland and Fuchs (1992) did not directly report the gain of the EHV neurons during VOR suppression, but reported that the pursuit sensitivity was 1.5 times the vestibular sensitivity, which implied a rotational sensitivity of \(~0.8 \text{ sp/s/°/s}~\) at 0.5 Hz, 31.4 °/s peak velocity. Roy and Cullen (2003) found eye position sensitivity for EHV cells to be \(1.5 \pm 1.9 \text{ sp/s/°}\) and the smooth pursuit position sensitivity to be \(1.0 \pm 1.1 \text{ sp/s/°}\) and the eye velocity sensitivity during smooth pursuit to be \(0.7 \pm 0.8 \text{ sp/s/°/s}~\) (for target velocities of 40 °/s at 0.5 Hz), which are closer to the values in this study. In their animals, measuring responses during VOR suppression at 40 °/s peak velocity and 0.5 Hz, the sensitivity re: velocity for type I and II EHV neurons was \(0.7 \pm 0.5 \text{ sp/s/°/s}~\), similar to our value \((0.63 \pm 0.35 \text{ sp/s/°/s})~\) at 30 °/s peak velocity. Overall, these comparisons suggest that our data likely reflect very similar neuronal populations to those previously described in this species, though differences due to selection biases, sampling errors, experimental protocol differences, and analysis techniques undoubtedly contribute to differences.

There is little data in the macaque literature to compare to the results presented here that investigated the effect of varying frequency on PVP and EHV responses. Ramachandran and Lisberger (2008) measured horizontal sinusoidal rotational responses of PVP and EHV neurons over a frequency range of 0.5 to 50 Hz in macaques with small (<20 °/s) peak velocities of rotation. Over the frequency range in common between the two studies (0.5 to 2.0 Hz) their data shows subtle high-pass dynamics, similar to the results presented here.
Similarly, Dickman and Angelaki (2004) explored the frequency response of 13 eye movement sensitive central neurons in rhesus macaques between 0.02 and 1 Hz. In the overlapping frequencies of 0.1 to 1.0 Hz between the two studies, they did not report increasing gain with increasing frequency. Even less data is available on the effect of increasing the amplitude of stimulation on the responses of secondary vestibular neurons. Roy and Cullen (2003) recorded EHV neurons at 0.5 Hz at peak velocities of 40 °/s and 80 °/s while suppressing the VOR and noted no difference in velocity gain. Roy et al. (2003), in a preliminary report of neurons in the direct VOR pathways, described a soft saturation with increasing peak velocities >200 °/s and inhibitory cut off at peak velocities below 100 °/s. Thus, their findings differ from those reported here in which saturation was not observed at higher velocities, but rather the response gain of neurons changed throughout the velocity range tested.

Limitations of the current study:

One difficulty in our study is that the animal's ability to suppress the VOR is a function of the stimulus velocity and frequency and is variable between animals. For all stimuli, an attempt was made to have the animals suppress VOR, but in circumstances for which the animal was unable to do so, mathematical corrections were used in an attempt to account for the eye contribution to the response. The methodology must be considered for both PVP and EHV neurons for slightly different reasons.

The characteristics of PVP neurons responses in relationship to eye velocity, eye position and head velocity under conditions of VOR with fixation,
VOR without fixation, fixation of static targets, and pursuit of moving targets has been wrestled with before (Chubb et al., 1984; Cullen and McCrea, 1993; McConville et al., 1996; Scudder and Fuchs, 1992; Tomlinson and Robinson, 1984), and other investigators have demonstrated that eye position, eye velocity, and vestibular sensitivities of PVP neurons can be related algebraically. Chubb et al. (1984) compared the responses of vertical-canal-related PVP neurons under conditions of vertical pursuit, VOR in the dark, and VOR suppression and found that the VOR in the dark response was best described by adding the response during suppression to the expected response from the eye movements. Scudder and Fuchs (1992) adjusted for eye movements using a similar mathematical correction to calculate the sensitivity of PVP neurons during VOR suppression and during VOR with a space-fixed target. In the current study, the slope of the discharge rate-position curve during fixation of static targets was used to determine the eye position sensitivity of PVP cells and this slope was applied to the other behavioral paradigms. Similarly, the slope of the discharge rate – eye velocity relationship during smooth pursuit, after adjusting the discharge rate for the eye position determined from target fixation, was used to determine the eye velocity sensitivity of these neurons. Next eye and position sensitivity corrections during head rotations were calculated. Tomlinson and Robinson (1984), did exactly this in examining the relationship between pursuit and VOR responses of vertical PVP neurons in macaques. The data from the studies described above support the appropriateness of the analysis method applied in this study. For PVP neurons, whether eye position was accounted for
had only modest effects on the head movement sensitivity calculated for the most vigorous head movements. The primary conclusion of this study, that sensitivity of PVP neurons depended on the peak head velocity of the stimulus, was not affected by whether eye position sensitivity of the neuron accounted for algebraically.

The best way to account for the eye sensitivity component of PVP responses during rotation is not entirely clear, and in the case of EHV cells, the problem is both more difficult and has a stronger influence on the results. As with PVP neurons, attempts to make algebraic sense of EHV neurons have been made in the past. Cullen et al. (1993) describe smooth pursuit cells in squirrel monkeys for which they argue the vestibular signal algebraically combines with eye movement signals. Scudder and Fuchs (1992) found that addition of the VOR suppressed response of EHV neurons to the pursuit sensitivity by linear vector addition over predicted the discharge rate during VOR with a space-fixed target by 11%. Though this estimation seems reasonable, they note that during stable-in-the-world gaze, two larger signals (the eye sensitivity and head velocity) are subtracted to produce a smaller signal, so measurement errors are increased. Angelaki et al. (2001) and Meng et al. (2005) were careful to match their visual (pursuit) and vestibular (translation or rotation) protocols so the eye movements were similar in their studies of sensorimotor processing in the VOR. They found that neural modulation under world stable gaze conditions could be satisfactorily predicted from a linear superposition of smooth pursuit and VOR cancellation responses for most EHV and PVP neurons. Roy and Cullen (2003)
also reported that, at 0.5 Hz and 40 or 80 °/s peak velocity, responses of EHV neurons during VOR in the dark can be reasonably well predicted by combining the responses during pursuit and those during VOR with suppression. As with PVP neurons, regardless of whether the eye movement influence on firing rates was accounted for, the primary finding of the study that the vestibular sensitivity of type I, but not type II, EHV neurons depended on the peak velocity of rotation.

**Implications for signal processing in the vestibulo-ocular reflex:**

Type I (or eye-ipsi) EHV neurons project to the spinal cord and may not be involved in the VOR (Boyle, 1993; Scudder and Fuchs, 1992). PVP and type II (eye-contra) EHV neurons are believed to be the most important cells in the vestibular nuclei for conveying vestibular information mediating the VOR (Scudder and Fuchs, 1992). Horizontal type I PVP cells project primarily to the contralateral abducens nucleus as excitatory neurons (Scudder and Fuchs, 1992; McCrea et al., 1987). Type II EHV neurons provide inhibitory input to the ipsilateral abducens nucleus (Scudder and Fuchs, 1992). This role of type II EHV cells in direct VOR pathways is complicated because they are inhibited during ipsilateral rotation but receive ipsilateral afferent excitatory input (Scudder and Fuchs, 1992; Broussard and Lisberger, 1992). These neurons receive competing excitatory commissural input from the contralateral labyrinth via the contralateral vestibular nuclei that is excitatory during contralateral rotation (Broussard and Lisberger, 1992). A large subset of EHV cells is floccular target neurons. Floccular target neurons receive inhibitory ipsilateral floccular input, the
sign and strength of which is modifiable and believed to be the basis of motor
learning in the horizontal VOR (Lisberger et al., 1994b,c).

As one considers the proposed roles for PVP and EHV neurons in the
VOR, the findings in this study are not that surprising. The rotational VOR has a
remarkably large linear dynamic range. In macaques, the horizontal rotational
VOR is essentially linear to frequencies of at least 25 Hz, accelerations of at least
15,000 °/s² (Huterer and Cullen, 2002; Ramachandran and Lisberger , 2005) and
velocities of 800 °/s (Tomlinson, 1990). In order to convey a signal with fidelity
over such a wide range, some form of dynamic compression is required to avoid
the limitations of neural systems, including rectification and saturation. While it is
counterintuitive that the interneuron in the VOR must be nonlinear to allow a
linear VOR over a wide range, this is likely the case.

Considering the abducens nucleus, one might also expect nonlinear
responses similar to those of PVP neurons. Abducens neurons have not been
similarly tested in primates with varying amplitudes of sinusoidal rotation, but
similar nonlinearities have been reported under other experimental conditions. In
rabbits, Stahl and Simpson (1995a,b) note that the sensitivity to rotational
velocity decreases for both vestibular nucleus and abducens nucleus neurons
with increasing peak amplitudes of rotation at 0.2 Hz. They argue that this
nonlinearity is compensated for in the abducens to eye movement transformation
to create a linear eye movement. In macaques, Fuchs et al. (1988) found that
the position and velocity sensitivities of abducens motor neurons were frequency
dependent during both smooth pursuit and VOR with a space-fixed target.
Because these authors held the amplitude of the target or the chair movement constant (± 10°), the velocity of the stimulus was also varied. Sylvestre and Cullen (1999) found that the relationship between the responses of abducens motor neurons and eye position and velocity was not constant but that as velocity increased, the position and velocity coefficients describing the discharge rate of individual neurons became smaller for a range of horizontal eye movement types (saccades, pursuit, slow phase VOR). Thus, the relationship between peak velocity and discharge rate of neurons for PVP and abducens motor neurons during the slow phase of VOR are likely quite similar (see Sylvestre and Cullen, 1999, figure 14). This amplitude nonlinearity is likely not resolved until the level of the mechanical movement of the eye (Stahl and Simpson, 1995b). This nonlinearity may be compensated for both with the viscoelastic properties of the eye plant and by antagonist eye muscles (Sylvestre and Cullen, 1999; Quaia et al., 2009).

Strong evidence exists for two pathways in the horizontal VOR. The first has been named the unmodifiable pathway, through which vestibular signals in irregular afferents modulate ipsilateral PVP neurons that project to the contralateral abducens nucleus. The second pathway is a modifiable pathway, through which vestibular signals on regular afferents modulate ipsilateral type II (or eye contralateral) floccular target neurons (EHV type II neurons), which inhibit ipsilateral abducens neurons (Ramachandran and Lisberger, 2005, 2006, 2008). During adaptation, the eye-to-head-velocity ratio on EHV neurons changes to fine tune the VOR. For such a purpose, a linear relationship between head
velocity and discharge rate might be more appropriate. At high head velocities, the vestibular drive to EHV cells is offset by the eye velocity influence which is opposite in sign. These cancelling influences might theoretically allow EHV cells to avoid nonlinearities with vigorous stimuli without requiring a change in sensitivity with peak velocity. This functional role of eye-contra (type II) EHV neurons is consistent with these neurons being the only ones in our study that do not show a statistically significant decline in sensitivity with increasing peak velocities of head rotation.

Similarities to other sensory systems:

The findings in this report and in Newlands et al. (2009) suggest that regardless of eye movement sensitivity, central vestibular neurons exhibit multiple nonlinearities in their responses to natural stimuli. These findings are consistent with other sensory systems. The challenge of extending the dynamic range of the system while maintaining sensitivity to small differences in level at very low levels of stimulation is ubiquitous in sensory physiology. Similarly, all sensory systems face limitations inherent in neuronal coding of information. The large dynamic range of sensory perception in relation to the relatively small dynamic range of neurons requires amplitude compression, which is a unifying theme in sensory physiology. In other sensory systems, nonlinear coding of intensity begins at the level of the sensory receptors (reviewed by Singer et al, 2009, for audition, olfaction, and vision). Mechanisms for extending the dynamic range of responses also include dynamic range adaptation, observed at the level
of the auditory nerve (Wen et al., 2009). Similar mechanisms of amplitude
compression and dynamic range adaptation are not apparent in the vestibular
nerve. The classic studies of Fernandez and Goldberg evaluated the linearity of
semicircular canal afferents to increasing amplitudes of rotation. Their data
demonstrated that the gains and phases of semicircular canal afferents in
squirrel monkeys are almost constant as peak velocity varies over a range of 0 to
250°/s at 0.05 Hz (Fernandez and Goldberg, 1971). Similarly, recording from
both regular and irregular afferents in chinchillas using velocity ramps of up to
875°/s, Plotnick and Goldberg estimate that the linear range in the excitatory
direction for regular afferents to exceed 1000°/s and for irregular afferents to
exceed 500°/s (Plotnick et al., 1999). Comparison of data collected in the
vestibular afferents to our data suggests that the nonlinear gain of the central
neuronal responses is not a reflection of the physiology of the semicircular canal
afferents. Similarly, data from slice preparations demonstrate linear input-output
relationships for central vestibular neurons to direct current injection (du Lac and
Lisberger, 1995) or to synaptic inputs (Bagnall et al., 2008). However,
preparation of brainstem slices disrupts cerebellar and brainstem circuits
involving central vestibular neurons. These circuits might be necessary for the
compressive nonlinearity seen in our recordings from awake, behaving primates.
Thus, the vestibular system may differ from other sensory systems in that the
amplitude compression occurs primarily centrally rather than peripherally.
The findings presented here demonstrate that amplitude compression
consistent with a power-law relationship between the excitatory stimulus velocity
and the increase in discharge rate of individual neurons is present in the vestibular nuclei. The data presented here do not address mechanisms. However, despite motor signals being present on central vestibular neurons related to eye movements, the vestibular nuclei are primary sensory nuclei, and thus the existence of the fundamental sensory mechanism of amplitude compression is not surprising. Mechanisms whereby intensity information coded in the vestibular nuclei is subsequently used for threshold detection, linear motor reflexes, and perception of movement are open areas for future investigation.
ACKNOWLEDGEMENT:

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**FIGURE LEGENDS:**

**Figure 1:** Two example neurons, one PVP (left column) and one EHV (right column) from the same animal. Responses to target steps of ± 20° (first rows), sinusoidal target (smooth pursuit) modulated at peak velocity of ± 40°/s at 0.5 Hz (second rows), and head fixed target during sinusoidal head rotation (VOR cancellation) with yaw rotation with peak velocity of ± 60°/s at 0.5 Hz. Epos – eye position relative to the head position. Evel – eye velocity relative to the head velocity. IFR – instantaneous firing rate averaged over time bins of 20 ms. Target – target position in space. Hvel – Head velocity in space.

**Figure 2:** Example EHV neuron during a period of fair suppression of the VOR that deteriorates to poor suppression of the VOR in the middle two cycles. Epos – eye position, Evel – eye velocity, IFR – instantaneous firing rate binned every 20 ms (gray). Black line is IFR adjusted for eye velocity sensitivity (1.5 sp/s°/s). Hvel – head velocity.

**Figure 3:** Bode plots (sensitivity to yaw rotation vs. frequency of yaw rotation; log-log plot) of A) PVP and B) EHV data holding peak velocity constant at 60°/s. Symbols (open circles and open triangles) represent trials in individual neurons. Square symbols are average values. NEM averages shown for comparison from Newlands et al. (2009) – Gray square symbols.
Figure 4: Plots of sensitivity to yaw rotation vs. peak velocity of yaw rotation (7.5 – 210°/s) at 0.5 Hz of PVP data (A) and EHV data (B). NEM averages shown for comparison from Newlands et al. (2009) – Gray square symbols.

Figure 5: Examples of PVP responses showing nonlinearities. A: rectified and slightly skewed response. B: Saturation (the neuron response shows flattening after an initial ramp up of firing with rotation in the excitatory direction). Peak rotational velocities 180°/s. Hvel – head velocity. All responses shown are averaged over 10 to 20 cycles. IFR binning at 20 msec. Phase adjusted IFR vs. head velocity.

Figure 6: A: Sensitivity to yaw rotation for excitatory and inhibitory half-cycles vs. peak velocity for type I and type II PVP neurons. Half-cycle sensitivity calculated as the slope of the line fitting phase-adjusted IFR vs. instantaneous head velocity for either the inhibitory or excitatory half-cycle. B: Excitatory vs. inhibitory sensitivity across all of the peak velocities at 0.5 Hz.

Figure 7: Log-log plot comparing the effect of the peak velocity of rotation on the output of either type I or type II PVP (A), EHV (B) and NEM (C) neurons. In all six plots, using all the data, a power function (y = b*x^a) is fit using a least squares algorithm (dotted line). The best fit equations are: Type I PVP – y=48.4*x^{0.201}, Type II PVP – y=40.0*x^{0.224}, Type I EHV – y=14.3*x^{0.416}, Type II EHV – y=27.2*x^{0.348}, Type I NEM – y=31.0*x^{0.210}, Type II NEM – y=20.4*x^{0.220}. 
REFERENCES:


Ewald JR. Physiologische Untersuchungen ueber das Endorgan des Nervus Octavus. Wiesbaden: Bergmann, 1892.


### Table I

Eye movement characteristics of neurons

<table>
<thead>
<tr>
<th></th>
<th>Eye position</th>
<th></th>
<th>Eye velocity</th>
<th></th>
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<tr>
<td>PVP type I</td>
<td>0.96 ± 0.76</td>
<td>35</td>
<td>0.20-2.5</td>
<td>0.51 ± 0.41</td>
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<tr>
<td>PVP Type II</td>
<td>0.66 ± 0.51</td>
<td>28</td>
<td>0.23-2.0</td>
<td>0.32 ± 0.19</td>
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<tr>
<td>EHV Type I</td>
<td>0.59 ± 0.55</td>
<td>10</td>
<td>0.20-2.0</td>
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<tr>
<td>EHV Type II</td>
<td>0.80 ± 0.55</td>
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<td>0.21-2.0</td>
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Table II – PVP rectification by frequency

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<tr>
<th>Type</th>
<th>Freq</th>
<th>Amp</th>
<th>N total</th>
<th>N rectified</th>
<th>Bias ± SD all (sp/s)</th>
<th>Bias ± SD rectified (sp/s)</th>
<th>Bias ± SD not rectified (sp/s)</th>
<th>Sensitivity ± SD rectified (sp/s°/s)</th>
<th>Sensitivity ± SD not rectified (sp/s°/s)</th>
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<tbody>
<tr>
<td>I</td>
<td>0.1</td>
<td>60</td>
<td>18</td>
<td>4</td>
<td>88 ± 71</td>
<td>43 ± 49</td>
<td>101 ± 72</td>
<td>1.1 ± 1.0</td>
<td>0.51 ± 0.30</td>
</tr>
<tr>
<td>I</td>
<td>0.2</td>
<td>60</td>
<td>29</td>
<td>5</td>
<td>71 ± 45</td>
<td>39 ± 47</td>
<td>77 ± 43</td>
<td>1.1 ± 1.0</td>
<td>0.54 ± 0.29</td>
</tr>
<tr>
<td>I</td>
<td>0.3</td>
<td>60</td>
<td>21</td>
<td>4</td>
<td>81 ± 50</td>
<td>63 ± 57</td>
<td>85 ± 49.0</td>
<td>1.4 ± 1.5</td>
<td>0.53 ± 0.33</td>
</tr>
<tr>
<td>I</td>
<td>0.5</td>
<td>60</td>
<td>34</td>
<td>8</td>
<td>68 ± 34</td>
<td>47 ± 41</td>
<td>76 ± 30</td>
<td>1.1 ± 1.0</td>
<td>0.66 ± 0.35</td>
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<tr>
<td>I</td>
<td>0.8</td>
<td>60</td>
<td>27</td>
<td>6</td>
<td>79 ± 50</td>
<td>58 ± 56</td>
<td>84 ± 48</td>
<td>1.5 ± 1.5</td>
<td>0.66 ± 0.35</td>
</tr>
<tr>
<td>I</td>
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<td>60</td>
<td>30</td>
<td>8</td>
<td>75 ± 44</td>
<td>56 ± 61</td>
<td>79 ± 36</td>
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<tr>
<td>I</td>
<td>1.5</td>
<td>60</td>
<td>18</td>
<td>4</td>
<td>96 ± 51</td>
<td>83 ± 60</td>
<td>100 ± 50</td>
<td>1.9 ± 1.7</td>
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<tr>
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<td>23</td>
<td>5</td>
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<td>93 ± 44</td>
<td>1.7 ± 1.9</td>
<td>0.80 ± 0.54</td>
</tr>
<tr>
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<td>0</td>
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<td>0.52 ± 0.27</td>
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<td>26</td>
<td>6</td>
<td>61 ± 34</td>
<td>25 ± 16*</td>
<td>71 ± 30*</td>
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<tr>
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<td>23</td>
<td>6</td>
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<td>35 ± 30</td>
<td>71 ± 31</td>
<td>0.75 ± 0.54</td>
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<td>7</td>
<td>63 ± 40</td>
<td>34 ± 16</td>
<td>73 ± 41</td>
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<tr>
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<td>26</td>
<td>7</td>
<td>64 ± 31</td>
<td>44 ± 25</td>
<td>72 ± 31</td>
<td>1.0 ± 0.5</td>
<td>0.71 ± 0.42</td>
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<td>27</td>
<td>7</td>
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<td>44 ± 30</td>
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<td>0.65 ± 0.32</td>
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<tr>
<td>II</td>
<td>1.5</td>
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<td>5</td>
<td>69 ± 32</td>
<td>41 ± 12</td>
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<td>1.1 ± 0.6</td>
<td>0.77 ± 0.43</td>
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<td>2.0</td>
<td>60</td>
<td>26</td>
<td>9</td>
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<td>52 ± 27</td>
<td>80 ± 30</td>
<td>1.4 ± 0.9</td>
<td>0.72 ± 0.38</td>
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* T-test p<0.05 for rectified compared to non-rectified
Table III – PVP rectification by peak velocity

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<tr>
<th>Type</th>
<th>Freq</th>
<th>Amp</th>
<th>N total</th>
<th>N rectified</th>
<th>Bias ± SD all (sp/s)</th>
<th>Bias ± SD rectified (sp/s)</th>
<th>Bias ± SD not rectified (sp/s)</th>
<th>Sensitivity ± SD rectified (sp/s°/s)</th>
<th>Sensitivity ± SD not rectified (sp/s°/s)</th>
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<tr>
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<td>7.5</td>
<td>4</td>
<td>0</td>
<td>82 ± 42</td>
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<td>82 ± 42</td>
<td>N.A.</td>
<td>0.96 ± 0.62</td>
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<tr>
<td>I</td>
<td>0.5</td>
<td>15</td>
<td>4</td>
<td>0</td>
<td>72 ± 34</td>
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<td>0.84 ± 0.86</td>
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<td>80 ± 38</td>
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<td>0.48 ± 0.27</td>
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<td>0.43 ± 0.35</td>
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<td>0.40 ± 0.33</td>
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<td>67 ± 45</td>
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<td>N.A.</td>
<td>65 ± 40</td>
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<td>41 ± 26*</td>
<td>85 ± 33*</td>
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<td>0.59 ± 0.26</td>
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<td>37 ± 37</td>
<td>89 ± 36</td>
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<td>0.52 ± 0.23</td>
</tr>
<tr>
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<td>9</td>
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<td>49 ± 31*</td>
<td>93 ± 46*</td>
<td>0.41 ± 0.19</td>
<td>0.35 ± 0.12</td>
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<td>10</td>
<td>69 ± 44</td>
<td>44 ± 29*</td>
<td>98 ± 41*</td>
<td>0.38 ± 0.18</td>
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<td>58 ± 31</td>
<td>93 ± 49</td>
<td>0.41 ± 0.16</td>
<td>0.25 ± 0.12</td>
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* T-test p<0.05 for rectified compared to non-rectified
### Table IV: EHV Bias firing rate

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<th>Freq</th>
<th>Amp</th>
<th>Type I N</th>
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<th>Type II N</th>
<th>Type II Bias ± SD (sp/s)</th>
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<td>38 ± 23</td>
<td>10</td>
<td>72 ± 40</td>
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</tbody>
</table>
Step target

PVP

EHV

Sinusoid target (smooth pursuit)

Head-fixed target during sinusoid whole body rotation

Fig. 1
Fig. 2
Fig. 3
Fig. 4
**A** PVP response: rectified

![Diagram A](image)

**B** PVP response: rectified and saturated

![Diagram B](image)

**Fig. 5**
Fig. 6

A

B

Excitatory sensitivity (sp/s/°/s)

Inhibitory sensitivity (sp/s/°/s)

Peak velocity (°/s)

Type I, Inhibitory

Type II, Inhibitory

Type I, Excitatory

Type II, Excitatory

Type I

Type II

Fig. 6
Fig. 7