A neural correlate for vestibulo-ocular reflex suppression during voluntary eye–head gaze shifts

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The vestibulo-ocular reflex (VOR) is classically associated with stabilizing the visual world on the retina by producing an eye movement of equal and opposite amplitude to the motion of the head. Here we have directly measured the efficacy of VOR pathways during voluntary combined eye–head gaze shifts by recording from individual vestibular neurons in monkeys whose heads were unrestrained. We found that the head-velocity signal carried by VOR pathways is reduced during gaze shifts in an amplitude-dependent manner, consistent with results from behavioral studies in humans and monkeys. Our data support the hypothesis that the VOR is not a hard-wired reflex, but rather a pathway that is modulated in a manner that depends on the current gaze strategy.

The vestibulo-ocular reflex (VOR) produces a compensatory eye movement of equal and opposite amplitude to the motion of the head. This reflex effectively stabilizes the visual world on the retina for the wide range of head motions that are generated during daily activities[1,2]. However, primates can also voluntarily redirect their visual axes to new targets in their environment. Under natural conditions, in which the head is not restrained, a combination of eye and head movements (a gaze shift) is commonly used by humans[3–9] and monkeys[10–15] to redirect the visual axis to a new point in space. During such voluntary orienting behaviors, however, the eye movements produced by the VOR would be counterproductive; the VOR would produce an eye-movement command in the direction opposite to that of the intended shift of the axis of gaze.

Bizzi and colleagues[10–12] proposed that the VOR remains functional during eye–head gaze shifts, such that it eliminates the contribution of the head movement to the change in gaze. In this schema, the 'linear summation hypothesis'[6], the actual eye movement generated at the level of the extraocular motoneurons reflects the summation of two signals: an intact VOR mediated by vestibular pathways (that is, a compensatory eye-movement signal) and a separate saccadic eye-movement signal that redirects the fovea in space. More recent studies have provided results that argue against the linear summation hypothesis. This hypothesis cannot account for the observation that accurate gaze shifts can be generated to targets beyond the mechanical limits of ocular motility[4–6,14]. Moreover, a number of behavioral studies have shown that the gain of the VOR (eye velocity/head velocity) is considerably attenuated during gaze shifts, and the amount of attenuation increases as gaze shift amplitude increases from approximately 10 to 100 degrees[5–7,13,15–18]. Previous studies have not, however, determined a neural correlate for this on-line suppression of the VOR during voluntary gaze shifts.

A simple three-neuron arc (vestibular afferents to interneurons in the vestibular nuclei to extraocular motoneurons) mediates the most direct pathway of the VOR[19]. Studies in head-restrained monkeys have shown that there are several classes of vestibular nuclei neurons that discharge in relation to horizontal passive whole-body rotation and/or eye movements[20–28]. Position-vestibular-pause (PVP) neurons are thought to constitute most of the intermediate leg of the direct VOR pathway; PVP neurons receive a strong monosynaptic connection from the ipsilateral vestibular afferents and, in turn, project directly to contralateral extraocular motoneurons[20,26,28].

Here we investigated whether VOR suppression measured behaviorally during voluntary eye-head gaze shifts could be accounted for by a comparable reduction in the activity of the direct VOR pathways (that is, PVP neurons). First, we characterized the discharges of PVP neurons in the head-restrained condition, and then we released the head to record the discharges of these neurons during voluntary eye–head gaze shifts.

Results

Neuronal activity in the head-restrained condition

Figure 1 shows discharge patterns of a typical PVP neuron, unit 46_1, during saccadic eye movements and passive whole-body rotation. This neuron was representative of the PVP neurons in our sample (n = 9) in that its firing rate increased as a function of contralateral eye position during spontaneous eye movements (Fig. 1a), and its firing rate increased during ipsilateral whole-body sinusoidal rotation in the dark (VORd; Fig. 1b). We also did a second whole-body rotation experiment in which the monkey canceled its VOR by fixating on a head-centered visual target that moved with the vestibular turntable (VORc; Fig. 1c). This design allowed us to dissociate a neuron's sensitivity to vestibular stimuli from its modulation related to eye movement. The activity of PVP neurons increased during ipsilateral whole-body sinusoidal rotation regardless of whether or not the animal was canceling its VOR (compare Fig. 1b and c). In addition, each of the neurons stopped firing (paused) during ipsilaterally directed saccades and vestibular nystagmus quick phases (Fig. 1a and b, arrows). A minority

of the PVP neurons (22%) also completely paused for saccades in the contralateral direction.

To quantify neuronal discharges, we calculated each neuron's resting discharge (bias), sensitivity to eye position (k_x, (spikes/s)/degree), and sensitivity to passive head velocity (g_x, (spikes/s)/(degrees/s)). For our sample of PVP neurons, the mean head-velocity sensitivity was significantly lower (~25%) when the monkey canceled its VOR compared with VORd (mean, g_{xc} = 1.23 ± 0.79 versus g_{xd} = 1.61 ± 0.52; $p < 0.05$). A similar decrease in sensitivity has been reported for PVP neurons in the squirrel monkey[20,29]. The discharges of 70% of the neurons in this study were also recorded during rapid passive head rotations in the dark on a fixed body (which elicited velocities as high as 300 degrees per s, with trajectories comparable to those generated in large gaze shifts described below). During these rotations, the firing rate of each cell was accurately predicted when gaze was stable (that is, during compensatory VOR), using a model based on the linear addition of each neuron's bias and head-velocity sensitivity during VORd.

Neuronal activity during voluntary eye-head gaze shifts

After a neuron was fully characterized during head-restrained experiments, the monkey's head was slowly and carefully released to allow full freedom of head motion. During this critical transition, the unit activity and waveform were monitored on an oscilloscope to ensure that the cell remained undamaged and well isolated. Visual targets were presented to the monkey[30] to elicit large gaze shifts of up to 75 degrees in amplitude.

We characterized the head-velocity signal carried by each PVP neuron during voluntary gaze shifts of two amplitude ranges: small, 20–30 degrees, and large, 50–60 degrees. Because these cells increased their activity for ipsilateral head movement during passive whole-body rotation (Fig. 1b), only ipsilateral gaze shifts were considered. Typical gaze shifts are illustrated in Figs 2b and c and 3 for the example PVP neuron unit 46_1. As previously demonstrated[31,32], eye and gaze velocity reached their peak values early during large gaze shifts; however, subsequent re-accelerations were often observed (Figs 2b and c and 3b). Most PVP neurons showed a cessation of activity (pause) that preceded the onset of ipsilateral gaze shifts by approximately 10 ms, similar to that previously reported[29,32]. None of the PVP neurons in our sample paused for the entire duration of ipsilaterally directed gaze shifts. In fact, for many neurons, activity resumed before the completion of the ocular saccade component of a gaze shift.

For each cell, we first investigated whether the eye-position and head-velocity sensitivities estimated during head-restrained saccade and VORd experiments could predict the firing rate (fr) of the neuron during combined eye–head gaze shifts. The head-restrained model was given by the following equation (model 1):

$$fr = bias + (k_x \times eye\ position) + (g_{xd} \times head\ velocity) \qquad (1)$$

We analyzed the example PVP neuron unit 46_1 using this model (Figs 2 and 3). Model 1 consistently overestimated the discharges of PVP neurons for ipsilaterally directed vestibular quick phases (VORd, Fig. 2a), during which all PVP neurons paused. However, model 1 provided an excellent estimate of PVP neuron activity before and after vestibular quick phases (Fig. 2a). Similarly, model 1 overestimated the discharges of each PVP neuron during large (50–60 degree) gaze shifts, but provided a reliable estimate of PVP neuron discharges immediately before and after gaze shifts, even though the head was still moving (Fig. 2b and c). In addition, the model did not overestimate the discharge of the neuron during the post-

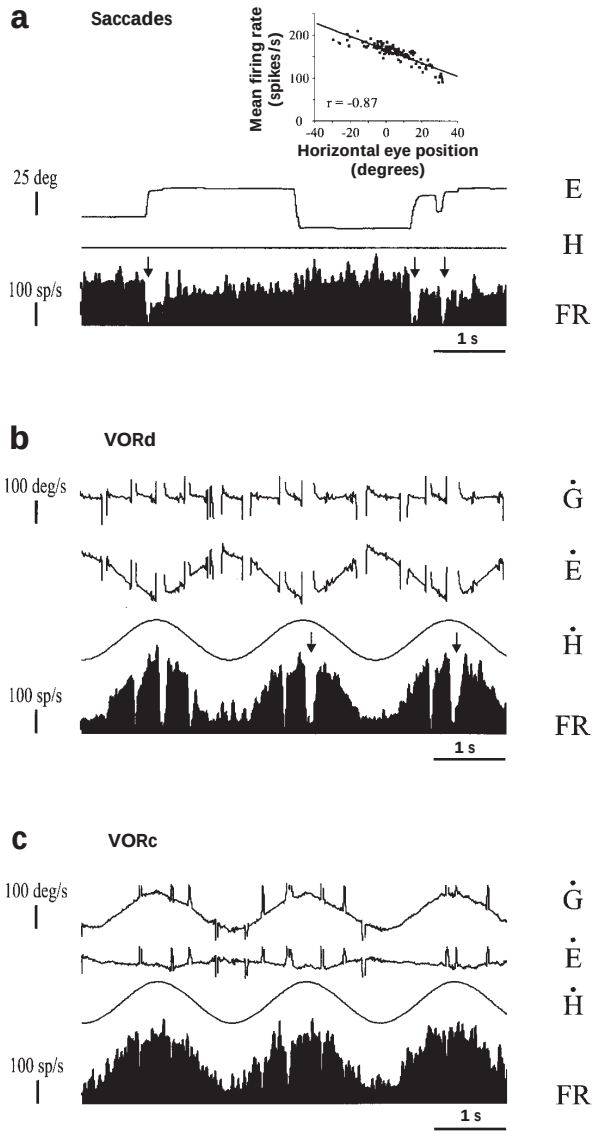

Fig. 1. Activity of an example PVP neuron (unit 46_1) during the head-restrained condition. **(a)** The PVP cell increased its discharge for contralateral eye movements (k_x = -1.55 spikes/s/degree; sample mean, k_x = -1.18 ± 0.7 spikes/s/degree) and paused for ipsilateral saccades (arrows). ***Inset*,** Mean neuronal firing rate was well correlated with horizontal eye position during periods of steady fixation (r = -0.87, n = 129). **(b, c)** Passive whole-body rotation (0.5 Hz, 80 degrees/s peak velocity) was used to study the neuron's response to head movements during VOR in the dark (VORd, (b)), and head movements during an experiment in which the monkey canceled its VOR by fixating a target that moved with the table (VORc, (c)). The head-velocity sensitivity coefficient of the PVP during VORd (g_{xd}) and VORc (g_{xc}) were 1.49 and 1.14 (spikes/s)/(degree/s), respectively. Unit 46_1 was typical of most PVP neurons in our study in that its modulation was less during VORc than during VORd. The arrows (b) indicate pauses in the activity of the PVP neurons during ipsilateral vestibular quick phases. Traces directed upward are in the ipsilateral direction. E, horizontal eye position; H, horizontal head position; FR, firing rate; Ġ, horizontal gaze velocity; Ė, horizontal eye velocity; Ḣ, horizontal head velocity. deg, degree; sp, spikes.

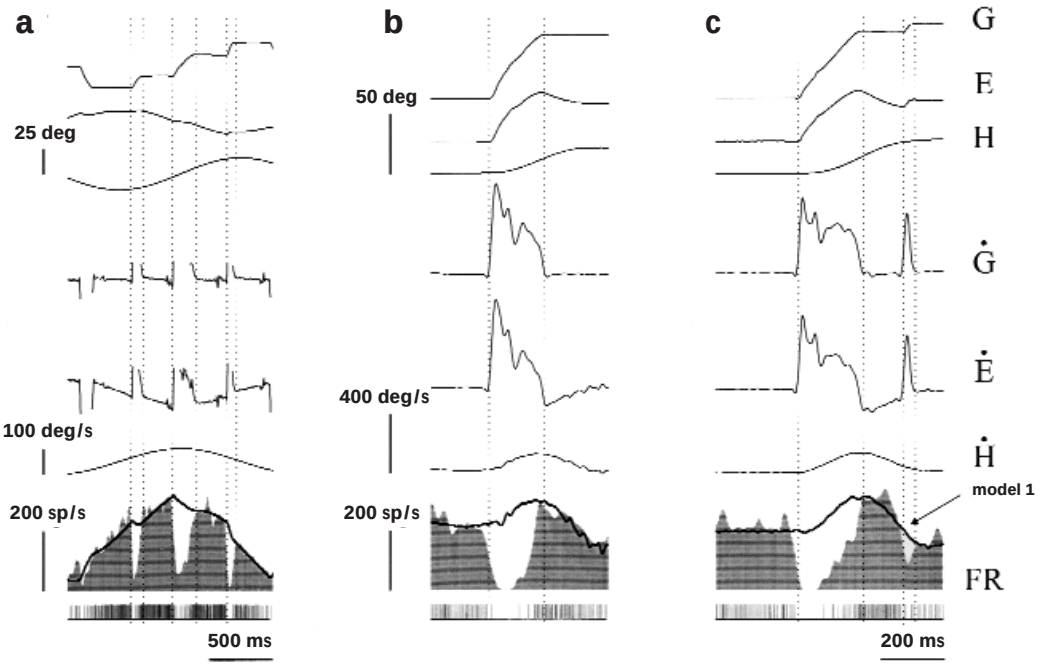

Fig. 2. Activity of an example PVP neuron (unit 46_1) before, during and after vestibular quick phases **(a)** and large gaze shifts **(b,c)**. The firing rate (shaded trace) is plotted above the output of the spike threshold discriminator for each gaze shift. A model based on head-restrained eye and passive head-movement sensitivities (arrow, model 1, dark trace) is superimposed on the firing rate traces. This model estimated accurately the discharge of PVP neurons before and immediately after quick phases and gaze shifts. In contrast, this model overestimated the discharge of this neuron during vestibular quick phases and gaze shifts. Because PVP neurons typically discharge at rates less than ~400 spikes/s, the firing rates predicted by model 1 were limited to values below this maximum. For example, this unit's firing rate was limited to 350 spikes/s, and accordingly the prediction of model 1 was not allowed to take on values exceeding this limit. G, horizontal gaze position; E, horizontal eye position; H, horizontal head position; FR, firing rate; $\dot{G}$, horizontal gaze velocity; $\dot{E}$, horizontal eye velocity; $\dot{H}$, horizontal head velocity; deg, degree; sp, spikes. Dotted vertical lines indicate the onset and offset of vestibular quick phases (a) and gaze shifts (b,c), using a criterion of ± 25 degrees/s.

saccadic interval even when followed by a second corrective saccade (Fig. 2c).

Model 1 consistently overestimated PVP neuron activity during small (20–30 degree), as well as large gaze shifts (Fig. 3). To quantify this observation, we determined the best estimate of each neuron's head-velocity sensitivity (g_{fs}) during small and large gaze shifts using the equation (model 2):

$$fr = bias + (k_x \times eye\ position) + (g_{fs} \times head\ velocity) \qquad (2)$$

where the values of the bias and k_x were taken from the head-restrained analysis (above), and the model was fit to a data set that contained 20 or more gaze shifts within each amplitude range. A percent attenuation index, $1 - (g_{fs} / g_{xd})$, was then calculated to compare the values that were estimated for g_{fs} to those estimated for g_{xd} in the head-restrained analysis. The example neuron here was typical in that its head-velocity sensitivity attenuation (compare model 1 and model 2) was greater for large gaze shifts (Fig. 3b) than for small ones (Fig. 3a).

Figure 4a further illustrates the amplitude-dependent attenuation of g_{fs} that we observed during gaze shifts for the example neuron. Gaze shifts (range, 20–65 degrees) were sorted into separate data sets, each spanning 5 degrees and containing 15 or more examples. The percent attenuation index of the head-velocity sensitivity during the gaze shift (Fig. 4a, filled symbols) was calculated for each amplitude range. During the gaze shift, the amount of attenuation of the head-velocity signal carried by PVP neurons tended to increase with gaze-shift amplitude. A particularly notable example of this relationship is shown for a second neuron, unit 39_1 (Fig. 4b). For our entire sample of PVP neurons, the percent attenuation of head-velocity sensitivity was significantly greater for large gaze shifts (50–60 degrees) than for small gaze shifts (20–30 degrees) (Fig. 4c, filled columns; 58% ± 10 versus 35% ± 15; $p < 0.05$).

We also used model 2 to obtain an estimate of head-velocity sensitivity over the interval of 10–80 ms that immediately followed each gaze shift. A post-movement drift in gaze often follows gaze shifts (Fig. 3b, left side), suggesting that the VOR is not fully operational during this interval[31]. We found attenuation of g_{fs} immediately after gaze shifts for our example PVP neurons (Fig. 4a and b, open symbols). However the head-velocity signal was much less attenuated after the gaze shift than during the gaze shift, and the level of attenuation did not vary systematically with gaze-shift amplitude (Fig. 4a and b; compare open symbols with filled symbols). For our entire sample of PVP neurons, the percent attenuation was significantly less ($p > 0.001$) during the post-gaze-shift interval (7% ± 8 and 16% ± 4 for small and large gaze shifts, respectively) than during the gaze shift itself. This result indicates that the head-velocity sensitivity of PVP neurons increased immediately after gaze shifts (Fig. 4c, open columns) and achieved a value only slightly less than that observed during VOR in the dark. The residual post-gaze-shift attenuation of the PVP neurons' responses was accompanied by a similar attenuation in the behavioral VOR gain (gain attenuation = 1–(eye velocity/head velocity); mean, 9% ± 5 and 7% ± 6 for small and large gaze shifts, respectively; Fig. 4c, shaded columns). In contrast, the behavioral VOR gain during VORd was 0.98 ± 0.06, corresponding to an attenuation of only ~2% from perfect gaze stabilization.

After we completed our characterization of the neuron dur-

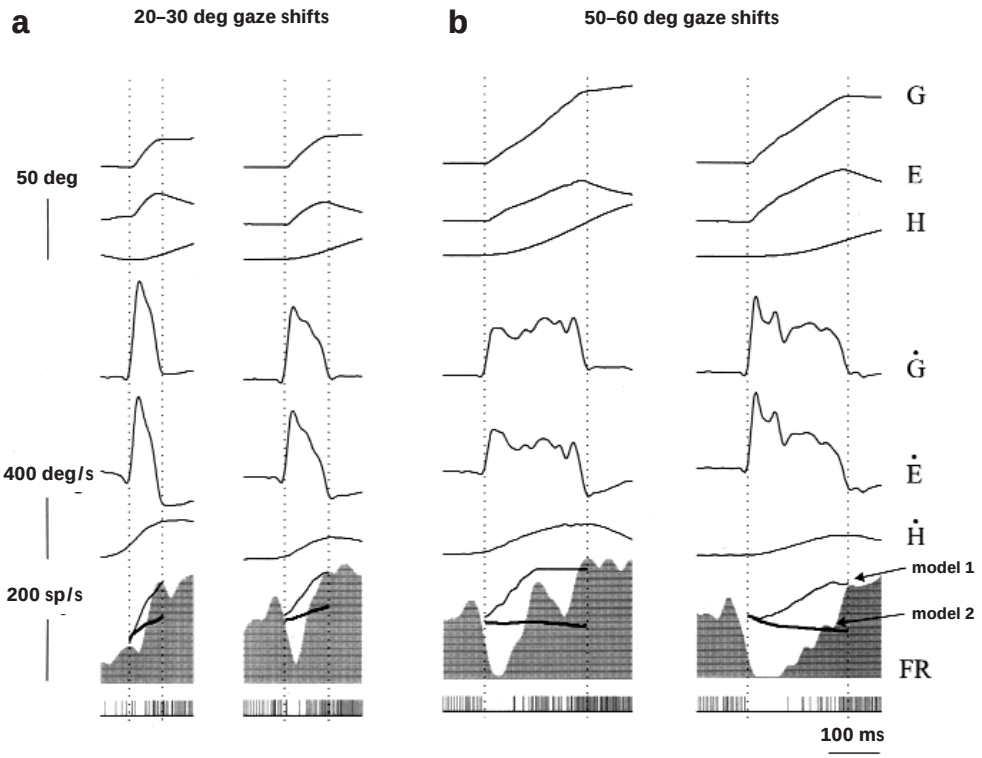

Fig. 3. Example gaze shifts of 20–30 degrees **(a)** and 50–60 degrees **(b)** amplitude for the example PVP neuron, unit 46_1. The firing rate (shaded trace) is plotted above the output of the spike threshold discriminator for each gaze shift. Superimposed on the firing rate trace are two model fits: models 1 and 2 (indicated by arrows). Model 1 overestimates the firing rate during gaze shifts of both amplitudes. G, horizontal gaze velocity; E, horizontal eye position; H, horizontal head position; FR, firing rate; Ġ, horizontal gaze velocity; Ė, horizontal eye velocity; Ḣ, horizontal head velocity; deg, degree; sp, spikes. Dotted vertical lines indicate the onset and offset of gaze shifts using a criterion of ± 25 degrees/s.

ing active gaze shifts, the monkey was returned to the head-restrained condition. Forty-four percent of the neurons in our sample remained well isolated after the monkey's head had been rerestrained. We confirmed that these neurons had not been lost or damaged by repeating the passive whole-body rotation experiments (VORd and VORc); neural discharges did not differ considerably in the second head-restrained characterization, compared to the initial head-restrained characterization.

Discussion

Here we compared the efficacy of the direct VOR pathways during whole-body rotation with that during voluntary orienting

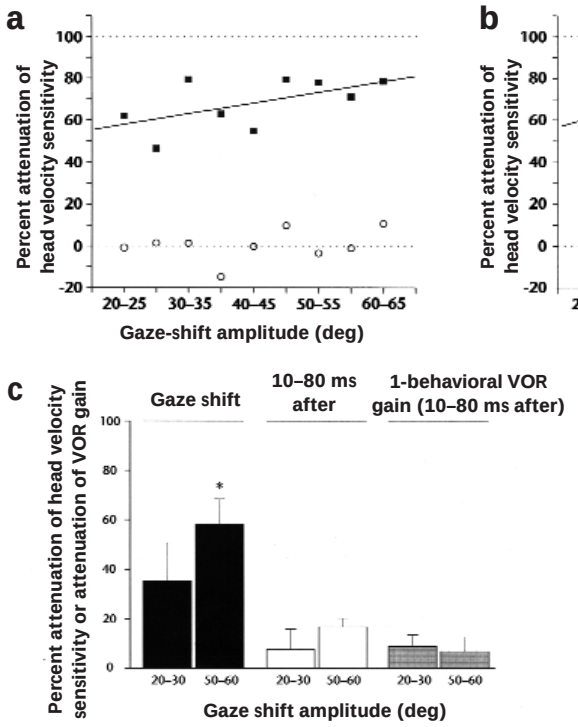

Fig. 4. Amplitude-dependent attenuation of head-velocity sensitivity during gaze shifts. **(a)** Relationship between the percent attenuation of the head-velocity sensitivity and gaze-shift amplitude for the example PVP neuron (unit 46_1) during gaze shifts (■; $r = 0.57$) and immediately (10–80 ms) after gaze shifts (○; $r = 0.32$). **(b)** Relationship between the percent attenuation of the head-velocity sensitivity and gaze-shift amplitude for a second PVP neuron (unit 39_1) during gaze shifts (■; $r = 0.87$) and immediately after gaze shifts (○; $r = -0.35$). **(c)** During gaze shifts, the percent attenuation of head-velocity sensitivity for our sample of PVP neurons was significantly different for gaze shifts of 20–30 degrees and 50–60 degrees (filled columns; 35% ± 15 versus 58% ± 10; $p < 0.05$, asterisk). In contrast, there was little attenuation in the post-gaze-shift interval (open columns; 7% ± 8 and 16% ± 4, respectively). The behavioral VOR gain (eye velocity/head velocity) was also slightly less than one during this interval (shaded columns; 9% ± 5 and 7% ± 6 for small and large gaze shifts, respectively).

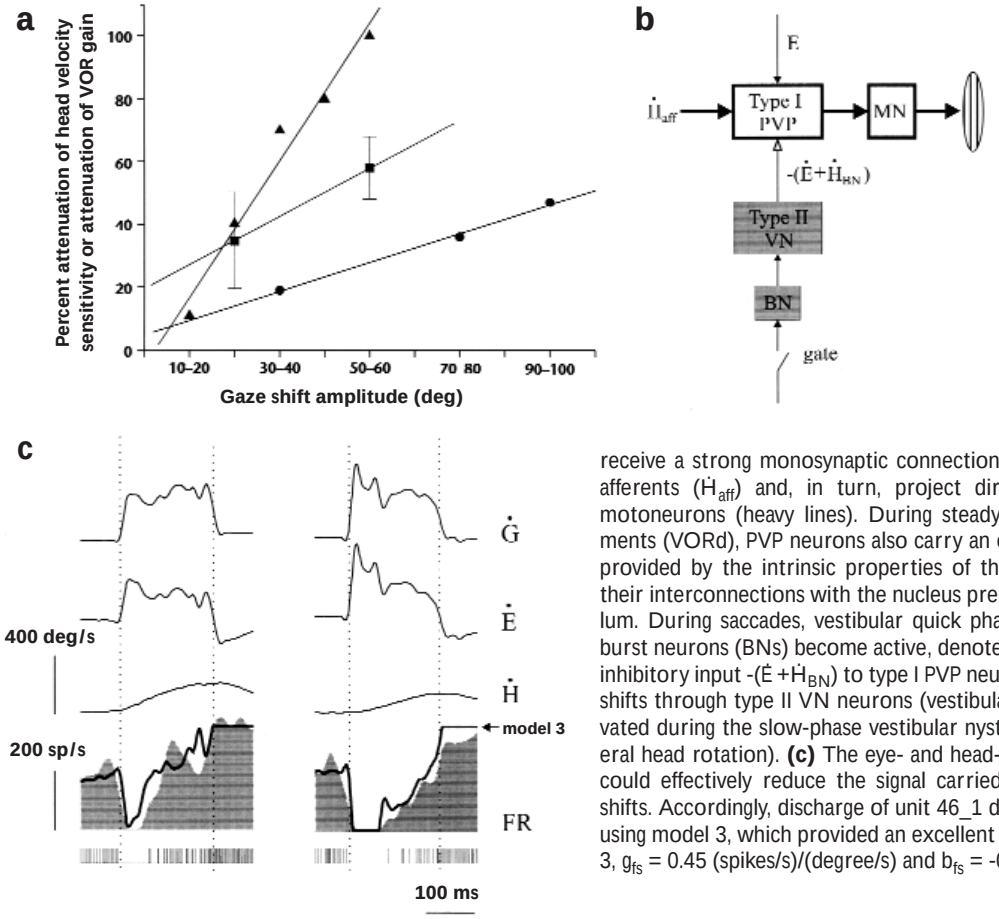

Fig. 5. Mechanisms for VOR suppression during gaze shifts. **(a)** Mean percent attenuation of PVP (■) head-velocity sensitivity during gaze shifts compared with percent attenuation of the VOR gain as measured in previous behavioral experiments during gaze shifts in humans (●) and monkeys (▲). **(b)** Brainstem mechanism that could produce an apparent reduction in VOR gain during gaze shifts. PVP neurons receive a strong monosynaptic connection from the ipsilateral vestibular afferents ($\dot{H}_{aff}$) and, in turn, project directly to primary extraocular motoneurons (heavy lines). During steady fixation and slow eye movements (VORd), PVP neurons also carry an eye position signal (E), which is provided by the intrinsic properties of these vestibular neurons and/or their interconnections with the nucleus prepositus hypoglossi and cerebellum. During saccades, vestibular quick phases and gaze shifts, brainstem burst neurons (BNs) become active, denoted by the 'gate'. BNs provide an inhibitory input $-(\dot{E}+\dot{H}_{BN})$ to type I PVP neurons (studied here) during gaze shifts through type II VN neurons (vestibular nuclei neurons that are activated during the slow-phase vestibular nystagmus generated by contralateral head rotation). **(c)** The eye- and head-velocity signals carried by BNs could effectively reduce the signal carried by PVP neurons during gaze shifts. Accordingly, discharge of unit 46_1 during gaze shifts was estimated using model 3, which provided an excellent fit of the unit discharge (model 3, g_{fs} = 0.45 (spikes/s)/(degree/s) and b_{fs} = -0.47 (spikes/s)/(degree/s)).

eye–head movements in the horizontal plane. PVP neurons constitute most of the intermediate leg of the simple three-neuron arc that mediates the direct VOR pathway. Our main conclusions are that head-velocity information is less effectively transmitted from the vestibular afferents to the extraocular motoneurons by these neurons during active orienting gaze shifts than during the compensatory VOR elicited by passive whole-body rotation, and that the head-velocity signal carried by the direct VOR pathway is more attenuated for large- than for small-amplitude gaze shifts.

Indeed, we found that the attenuation of the head-velocity signal carried by PVP neurons during gaze shifts corresponds with the results of behavioral studies that have characterized the VOR gain during gaze shifts in monkeys and humans[5–7,13,15–18]. We plotted the percent attenuation of the VOR versus gaze-shift amplitude based on previous studies in the rhesus monkey[13] and in humans[18] (Fig. 5a). For comparison, we superimposed a plot of percent attenuation of PVP head-velocity sensitivity during gaze shift versus gaze-shift amplitude from this study. The variability between the two behavioral data sets demonstrates the uncertainty of previous estimates of VOR gain. However, the variabilities within our data set and that of the human study[18] are similar (±15% and 10% versus ±19%, 11% and 13%, respectively). Although our data do not exactly match either of the two behavioral data sets, the similarity in trends is strong evidence that the amplitude-dependent reduction of the head-velocity signal carried by direct VOR pathways is at least partly responsible for the amplitude-dependent decrease in VOR gain that has been reported in behavioral studies.

We also found that the average head-velocity sensitivity of PVP neurons recovered significantly within the first 10–80 ms after the end of a gaze shift. The residual attenuation of the PVP head-velocity signal during this period was minimal (~12%). This discharge attenuation is in agreement with the observation that the actual gain of the behavioral VOR immediately after a gaze shift is, on average, slightly less than one during this interval (~8%). Our results do not agree with those of the recent behavioral study in humans[18], which reported that the VOR returns to a value greater than unity at the end of gaze shifts. This discrepancy may have arisen from a difference in the head-velocity profiles used in the two studies. These authors[18] used head perturbations of 12–20 Hz to investigate the VOR gain, whereas we studied natural head movements, which have very little frequency content above 5 Hz[1].

Several behavioral studies have attempted to characterize the time course of the suppression of the VOR during gaze shifts. The VOR was first reported to be switched from an 'on' to an 'off' state during gaze shifts[6]. A gradual restoration of the VOR was also proposed[5], furthered by findings that the gain of the VOR fell off exponentially as gaze shift amplitude increased[7]. Other authors suggest that the VOR gain increases from zero to unity during the last 40 ms of a gaze shift[16]. The most recent proposal is that VOR gain decreases exponentially (time constant, ~50 ms) from the onset of the gaze shift[18]. In our analysis, we estimated average head-velocity sensitivities for each neuron in two intervals: during the gaze shift and immediately after the gaze shift. By estimating average head-velocity sensitivities, we have not attempted to account

for possible dynamic modulation of the gain of VOR pathways within each of these intervals; the precise time course of attenuation of VOR pathways during gaze shifts remains to be determined.

The attenuation that we observe during active gaze shifts can be accounted for by known brainstem mechanisms (Fig. 5b). Burst neurons in the brainstem paramedian pontine reticular formation generate a burst of spikes to drive horizontal saccadic eye movements, but do not discharge during either steady fixation or slow eye movements, including VORd slow phases[33–40]. Burst neurons project directly to neurons in the vestibular nuclei (VN), which are activated during the slow-phase vestibular nystagmus generated by contralateral head rotation (type II VN neurons)[36]. These type II VN neurons then send inhibitory projections to VOR interneurons, which are activated during slow-phase vestibular nystagmus generated by ipsilateral head rotation (type I PVP neurons)[41]. The activity of brainstem burst neurons (inhibitory burst neurons and presumably the excitatory burst neurons) carry head- as well as eye-velocity signals during gaze shifts[30]. The eye- and head-velocity signals carried by excitatory burst neurons could have an inhibitory effect on the activity of type I PVP neurons (the subject of this study) through type II VN neurons.

We propose that the signals related to eye and head movement carried by brainstem burst neurons (which are only active during saccades, vestibular nystagmus quick phases and gaze shifts) contribute to decreasing the efficacy of the direct VOR pathways during gaze shifts. Indeed, we found that a model with separate terms for eye- and head-velocity could produce an excellent fit to PVP neuron discharges during gaze shifts (model 3, Fig. 5c):

$$\text{fr} = \text{bias} + (k_x \times \text{eye position}) + (g_{fs} \times \text{head velocity}) + (b_{fs} \times \text{eye velocity}) \quad (3)$$

Other brainstem mechanisms could also contribute to modulating the vestibular sensitivity of vestibular neurons during active gaze shifts. For example, it has been proposed that a centrally generated copy of the head efference signal could be used to regulate the vestibular sensitivity of PVP neurons during active head movements[29]. Future efforts will need to consider the complex interactions between the brainstem circuits that generate voluntary eye movements (such as saccades) and vestibularly driven eye movements to realistically model the control of gaze during voluntary orienting behaviors.

Methods

Three monkeys (two *Macaca mulatta* and one *Macaca fasicularis*) were prepared for chronic extracellular recording. The surgical preparation and extracellular recording techniques have been described[30]. All experimental protocols were approved by the McGill University Animal Care Committee and complied with the guidelines of the Canadian Council on Animal Care. During the experiments, the monkey was seated in a primate chair, which was fixed to the suprastructure of a vestibular turntable. Monkeys were trained to track a target (a HeNe laser spot projected onto a cylindrical screen) for a juice reward. The target was stepped along the horizontal plane over a range of ± 30 degrees in the saccade experiment and ±40 degrees in the gaze shift experiment. Behavioral experiments, target and turntable motion and the storage of data were controlled by a UNIX-based real-time data acquisition system (REX)[42]. Gaze- and head-position signals, target position, vestibular turntable velocity and unit activity were recorded on a DAT tape for later playback and analysis. Off-line, the position signals were low-pass filtered at 250 Hz (8 pole Bessel filter) and sampled at 1000 Hz. The gaze- and head-position signals were digitally filtered at 125 Hz. Eye position was calculated from the difference between the recorded gaze- and head-position signals. Gaze-, head- and eye-positions were differentiated to produce velocity signals. Locations of the medial and lateral vestibular nuclei were determined relative to the abducens nucleus, and extracellular recordings were made in these nuclei. The abducens nucleus was identified based on its stereotypical discharge patterns during eye movement and whole-body rotation experiments[21]. Neural spike trains were determined using a windowing circuit (BAK) that was set manually to generate a pulse coincident with the rising phase of each action potential. The neural discharge was represented using a spike density function in which a Gaussian function was convolved with the spike train[43].

A neuron's eye-position sensitivity coefficient, k_x, spikes/s/degree, was determined by plotting its mean firing rate against eye position during periods of steady fixation in the saccade experiment and calculating the slope of the relationship. The eye-position contribution to the neuronal discharge (the product of k_x and eye position) was subtracted from the firing rate before head-velocity sensitivities during VORd and VORc were determined. Statistical significance was determined by using a paired Student's *t*-test. A least-squared regression analysis was then used to determine each unit's phase shift relative to head velocity, resting discharge (bias, spikes/s) and head-velocity sensitivity (g_x, in (spikes/s)/(degrees/s)). This analysis was done during VORd and VORc to obtain two estimates of a cell's head-velocity sensitivity (g_{xd} and g_{xc} respectively). Only unit data from periods of slow-phase vestibular nystagmus (VORd) or steady fixation (VORc) that occurred between quick phases of vestibular nystagmus and/or saccades were included in the analysis.

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