

Studies of the Role of the Paramedian Pontine Reticular Formation in the Control of Head-Restrained and Head-Unrestrained Gaze Shifts

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ABSTRACT: Results of three experiments related to the role of the paramedian pontine reticular formation (PPRF) in the control of gaze are described. (1) Chronic unit recording methods, used to study the on-directions of short-lead burst neurons in head-restrained monkeys, and (2) reversible inactivation techniques confirmed the traditional view of the importance of PPRF in the control of horizontal eye movements. Reversible inactivation of neurons in the vicinity of identified short-lead burst neurons produced dramatic reductions in the speed of saccades to horizontal target displacements. The reductions in velocity were largely compensated for by an increase in saccade duration. Only minor, if any, effects were observed upon the velocity, duration, and amplitude of saccades to upward target displacements. (3) Microstimulation was applied to omnipause neurons to gate activity of excitatory burst neurons that discharge during coordinated eye-head movements. The microstimulation failed to noticeably slow (prevent) head movements when stimulation was applied during (prior to onset of) gaze shifts, suggesting that signals relayed to motoneurons innervating the neck muscles are not inhibited by the omnipause neurons. In other words, the desired gaze signal is parsed into eye and head pathways upstream of the excitatory burst neurons.

KEYWORDS: saccades; PPRF; reversible inactivation; gaze; OPNs; microstimulation

INTRODUCTION

Most models of the neural control of saccadic eye movements assume that neurons in the paramedian zone of the pontine reticular formation (PPRF) generate motor command signals that effect changes in the horizontal positions of the eyes during saccades and the quick phases of nystagmus. This assumption is based upon accumulated evidence obtained from microstimulation, lesions, clinical, anatomical, and chronic single-unit recording data (see Refs. 1–4 for reviews).

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Recently, the traditional view of PPRF function has been challenged in two ways. First, one model⁵ assumes that saccade commands are not decomposed into horizontal and vertical commands until the level of the motoneurons. The concept was motivated by data indicating that individual neurons in PPRF discharge before vertical saccades⁶ and by findings suggesting that the “on-direction” of PPRF neurons may be distributed continuously, rather than clustered around horizontal (or vertical) directions.⁷ Second, recent papers have suggested that neurons in the PPRF send both eye-velocity and head-velocity signals to extraocular muscle motoneurons. This suggestion emerged from studies of the activity of inhibitory burst neurons (IBNs) during eye, head, and gaze movements generated by monkeys without head restraint. Cullen and Guitton⁸ found that for most of the IBNs studied the number of spikes in a burst was better correlated with gaze amplitude than with the amplitude of either the eye or head components of the gaze shift. Below, we report data relevant to the issues raised by these challenges to the traditional view of PPRF function.

EXTRACTION OF HORIZONTAL AND VERTICAL COMMAND SIGNALS

Best Direction of Short-Lead Burst Neurons in PPRF

It is surprising that issues about the on-direction of cells in PPRF are still unresolved 30 years after chronic unit recording methods allowed the first descriptions of pontine cells with saccade-related and eye-position-related activity.^{9–12} However, untrained subjects were used in early experiments, and the range of saccade directions and amplitude generated while the activity of a cell was being studied was not under experimental control. Also, the stability, accuracy, and precision of the methods used to record eye position in these early studies were less than ideal.

Descriptions of the on-direction of burst neurons in PPRF found in more recent papers are usually secondary to the major purpose of the paper. For example, the evidence that the on-direction short-lead burst neurons (SLBNs) can have a large vertical component was obtained, primarily, from intra-axonal recordings performed in experiments designed to trace anatomical pathways following the intracellular injection of tracer substances.^{7,13} The number of saccade directions and amplitudes was, of necessity, small. Moreover, these experiments were also performed in untrained animals, and the distribution of saccade directions and amplitudes used for estimates of on-direction was limited. Below, we present data obtained from 18 SLBNs under experimental conditions that allowed greater control over saccade direction and amplitude.

The activity of SLBNs in the PPRF was recorded while rhesus monkeys, with their heads restrained, generated saccades to small (0.1°) visual targets for a liquid reward. The targets consisted of an array of LEDs located on a tangent screen. The array spanned 48° horizontally and 40° vertically, with a 2° or 4° spacing. Eye positions were recorded using the search-coil method.¹⁴ Horizontal and vertical eye positions were sampled every 2 ms, and the digitized interspike intervals were recorded with 100 μ s resolution. Quantitative analysis was done off-line. Eye velocity was computed from the horizontal and vertical eye position data using a 5-point central difference differential algorithm. Saccade onset and offset were defined by a velocity criterion, usually $30^\circ/\text{s}$. Burst onset and offset, defined as the first spike and the last

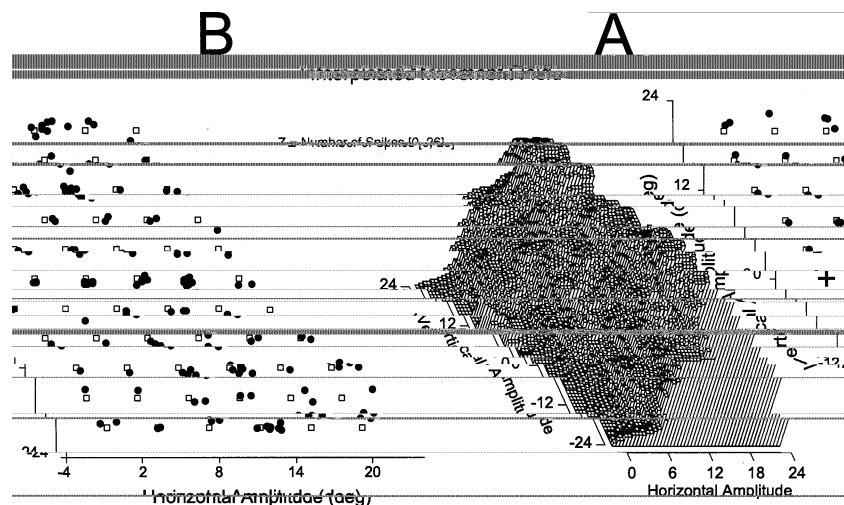


FIGURE 1. Direction and amplitude of saccades used to construct a three-dimensional movement field of an SLBN (# spikes as a function of the horizontal and vertical amplitude). (A) Locations of visual targets (*open squares*) with respect to the initial fixation target (+) and the end points (*filled circles*) of 160 primary saccades made to acquire these targets. The saccade-related discharge was recorded concurrently with the behavioral data shown. (B) The number of spikes (*z* axis, ranges from 0 to 26) is plotted as a function of horizontal and vertical amplitude for the same trials shown in A. The plot was obtained by interpolating from the 160 discrete values.

spike, respectively, in a saccade-related burst, were determined by the investigator. Linear and curvilinear regression analyses were used to examine the relationship between SLBN burst and saccade metrics. The best direction of a given SLBN was determined using the method described by Henn and Cohen.¹⁵ For each saccade in a data set, a component amplitude (DPOS) along a specified projection plane was calculated as

$$\text{DPOS} = \text{saccade vector amplitude} * \cos(\text{saccade direction angle} - \text{projection plane})$$

for projection planes ranging from 0° to 360° in 1° increments. The plane showing the highest correlation between number of spikes and component amplitude (DPOS) along the given projection plane was defined as the best direction.

FIGURE 1A illustrates the range of saccade amplitudes and directions generated during behavioral trials while we were recording the activity of one SLBN. Panel B is an interpolated movement field plot of the number of spikes in the saccade-related burst of activity as a function of the horizontal and vertical amplitude of the saccade. Visual inspection of the movement field plot indicates that much of the variance in the number of spikes in the saccade-related burst can be attributed to the amplitude of the horizontal component of the saccade. The Henn and Cohen analysis indicated that DPOS was 0 for this neuron.

Results of the Henn and Cohen analysis for a different SLBN are illustrated in FIGURE 2. Panel A plots the number of spikes in the saccade-related burst as a func-

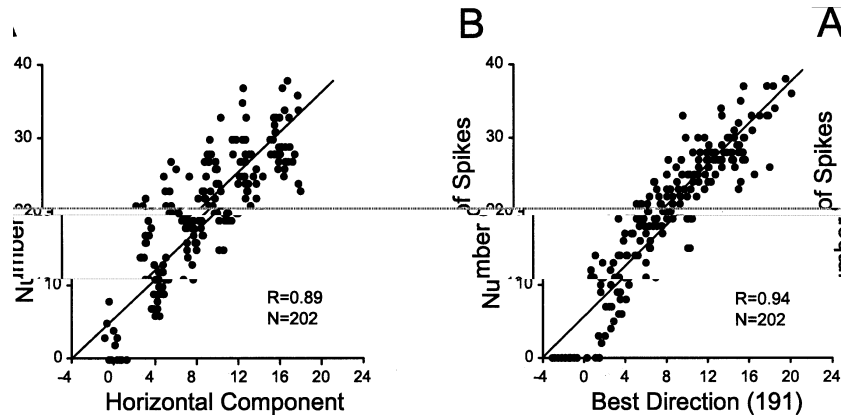


FIGURE 2. The number of spikes in the discharge of a SLBN as a function of the horizontal component versus the best direction component. (A) The number of spikes plotted as a function of the amplitude of the horizontal component (correlation coefficient = 0.89). (B) The number of spikes as a function of the amplitude of the best direction component (correlation coefficient = 0.94). The best direction = 191° as determined by the technique of Henn and Cohen (see METHODS). The trials in A and B are identical ($n = 202$).

TABLE 1. “Best direction” analysis for SLBNs in PPRF

Best Direction	R – Best Direction	R – Horizontal or Vertical	Direction	N
0	.91	.91	Right	120
0	.94	.94	Right	244
1	.97	.97	Right	147
2	.76	.75	Right	205
3	.96	.95	Right	293
5	.97	.96	Right	241
355	.88	.86	Right	128
353	.80	.77	Right	242
8	.97	.94	Right	128
11	.96	.90	Right	609
50	.76	.55	Right	304
181	.89	.88	Left	158
185	.96	.94	Left	174
191	.92	.85	Left	187
193	.85	.73	Left	228
276	.93	.89	Down	113
83	.95	.91	Up	325
101	.88	.83	Up	296

By column: (1) Best direction determined using the method of Henn and Cohen. (2) Correlation coefficient for the relationship between number of spikes and component amplitude along a projection plane of the best direction of the cell. (3) Correlation coefficient for the relationship between number of spikes and the amplitude of the movement along cardinal (horizontal or vertical) directions. (4 and 5) Optimal saccade direction (up, down, left or right) and the number of trials used in computing the correlation coefficients.

tion of the horizontal component of the movement. Panel B plots the same data as a function of the component amplitude along a projection plane of 191° , the best direction for this cell. The scatter of points about the line of best fit is reduced, and the correlation coefficient increases from 0.89 to 0.94. TABLE 1 presents results of the "best direction" analysis for 18 PPRF SLBNs, including three that discharged most vigorously in association with vertical saccades. The best direction of 9 of the 18 cells was within 5° of either the horizontal or vertical axes; the best direction of 13 of the 18 cells was within 10° ; and the best direction of 17 of the 18 cells was within 20° of horizontal or vertical axes. The best direction of one cell was 50° .

These results are in good agreement with two descriptions of the best direction of IBNs. Scudder and colleagues¹⁶ noted that although the best directions of IBNs were seldom purely horizontal, most clustered near the horizontal axes. The extent of the spread, measured as root-mean-square deviation from horizontal, was only 14.2° for SLBNs and 9.9° for long-lead burst neurons. Cullen and Guitton¹⁷ reported that the preferred directions in their sample of IBNs varied from 29° upward to 30° downward. The mean preferred direction was -1° , closely aligned with the horizontal plane. Based upon these results and the data presented in this paper, models in which information about horizontal components of saccades is not extracted until the level of extraocular motoneurons is not necessary. Moreover, the assumptions of the Quia and Optican model⁵ that the on-directions are evenly distributed and equally represented are not supported by available data. It is true, however, that the on-directions of the pontine neurons having monosynaptic connections with motoneurons are unknown.

Reversible Inactivation of Neurons in Rostral PPRF

Most models of the saccadic system assume that neurons in PPRF extract a signal of horizontal eye displacement and that a similar signal specifying the vertical component of saccades resides in the rostral intrastitial nucleus of the medial longitudinal fasciculus (rIMLF) and other nuclei in rostral midbrain. These models predict that reversible inactivation of cells in PPRF should affect primarily horizontal movements and that alterations in the accuracy or speed of saccades to vertical targets would be small.

Reversible lesions were produced using the recording/injection probe developed by Malpeli and Schiller¹⁸ and was accomplished by pressure injection of 50–200 nl of 2% lidocaine. In these initial experiments, we used lidocaine because, based upon results of our experiments in the SC, the size of the affected area is smaller with lidocaine than with muscimol injections. Also, lidocaine injections produced behavioral effects that lasted for 20–40 min, whereas the effects of muscimol could persist for several hours. Thus, with lidocaine injections it was possible to make multiple injections during an experimental session. Also, the extent to which performance deficits could be attributed to behavioral strategies rather than disruption of neural function could be assessed by observing the pattern and time course of behavioral recovery.

FIGURE 3 illustrates the effect of consecutive inactivations on saccades to targets presented either 20° to the left or 20° above the fixation target, with target location randomly interleaved. The lidocaine was injected twice into the rostral PPRF at the site of SLBN activity. Separate panels plot the peak velocity, duration, and amplitude

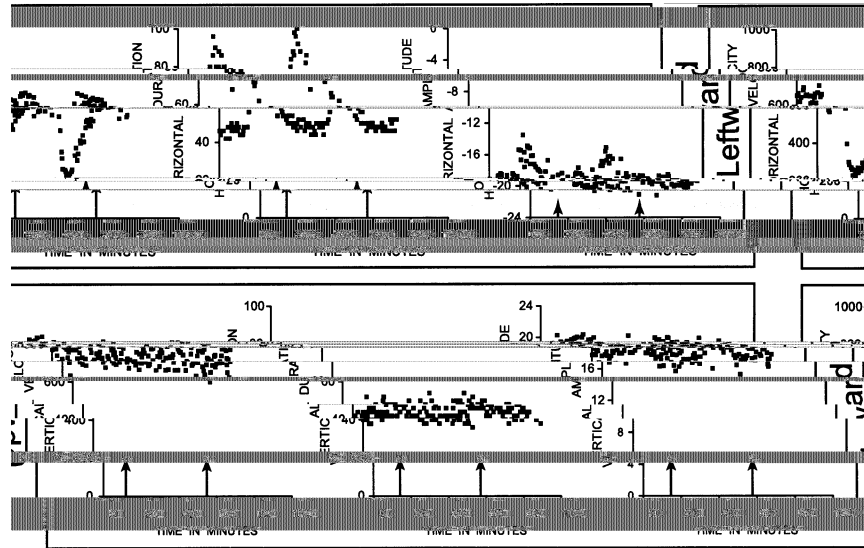


FIGURE 3. Upward vs. leftward movements: the effect of consecutive injections of lidocaine on saccades to horizontal and vertical target displacements. **Top panel:** peak velocity, duration, and amplitude of the horizontal component of saccades (target = $-20, 0$) as a function of time. **Bottom panel:** peak velocity, duration, and amplitude of the vertical component of saccades (target = $0, 20$) as a function of time. Each point represents a measurement from a single trial and the arrows indicate time of injections. The measurements shown in the two panels are synchronous because the targets were randomly interleaved. Amount of lidocaine: injection #1, 150 nl; #2, 200 nl.

of movements to horizontal (top) and vertical (bottom) target displacements measured during a 90-min period beginning about 15 min before the first of two injections (upward arrows). Before the injections, peak velocity of leftward movements was about $600^{\circ}/s$, but dropped to about $250^{\circ}/s$ after the injection and gradually recovered over the next 20–30 min. With a similar time course, the duration of the saccades increased from control values of about 45 ms to as much as 95 ms, and then returned to control. The lengthened duration counterbalanced the reduced velocity, but not entirely, as saccades remained hypometric (see figure legend for more details). Referring to the bottom panels, note that the inactivation of neural activity in rostral PPRF had only minor, if any, effects upon the velocity, duration, and amplitude of saccades to upward target displacements.

In summary, all eight inactivations caused an increase in the duration and a decrease in the peak velocity of horizontal saccades in the ipsilesional direction, while the results for upward saccades were inconsistent and relatively small. For saccades of both directions, the amount of lidocaine and the location of the injection within the PPRF influenced the magnitude of the effect. Across the eight injections, the average maximal reduction in horizontal peak velocity was $389.4^{\circ}/s$ for saccades to targets that were displaced 20° horizontally. For the same saccades, the average maximal increase in duration was 32.6 ms. The prolonged duration compensated for the diminished velocity, but usually not completely, with the saccades still hypometric

for six of eight injections. For saccades to targets that were displaced 20° vertically, no significant influences occurred on the vertical peak velocity of six of eight injections. For the remaining two injections, the significant effects were minor and in opposite directions (maximal change: 32.9°/s and -54.8°/s). Six of the eight injections produced a relatively small but significant increase in vertical duration (average maximal change 5.1 ms), while the other two caused no effect and a decrease in duration (3.4 ms). Because the duration of the vertical component increased slightly without an associated decrease in peak velocity for five of eight injections, the amplitude of the vertical component increased for these five injections (average maximal change 1.7°), while the other three injections induced no effect ($n = 2$) and a decrease (-1.7°) in amplitude.

It is not possible to attribute the behavioral consequences of the lidocaine injections to disruption of the activity of a particular functional class of PPRF neurons. Lidocaine injections into the rostral PPRF are presumed to affect the activity of all neural elements in the area of the injection. This includes (a) the SLBNs described in the previous section—cells generating a burst of activity primarily associated with the horizontal component of saccades and the quick phases of nystagmus; (b) long-lead burst neurons that may generate less discrete saccade-related bursts and have peak activity associated with movements that are not purely horizontal; (c) a smaller number of cells discharging maximally before vertical movements; and (d) fibers passing through the region of the injection. Despite the many types of neurons affected by the injections, the reversible lesions in rostral PPRF produce large changes in the velocity and duration of saccades to horizontal targets without having major effects upon the velocity, duration, or amplitude of saccades to randomly interleaved vertical targets. The deficits we observed were similar to those described by other researchers following permanent and more extensive lesions in the same regions of the brainstem.^{4,19,20} Although not studied in detail, we did note that injections at more caudal sites disrupted both horizontal and vertical rapid eye movements. Caudal injections involving the vestibular nuclei and nucleus prepositus hypoglossi produced gaze-holding deficits and gaze-evoked nystagmus similar to what has been previously reported.²¹⁻²³

Collectively, the recording and reversible inactivation data presented in this paper add to the large body of evidence indicating that cells in the PPRF specialize in the generation of commands for horizontal eye movements. Because we did not measure torsional rotations of the eye, our data do not address the suggestion²⁴ that the PPRF contains a three-dimensional arrangement of signals similar to what has been observed in the rostral midbrain.

STIMULATION OF PONTINE OMNIPAUSE NEURONS DURING AND BEFORE COORDINATED MOVEMENTS OF THE EYES AND HEAD

The PPRF contains neurons involved in the control of movements of the eyes and the head. Anatomical studies have shown that projections to both neck and eye motoneuron pools arise from both the oral and caudal divisions of the PPRF.²⁵ Signals carried by reticulospinal neurons are related to both neck muscle activity and eye position. Single pontine neurons send axons to the spinal cord and to the motor nuclei of extraocular muscles, allowing the same signals to be sent to neck and eye muscles

(see Grantyn and Berthoz²⁶). Microstimulation at the site of putative excitatory burst neurons (EBNs), which are a subset of the SLBNs embedded within the complicated pontine network of signals for gaze control, produce slow, constant velocity changes in gaze accomplished by coordinated movements of the eyes and head. The ratio of eye/head contribution to the slow change in gaze direction depends upon the initial positions of the eyes in the orbits.²⁷

According to traditional views of PPRF function, the EBNs issue a saccadic eye movement command that is delivered, monosynaptically, to the extraocular motoneurons (eye pathway, FIGURE 4A). More specifically, the number of spikes in the saccade-related burst generated by the EBNs is tightly correlated with saccade amplitude. During coordinated eye-head movements, however, the spike count of EBNs is better correlated with gaze amplitude than with the eye component.²⁸ This observation also holds for the putative inhibitory burst neurons,^{8,29} which are thought to reflect the activity of EBNs.

Omnipause neurons (OPNs) located within *nucleus raphe interpositus*, a discrete collection of cells found along the midline of the PPRF, discharge tonically during fixation and are silent during saccades. They monosynaptically inhibit the EBNs³⁰ and, thus, gate the activity of cells generating a command for an eye movement. Selectively activating OPNs using microstimulation interrupt ongoing saccades or delays their onset.^{12,31,32} Paré and Guitton³³ stimulated the OPN region in cats during ongoing eye-head gaze shifts. They state that stimulation interrupted transiently both eye and head movements and that head deceleration was seen less frequently than gaze deceleration. It is difficult to assess the validity of these statements because data from only six trials are presented.

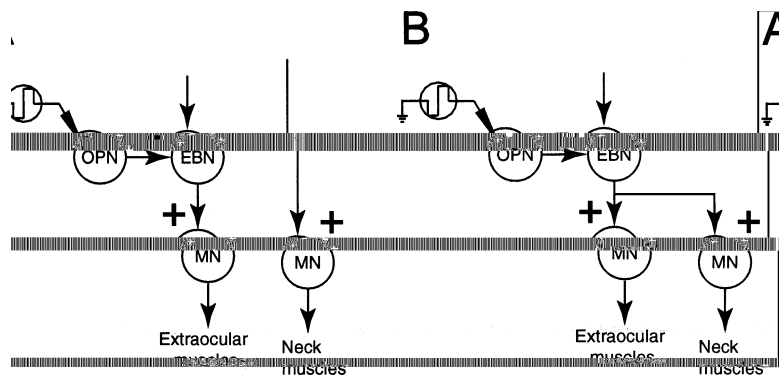


FIGURE 4. Rationale of the OPN stimulation experiment. Based on head-restrained experiments, the OPNs gate activity of EBNs that generate a command for an eye movement (pathway leading to extraocular muscles). For coordinated eye-head movements, if the command conveyed to neck motoneurons arises from structures above the level of EBNs (**A**), stimulation of the OPNs during a gaze shift will perturb only the eye component; the head movement should remain unattenuated. If the command relayed to the neck motoneurons is parsed downstream of the EBNs (**B**), stimulation of the OPNs is predicted to attenuate both eye and head trajectories.

To distinguish between the traditional and recently suggested roles of the EBNs in control of gaze, we exploited the antagonistic relationship of EBN and OPNs. If EBNs encode eye movements only, then the command signals to motoneurons innervating neck muscles must arise from structures upstream of the EBNs (FIG. 4A). Hence, stimulation of the OPNs during a gaze shift will perturb only the eye component; the head movement should remain unattenuated. If, on the other hand, the command relayed to the neck motoneurons is parsed downstream of the EBNs (FIG. 4B), stimulation of the OPNs is predicted to attenuate both eye and head movements.

FIGURE 5A illustrates the typical effects of OPN stimulation triggered on gaze onset. After a short latency, OPN microstimulation halted the change in gaze, typically until the end of stimulation, but had little, if any, effect on the head movement. Thus, the effect was mediated via a perturbation in the eye trajectory: the ocular component stopped in mid-flight and the eyes immediately began to counterrotate to stabilize gaze (gain of vestibulo-ocular reflex was 1). Stimulation prior to the onset of the gaze shift (FIG. 5B) delayed the onset of gaze, but the head movement was typically initiated during the microstimulation and, therefore, led gaze onset. The direction of gaze was stable during the stimulation because the eyes counterrotated in the orbits, thereby compensating for the ongoing head movement. At the end of the microstimulation train, the gaze shift was initiated by a saccadic eye movement in the same direction as the ongoing head movement.

To identify potential perturbations in head movements, head velocity traces were examined after aligning them on onset of head movement. FIGURE 6 superimposes head velocities of control (thin lines) and stimulation (thick lines) trials; both individual trials (left panels), and their mean ± 2 standard errors (right panels) are plotted. For trials when stimulation was triggered after gaze onset (top panels), the traces from stimulation trials are plotted until the onset of the resumed movement. For trials when stimulation was applied prior to gaze onset (bottom panels), the traces from stimulation trials are plotted until the approximate onset of the gaze shift. If EBNs transmit movement commands to the neck motoneurons as well as the extraocular motoneurons, the head velocity traces should deviate from the control traces and should reach zero velocity if the head movement were completely arrested. When stimulation is applied prior to gaze onset, the head velocity should remain at zero if the EBNs are the sole source of neck muscle motoneuron activation associated with the command for a gaze shift. The head velocity traces did not differ significantly during control and stimulation conditions regardless of when the OPNs were stimulated. These results provide strong support for the hypothesis that the EBNs participate in the eye pathway and that commands to motoneurons innervating the neck muscles originate from structures upstream of the EBNs.

Why then does the relationship between measures of the neural discharge of EBNs and saccade parameters deteriorate during combined eye-head movements? One possibility that has been suggested³⁴ is illustrated in FIGURE 7. Assume the EBNs generate commands for eye movements and that commands for moving the head originate in an independent, parallel pathway, as illustrated in Panel A. Movements of the head can influence the innervation signals reaching extraocular muscles via the vestibulo-ocular reflex (VOR) which is assumed to have a gain of 1 during the hypothetical eye and head movements illustrated. Panels B and C plot eye position and velocity, respectively, as a function of time for four movements. A signal to

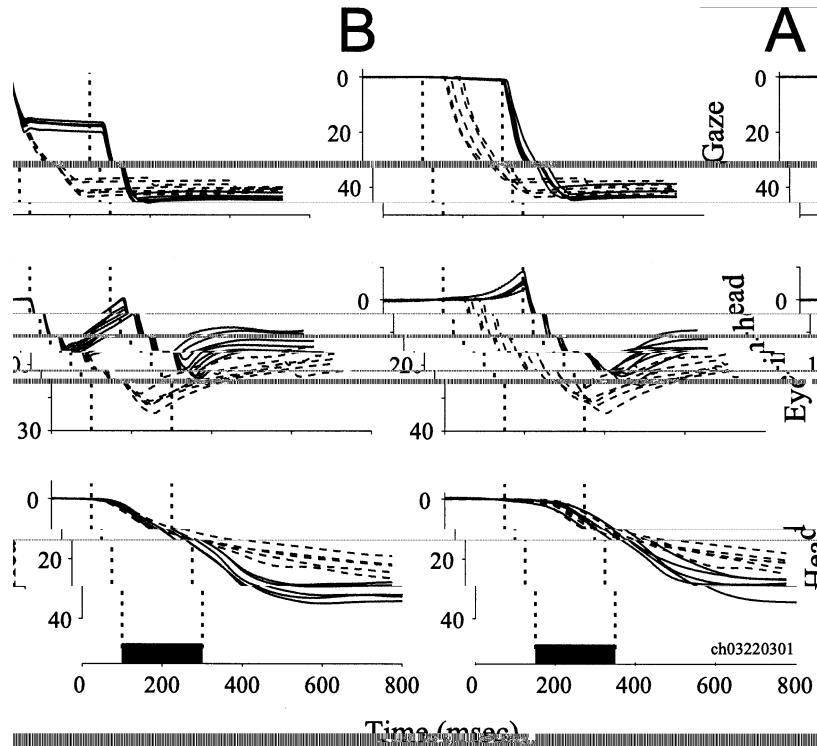


FIGURE 5. Stimulation of an OPN region during head-unrestrained gaze shifts attenuates only the eye component. Plots of control (*dashed lines*) and stimulation (*solid lines*) traces of horizontal gaze (*top*), eye-in-head (*middle*), and head (*bottom*) movements to a briefly flashed target. Stimulation was triggered either during (**A**) or before (**B**) gaze onset. Data was aligned on gaze onset in **A** and on cue to initiate movement in **B**. Stimulation onset always occurred at 150 ms for the trials displayed in **B**. Stimulation duration: 200 ms.

move the head did not occur during two of the movements (solid lines; head-restrained condition) when commands to move the eyes 20° and 10° to the right were issued. Commands to move the head 30° to the right and the eyes 20° to the right occurred on the other two trials (dashed lines; head-unrestrained condition).

In the absence of a head movement, measures of executed eye movements can serve as *estimates of the saccadic commands* transmitted to the extraocular motoneurons from the EBNs. This is possible because the motoneurons received no additional inputs from other oculomotor subsystems (e.g., vergence, pursuit, vestibular) during the time when the saccade command is being implemented. Thus, the amplitude (peak velocity) of the movement is tightly correlated with the number of spikes (peak discharge rate) in the saccade-related burst, allowing us to examine how information about the parameters of saccadic movements may be represented in the activity of EBNs.

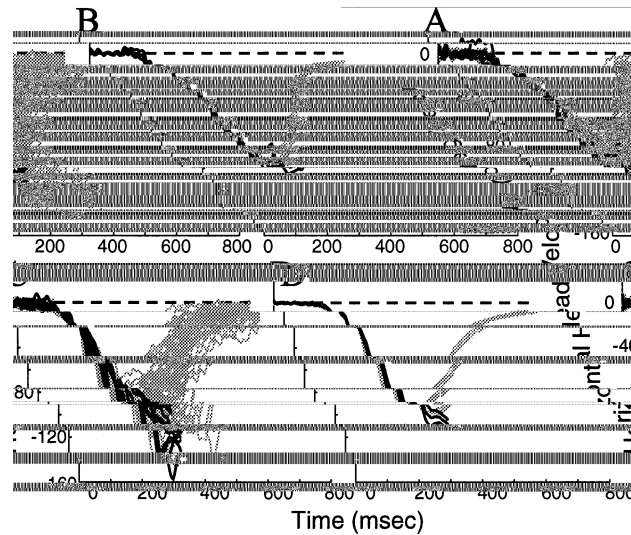


FIGURE 6. Lack of effects of stimulation of an OPN region on head movements. Plots display horizontal head velocity as a function of time for control (*thin, gray lines*) and stimulation (*thick, black lines*) trials. All traces, aligned on head onset, are from same stimulation site as that shown in FIGURE 4. Individual control and stimulation trials (**A,C**) as well as their means and ± 2 standard errors from the mean (**B,D**) are illustrated. (**A,B**) Stimulation was triggered on gaze onset. Stimulation trials are shown until the onset of the resumed movement. (**C,D**) Stimulation was triggered prior to gaze onset. One hundred milliseconds before head onset and 280 ms of the head movement are plotted for the stimulation trials; this corresponds roughly to the time period prior to the onset of the gaze shift. Note that the control traces show head movements when a gaze shift was executed.

When the motoneurons are receiving inputs from the other oculomotor sub-systems while a saccade is being generated, measurements of the amplitude or velocity of the executed saccade will be *unreliable and misleading estimates* of the command issued by EBNs. Correlations of EBN activity with the amplitude, velocity, and duration of the executed eye movement cannot be used to make inferences about the motor signals being generated by EBNs because of the dissociation between the command that is issued and the movement that is executed. This dissociation is illustrated by the two simulated movements shown with dashed lines (FIG. 7, B and C). The EBNs generated identical commands for a 20° movement on both trials. Similarly, the command for a 30° head movement was the same except that it was issued 60 ms earlier for one of the movements. Because the head was moving and the VOR was active with a gain of 1, the executed eye movements had amplitudes of 18° and 15° and peak velocities of $350^\circ/\text{s}$ and $275^\circ/\text{s}$ instead of the 20° amplitude and $375^\circ/\text{s}$ peak velocity that would have occurred if the head had not been moving. Because of the dissociation between the issued command and the executed movement, relationships between movement parameters and the activity of any neuron issuing an oculomotor command above the level of the motoneurons are not informative in the absence of accurate information about other influences on

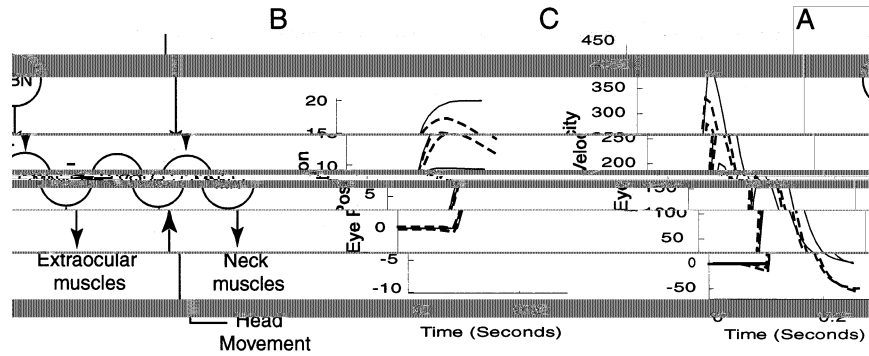


FIGURE 7. (A) Excitatory burst neurons (EBNs) generate commands for eye movements that are transmitted monosynaptically to extraocular muscle motoneurons (MN). Signals to neck muscle motoneurons are assumed to originate in an independent parallel pathway. Movements of the head can influence the innervation of extraocular muscles via the vestibulo-ocular reflex (VOR) which is assumed to have gain of 1. (B) Plots of eye position as a function of time for four hypothetical eye movements. *Solid lines*: movements occurring when commands to move the eye 20° to the right or 10° to the right were generated by EBNs and when a command to move the head was not issued. *Dashed lines*: movements occurring when a command to move the eye 20° to the right was issued in conjunction with a command to move the head 30° to the right. The difference in the amplitude of the two eye movements occurs because the command to move the head occurred 60 ms earlier in one case. (C) Plots of eye velocity as a function of time for the same four movements.

motoneuron activity. Thus, the data obtained by correlating the activity of neurons above the level of motoneurons with various movement parameters has limited usefulness.

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