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# Visual Responses on Neck Muscles Reveal Selective Gating that Prevents Express Saccades

**Brian D. Corneil,[1,2,*] Etienne Olivier,[3] and Douglas P. Munoz[1]**
[1]CIHR Group in Sensory-Motor Systems
Centre for Neuroscience Studies
Department of Physiology
Queen's University
Kingston, Ontario K7L 3N6
Canada
[2]Department of Physiology and Pharmacology
Department of Psychology
University of Western Ontario
London, Ontario N6A 5C1
Canada
[3]Laboratory of Neurophysiology
School of Medicine
University of Louvain
1200 Brussels
Belgium

## Summary

**Express saccades promote the acquisition of visual targets at extremely short reaction times. Because of the head's considerable inertia, it is unknown whether express saccades are accompanied by a parallel command to the head. Here, by recording electromyographic (EMG) activity from monkey neck muscles, we demonstrate that visual target presentation elicits time-locked, lateralized recruitment of neck muscles at extremely short latencies (55–95 ms). Remarkably, such recruitment not only accompanies express saccades, but also precedes nonexpress saccades, occasionally by up to 150 ms. These results demonstrate selective gating of components of descending commands from the superior colliculus to prevent express saccades yet permit recruitment of a head orienting synergy. We conclude that such selective gating aids eye-head coordination by permitting force development at neck muscles while a decision to commit to a gaze shift is being made, optimizing the contribution of the more inertial head to the ensuing gaze shift.**

## Introduction

A central issue in sensorimotor control is how the brain controls rapid yet accurate movements in a complex and changing environment. Insights into how the brain copes with these conflicting demands can be gained by examining the reaction times taken to perform various tasks. An elemental psychophysical observation is that reaction times usually exceed the conduction time of the shortest neural pathway between sensory transducer and motor effector (Luce, 1991), permitting the brain the time to decide upon a contextually appropriate course of action. For example, reaction times for visually guided saccades usually range between 150 and 350 ms (Carpenter, 1988), even though both humans and monkeys can generate express saccades at much shorter reaction times (70–100 ms in monkeys [Fischer and Boch, 1983]; 80–120 ms in humans [Fischer and Weber, 1993]).

The neurophysiological events preceding saccades generated at express and nonexpress reaction times have been elucidated. Within the intermediate and deep layers of the superior colliculus (dSC), dual bursts of activity aligned to both visual and motor events precede saccades generated at nonexpress reaction times (Sparks and Hartwich-Young, 1989). Prior to express saccades, these visual and motor bursts merge into a single, unified burst (Edelman and Keller, 1996; Dorris et al., 1997; Sparks et al., 2000), as if the incoming visual signal is transformed directly into the saccadic motor command. Although such observations are consistent with the notion that express saccades are a class of reflexive saccades, express saccades are predicated on the advanced preparation of saccadic motor programs (Paré and Munoz, 1996) attributed to higher cognitive processes typically not required for reflexes. Regardless, the paramount importance of retinal stability for foveal vision has led to the development of brainstem neural circuits downstream from the dSC that tightly constrain the timing of saccade generation (Munoz et al., 2000; Scudder et al., 2002), usually preventing express saccades and permitting the brain more time to decide where and when to shift the visual axis.

Recent work in the monkey demonstrates that the dSC fulfills a more general role in controlling gaze shifts composed of coordinated eye-head movements (Segraves and Goldberg, 1992; Freedman et al., 1996; Freedman and Sparks, 1997a; Klier et al., 2001). Eye-head gaze shifts are a model system for understanding how the brain controls multisegmental motion, as the unitary gaze signal within the dSC must be decomposed into the appropriate motor commands to move the eyes and head. Although the neural mechanisms underlying such decomposition remain obscure, one can assess whether the generation of head orienting commands is subjected to the same timing constraints as saccadic eye movements by comparing the timing of head orienting commands accompanying saccades generated at express and nonexpress latencies. To do this, we recorded neck EMG activity in either head-restrained or unrestrained monkeys performing a simple oculomotor "gap" task (Saslow, 1967) customized to promote the generation of express saccades (Figure 1A, Experimental Procedures; Fischer and Weber, 1993). Recording neck EMG circumvents inertial, anatomical, and neuromechanical complexities of the head plant (Peterson and Richmond, 1988) and provides insights into the neural control of head motion beyond what could be gained by examining head movement kinematics alone (Corneil et al., 2001, 2002a, 2002b). Contrary to what would have been expected if the eye and head were to share similar timing constraints, we found that short-latency neck EMG responses time-locked to visual target presentation accompany not only express saccades, but also saccades generated at much longer reaction times.

*Correspondence: bcorneil@uwo.ca

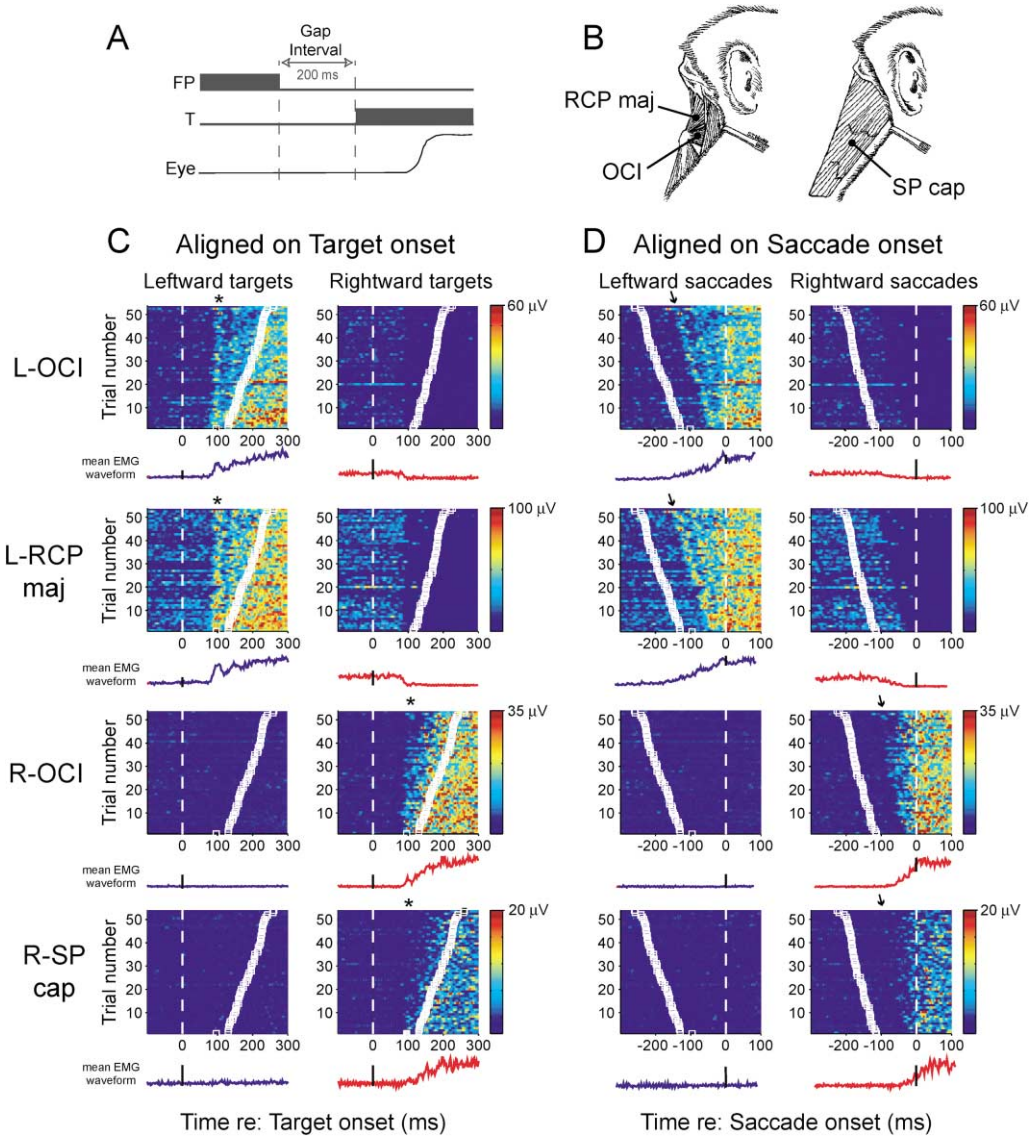


Figure 1. Neck EMG during the Gap Task

(A) Monkeys looked from a central fixation point (FP) to a peripheral target (T) randomly presented to either the right or left, maintaining central fixation during the 200 ms "gap" interval between FP disappearance and T presentation.

(B) Schematic line drawings of implanted neck muscles examined in this manuscript.

(C) EMG activity for left obliquus capitis inferior (L-OCI), left rectus capitis posterior major (L-RCP maj), right OCI (R-OCI), and right splenius capitis (SP cap) aligned on the presentation of targets located 30° to the left or right (white vertical dashed lines denote target presentation). Within the subplots, each row conveys the EMG activity recorded during a single trial, and the trials are sorted by the reaction time of the ensuing saccade (white squares). Asterisks are placed above the transient burst of neck EMG aligned on target presentation.

(D) Neck EMG for the same muscles, aligned on saccade onset (white vertical dashed line). White squares denote the time of target presentation, prior to saccade onset. Slanted arrows point to bursts of neck EMG time-locked to target presentation. The mean EMG waveforms across all trials are shown beneath each subplot. Bars aligned on target presentation (C) or saccade onset (D) denote 10 μV for L-OCI and R-OCI, 20 μV for L-RCP maj, and 5 μV for R-SP cap (these bars may be different sizes for leftward and rightward targets).

These results attest to a previously unrealized capacity of the circuitry downstream from the dSC to selectively deliver a component of an orienting command to the head regardless of the reaction time of the ensuing saccade.

## Results

### Visual Responses on Neck Muscles

We report EMG activity recorded from a number of dorsal neck muscles: obliquus capitis inferior (OCI), rectus capitis posterior major (RCP maj), and splenius capitis (SP cap) (Figure 1B). All three muscles participate in turning the head in the ipsilateral horizontal direction and also play a subsidiary role in pitching the head up (Corneil et al., 2001). OCI and RCP maj are suboccipital muscles that invest the deepest layer of the dorsal neck musculature, whereas SP cap is a much larger and more superficial muscle spanning multiple cervical vertebrae (Richmond et al., 2001).

Performance in the gap task (Figure 1A) elicited stereotyped changes in EMG recorded from all three dorsal

neck muscles. In Figure 1C, neck EMG from bilateral OCI, left RCP maj, and right SP cap is aligned on target presentation, segregated by whether the target was presented 30° to the left or right. Despite head restraint, the side of target presentation elicited patterns of neck EMG that diverged approximately 80–90 ms after target presentation in this example. Transient bursts of activity of about 20 ms in duration consistently appeared in neck muscles after ipsilateral target presentation (asterisks in Figure 1C, above panels of neck EMG following targets presented in ipsilateral visual hemifield). This transient burst was followed by an interval of tonic activity that persisted to the time of saccade onset, after which neck EMG increased still further, likely due to the acquisition of eccentric eye positions (André-Deshays et al., 1988; Corneil et al., 2002a). For the left OCI and RCP maj, neck EMG decreased about 80–90 ms following contralateral target presentation and remained low for the duration of the trial. Although these lateralized patterns of neck EMG recruitment mimic those accompanying horizontal head turns (Corneil et al., 2001), a remarkable feature is that they occurred at a fixed latency after target presentation, regardless of the ensuing saccadic reaction time. In extreme cases, lateralized patterns of neck EMG were observed up to 150 ms *before* saccade onset (Figure 1C, top rows). Another way of visualizing this phenomenon is to align neck EMG on saccade onset (Figure 1D). Here, the time-locked nature of the lateralized neck EMG relative to target presentation is apparent as it tracks backward, earlier relative to longer-latency saccades (Figure 1D, arrows).

There is some variability in the data presented in Figures 1C and 1D. In particular, the left neck muscles were more active than the right neck muscles prior to target presentation. As a consequence, the transient bursts following ipsilateral target presentation and the decrease in neck EMG activity following contralateral target presentation were more apparent on the left neck muscles. Such asymmetries occurred because the body, in this example, was turned slightly to the right under the chair-restrained head, leading to the development of postural activity on the left neck muscles similar to what we have reported previously (Corneil et al., 2001). Although this posture was consistent during the experimental session shown in Figures 1C and 1D, both animals adopted variable idiosyncratic postures across different experimental sessions, which could be biased to either direction.

Another source of variability apparent in Figures 1C and 1D is the occasional lack of transient neck EMG bursts following ipsilateral target presentation. This was particularly evident for some trials of R-SP cap activity, but also occurred for R-OCI prior to longer latency saccades. Note, however, on such trials that the activity of the leftward muscles still decreased prior to these rightward saccades, implying that neck EMG still changed at a time-locked latency following visual target presentation. The presence or absence of neck EMG bursts on a particular muscle depended on three factors. First, as described above, body posture influenced the magnitude of neck EMG bursts following ipsilateral target presentation. Second, as will be shown below, the frequency of transient neck EMG bursts was slightly less in the larger SP cap muscle than the suboccipital OCI and RCP maj muscles, consistent with the recruitment sequence observed during natural head turns (Corneil et al., 2001). Finally, as will be described below, there is a relationship between the magnitude of the transient neck EMG burst and the ensuing saccadic reaction time. Thus, although the presence or absence of neck EMG bursts on a given trial on a particular muscle depends on a number of factors, the patterning of neck EMG activity across multiple muscles still display consistent, stereotypical changes at a time-locked latency following visual target presentation.

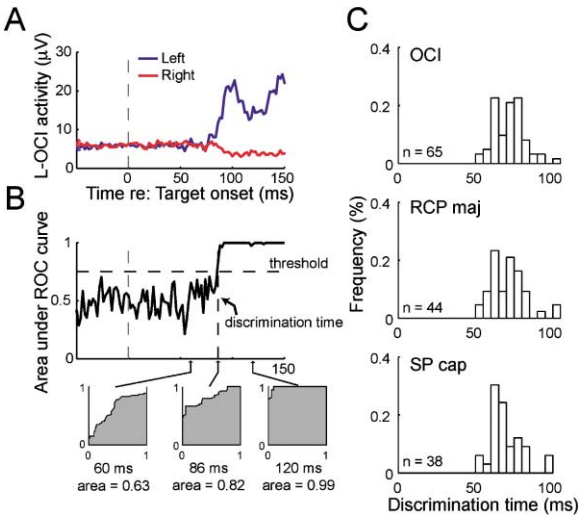


Figure 2. Analysis of Discrimination Time of Target Laterality from Neck EMG

(A) Overlapping mean EMG waveforms for L-OCI aligned on the onset of leftward (blue) or rightward (red) targets, using data from Figure 1C aligned on target presentation (vertical dashed line). The mean EMG waveforms diverge around 80 ms after target presentation.

(B) ROC analysis was performed every 2 ms from 300 ms before to 300 ms after target presentation and is shown here from 50 ms before to 150 ms after target presentation. The area under the ROC curve (shown for selected periods in the insets) indicates the probability that an ideal observer could correctly discern the side of target presentation based on the differential distribution of neck EMG activity at that instant. Discrimination (86 ms in this example) occurred if the area under the ROC exceeded a threshold of 0.75 (horizontal dashed line) for at least 10 of the following 16 ms.

(C) Frequency histograms (bin width = 5 ms) of the divergence times for OCI, RCP maj, and SP cap pooled across monkeys, the side of muscle recording, and multiple experimental sessions.

### Timing and Reliability of Visual Responses on Neck Muscles

We employed receiver operating characteristic (ROC) analysis (Bradley et al., 1987) to determine when after target presentation neck EMG discriminated between ipsilaterally and contralaterally presented targets (Experimental Procedures). In Figure 2A, the mean target-aligned EMG waveforms for L-OCI are contrasted for leftward and rightward saccades, and the time course of the area under the ROC curve for these data is shown in Figure 2B. Note around 80 ms after target presentation that the area increased sharply to values near 1, implying that the activity recorded from L-OCI disclosed the side of target presentation. We defined the discrimination time as the time after target presentation when this area

Table 1. Reliability, Discrimination Times, and Burst Duration of Lateralized Changes in Neck EMG Following Visual Target Presentation

| Muscle | Data Set | Reliability | Discrimination Time (ms) | Burst Duration (ms) |
|---|---|---|---|---|
| OCI | all saccades, head restrained | 90% (65/72) | 72 ± 12 | 20 ± 8 |
| | saccades > 100 ms | 90% (56/62) | 78 ± 12 | 18 ± 6 |
| | all saccades, head unrestrained | 95% (21/22) | 78 ± 12 | 20 ± 4 |
| RCP maj | all saccades, head restrained | 83% (44/53) | 72 ± 12 | 20 ± 8 |
| | saccades > 100 ms | 80% (37/46) | 70 ± 12 | 18 ± 6 |
| | all saccades, head unrestrained | 85% (12/14) | 74 ± 14 | 18 ± 4 |
| SP cap | all saccades, head restrained | 76% (38/50) | 74 ± 16 | 20 ± 8 |
| | saccades > 100 ms | 68% (30/44) | 78 ± 16 | 20 ± 6 |
| | all saccades, head unrestrained | 91% (11/12) | 78 ± 14 | 18 ± 6 |

Values are shown separately for the three different muscles and are presented separately for all head-restrained data, all head-restrained data following the exclusion of express saccades with reaction times less than 100 ms, and all head-unrestrained data. Reliability quantifies the fraction of experimental sessions (given in parentheses) in which ROC analysis yielded a discrimination time. Values for the discrimination time and burst duration are given as means ± standard deviations. The number of head-restrained experimental sessions is lower after excluding express saccades because, on some sessions, there remained fewer than 10 saccades with reaction times greater than 100 ms.

exceeded a threshold of 0.75 for at least 10 of the ensuing 16 ms, providing a measure of the time of lateralized neck muscle recruitment. Over all experimental sessions with a minimum of 10 trials per direction, discrimination occurred reliably in all three muscles, with discrimination times ranging between 55 and 100 ms (Table 1; Figure 2C).

A concern about these discrimination times is that they could result solely from express saccades generated at short reaction times, because increases in neck EMG accompany saccades and eccentric orbital positions (André-Deshays et al., 1988, 1991; Corneil et al., 2001). We therefore repeated the ROC analysis after excluding express saccades, defined as saccades with reaction times less than 100 ms (Experimental Procedures). In Figure 3, we compare directly the mean EMG waveforms and ROC curves from another experimental session in which a high proportion of express saccades were generated to the left. The exclusion of express saccades imparted only minor effects on both the mean EMG waveforms and on the discrimination time determined by the ROC analysis for both L-OCI (Figures 3A and 3B) and R-OCI (Figures 3C and 3D). Such trends were apparent over all experimental sessions in which a minimum of 10 trials remained after exclusion of express saccades (Table 1). Thus, short latency, lateralized changes in neck EMG following visual target presentation were not artifacts of express saccades, but rather occurred at a fixed interval after target presentation regardless of the reaction time of the ensuing saccade.

We also determined the duration of the transient burst of neck EMG observed in muscles ipsilateral to the side of target presentation. From the mean EMG waveform in those experimental sessions in which a discrimination time was derived, we measured the width of the burst at half of its peak value, relative to the baseline determined over the 50 ms preceding target presentation. Over all experimental sessions, the duration of the burst in all muscles was around 20 ms (Table 1). Furthermore, although the durations of such bursts were slightly shorter after express saccades were excluded, such changes were not significant (Table 1; for all muscles, paired t test of burst durations with and without express saccades, $p > 0.2$).

Finally, we also analyzed the reliability of discrimination as a function of target eccentricity, which ranged between 5° and 35° with the head restrained. For RCP maj, discrimination occurred significantly more frequently following the presentation of more eccentric targets (Table 2; $\chi^2$ test, $p < 10^{-3}$). A similar, albeit insignificant, trend is apparent for OCI (Table 2; $\chi^2$ test, $p = 0.27$) but not SP cap (Table 2; $\chi^2$ test, $p = 0.9$). While we had expected that discrimination would occur more reliably with more eccentric targets, as larger gaze shifts are usually accompanied by larger head movements (Freedman and Sparks, 1997b), it should be noted that we did not scale target size or brightness to keep the salience constant across different eccentricities (Romavo and Virsu, 1979). We suspect such M scaling would have increased the reliability of neck EMG responses to more eccentric targets. A similar analysis of the discrimination timing and reliability depending on whether the ipsilateral target was presented in the upper or lower visual hemifield revealed no significant relationship (discrimination time: t test, $p > 0.4$ for all muscles; reliability: $\chi^2$ test, $p > 0.58$ for all three muscles). Even though the neck muscles we recorded play a subsidiary role in upward head pitches (Corneil et al., 2001), the lack of changes in the pattern of neck muscle recruitment across different target elevations that could vary by as much as 90° in radial angle is consistent with previous findings that the vertical contribution of the head to oblique gaze shifts is less than the horizontal contribution (Freedman and Sparks, 1997b).

### Neck EMG Predicts Saccadic Reaction Time

Despite the consistency in discrimination times, a closer examination of Figures 1C and 1D suggests an inverse relationship between the magnitude of muscle activation and the reaction time of ensuing saccades to ipsilateral targets (i.e., leftward saccades for L-OCI, rightward saccades for R-OCI). On a trial-by-trial basis in Figure 4A, we contrasted the reaction time of the ensuing ipsilateral saccade versus the magnitude of neck EMG integrated over the first 10 ms following the discrimination time determined by the ROC analysis; least squares regression lines revealed significant inverse relationships for all four muscles ($p < 0.0005$ for L-OCI, L-RCP maj, R-OCI; $p < 0.05$ for R-SP cap). Over the sample in which ROC analysis provided a discrimination time, a

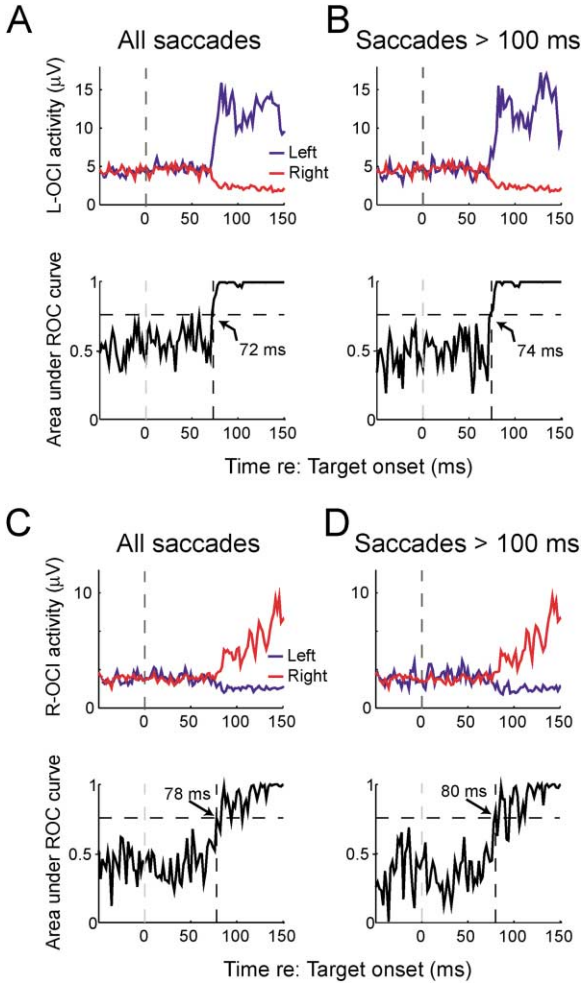


Figure 3. Analysis of Neck EMG with and without Express Saccades

Comparison of mean neck EMG waveforms and area under the ROC curve for L-OCI (A and B) and R-OCI (C and D) before (A and C) and after (B and D) excluding express saccades. Same format as Figure 2. The exclusion criteria removed 22 of 43 saccades to a target located at 15° left, and 3 of 39 saccades to a target located at 15° right, yet the mean EMG waveforms and the derived discrimination times were very similar.

similar significant inverse relationship was observed in 19 of 65 sessions for OCI, 11 of 44 sessions for RCP maj, and 8 of 38 sessions for SP cap (Figures 4B and 4C). Furthermore, the distributions of slopes and correlation coefficients for all least squares regression lines were significantly skewed in the negative direction (t test versus zero, $p < 0.005$ for all distributions). These results indicated a predominantly inverse relationship between neck EMG activity and the ensuing saccadic reaction time for ipsilateral targets. This inverse relationship persisted even after excluding ipsilateral express saccades (not shown), emphasizing again that this phenomenon was not simply due to trials with the shortest saccadic reaction times. This inverse relationship is observed only if the ensuing saccade goes in the direction ipsilateral, not contralateral, to the muscle under consideration.

Table 2. Reliability of Lateralized Changes in Neck EMG Aligned on Target Presentation, across Different Ranges of Target Eccentricity

| | Target Eccentricity | | |
|---|---|---|---|
| Muscle | 5 to 15° | 15 to 25° | >25° |
| OCI | 88% (14/16) | 89% (31/35) | 100% (20/20) |
| RCP maj | 56.25% (9/16) | 91% (21/23) | 100% (14/14) |
| SP cap | 75% (12/16) | 80% (16/20) | 78% (11/14) |

## Visual Responses on Neck Muscles during Eye-Head Gaze Shifts

In a limited number of sessions, we released the monkeys' heads and examined neck EMG accompanying eye-head gaze shifts. For target eccentricities up to 35°, the patterns of head-unrestrained neck EMG aligned on target presentation were identical to that already presented when the head was restrained. In Figure 5A, neck EMG is aligned to the presentation of an extremely eccentric target located 90° to the left. Even with such an eccentric target presented in otherwise complete darkness, neck EMG became lateralized shortly after target presentation, increasing on the left muscles and decreasing on the right muscles. Although these lateralized changes were more variable and not as prominent as in Figure 1, likely because the targets were not M scaled for size or saliency, ROC analysis revealed discrimination times of 86 ms for L-OCI, 90 ms for L-RCP maj and R-OCI, and 78 ms for R-SP cap.

With the head unrestrained, it is more difficult to determine whether neck EMG lateralization occurred at a fixed interval relative to target presentation or to the onset of the gaze shift or head motion. In this example, leftward head motion usually preceded leftward gaze shifts, with head movements starting between 52 ms before to 34 ms after gaze shift onset (mean head-regaze lead time = 20 ± 16 ms; compensatory eye movements from the vestibulo-ocular reflex [VOR] kept gaze stable when the head movement led gaze shift onset). When realigned to gaze shift onset (Figure 5B), it is apparent that neck EMG did not become lateralized at a fixed interval relative to the gaze shift, as changes in neck EMG preceded gaze shifts by anywhere between 10 and 100 ms. Surprisingly, when neck EMG is realigned to the onset of head motion (Figure 5C), there is again no fixed relationship between the timing of neck EMG recruitment and head motion onset: L-OCI and L-RCP maj became active about 70–100 ms prior to longer latency head movements and about 30–50 ms prior to shorter latency head movements. Despite the variability in the timing of neck EMG recruitment relative to head movement onset, the mean EMG waveforms for L-OCI and L-RCP maj demonstrated a local peak of activation around 30 ms prior to head movement onset (Figure 5C), presumably providing the main forces for initiating the head movement.

To examine further the variability in the timing of neck EMG lateralization relative to head movement onset, we performed ROC analysis on target-locked changes in neck EMG activity after segregating the database on whether the onset of head motion was less than (Figure 6A) or greater than (Figure 6B) the median leftward head movement reaction time (154 ms). ROC analysis for L-OCI revealed that discrimination times differed very

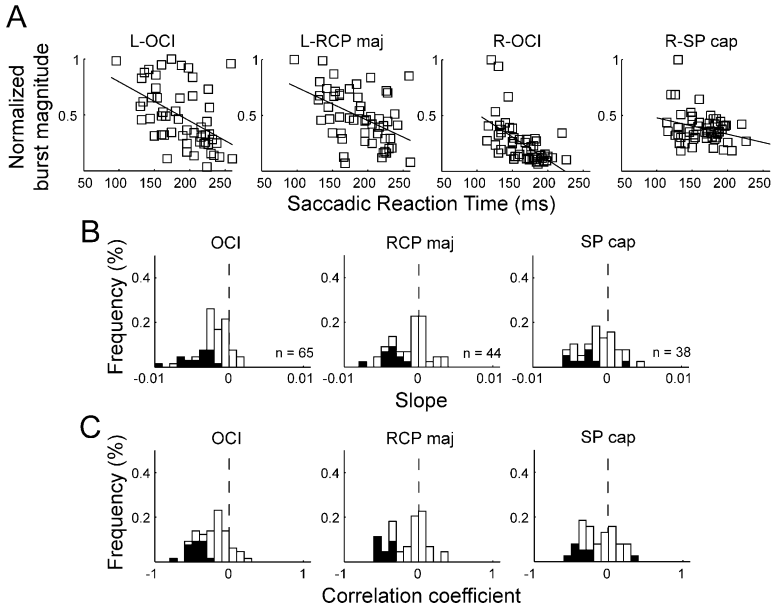


Figure 4. The Magnitude of Target-Aligned EMG Activity Is Inversely Related to the Ensuing Reaction Time

(A) Trial-by-trial correlation between saccadic reaction time and the magnitude of EMG activity integrated over the first 10 ms following the discrimination time determined by the ROC analysis, using the data from Figure 1C for ipsilaterally presented targets. The integrated EMG magnitude is normalized to the peak observed value to permit comparison across different muscles and monkeys. The solid line denotes a significant regression line ($p < 0.0005$ for L-OCI, L-RCP maj, and R-OCI; $p < 0.03$ for SP cap).

(B and C) Frequency histograms of the slopes (B) and r values (C) of the EMG magnitude-reaction time regressions over multiple experimental sessions, for all three muscles, pooled across experimental sessions, monkeys, and side of muscle recording. All distributions show a significant negative skewing (t test versus zero, $p < 0.005$) for slopes and r values, indicating a predominantly inverse relationship between burst magnitude and reaction time. Solid bins denote significant linear regressions within individual sessions ($p < 0.05$).

little with shorter or longer latency head movements (84 versus 86 ms, respectively), and similar results were observed for the other muscles (L-RCP maj, 88 versus 104 ms; R-OCI, 84 versus 92 ms; R-SP cap, 78 versus 96 ms). It is important to stress that we are considering only the timing of initial neck EMG lateralization, which occurred here at a relatively fixed interval after target presentation compared to gaze and head onset. In this example, the cumulative effect of lateralized neck EMG over the ensuing 30–70 ms almost always produced head motion prior to gaze shift onset.

Across the sample of eye-head gaze shifts made to targets between 15° and 90°, the reliability and timing of lateralized changes in neck EMG resembled the head-restrained condition, as did the durations of the transient bursts on the muscles ipsilateral to the target (Table 1; burst durations were not significantly different from the head-restrained data: t test, $p > 0.55$ for all muscles). As observed when the head was restrained, the magnitude of neck EMG integrated over 10 ms after the discrimination time was inversely related to the reaction time of the ensuing gaze shift (t test of slopes and correlation coefficients versus zero, $p < 0.05$ for OCI and RCP maj; $p = 0.20$ for SP cap). The magnitude of this integrated neck EMG was also inversely related to the reaction time of the accompanying head movement (t test of slopes and correlation coefficients versus zero, $p < 0.05$ for all muscles) but somewhat surprisingly was not systematically related to other kinematic aspects of head motion (e.g., amplitude, peak velocity, or peak acceleration). However, a strong positive relationship between neck EMG and head amplitude, peak velocity, and peak acceleration was observed when neck EMG was integrated over a period from 75 ms before head motion onset to the time of peak head velocity, as reported previously (Corneil et al., 2001) ($p < 0.05$ for all muscles and kinematics). Overall, the data set obtained with the head unrestrained emphasizes that the time-locked changes in neck EMG following visual target presentation are not simply artifacts of head restraint but rather are common features of eye-head gaze shifts and attest to neck muscle recruitment well in advance of gaze shift onset.

## Discussion

Our data demonstrate that the presentation of a visual target elicits short latency, time-locked neck muscle recruitment, activating ipsilateral muscles and inhibiting contralateral muscles relative to the side of target presentation. This consistent and lateralized pattern of neck EMG differs fundamentally from the habituating and bilateral patterns of neck EMG elicited by the acoustic startle reflex (Brown et al., 1991; Siegmund et al., 2001). Instead, regardless of whether the head is restrained or not, the presentation of a visual target leads to recruitment of neck muscles in a pattern known to generate head turns (Corneil et al., 2001).

The most surprising result is that such lateralized patterns of neck EMG are recruited time-locked to visual target presentation, usually well in advance of saccadic eye movements or gaze shifts. With the exception of express saccades wherein eye movements are nearly synchronous with this neck muscle recruitment, this suggests that a component of an orienting command begins to act selectively on the head plant, but not the eye plant, well before saccade onset. This supposition hinges critically on how appropriate it is to use eye movements as a proxy for the neural commands issued to the eye plant. Is it possible that the eye plant received analogous, short latency orienting commands that, perhaps either because of ocular biomechanics or cocontraction of other extraocular muscles, did not result in eye motion?

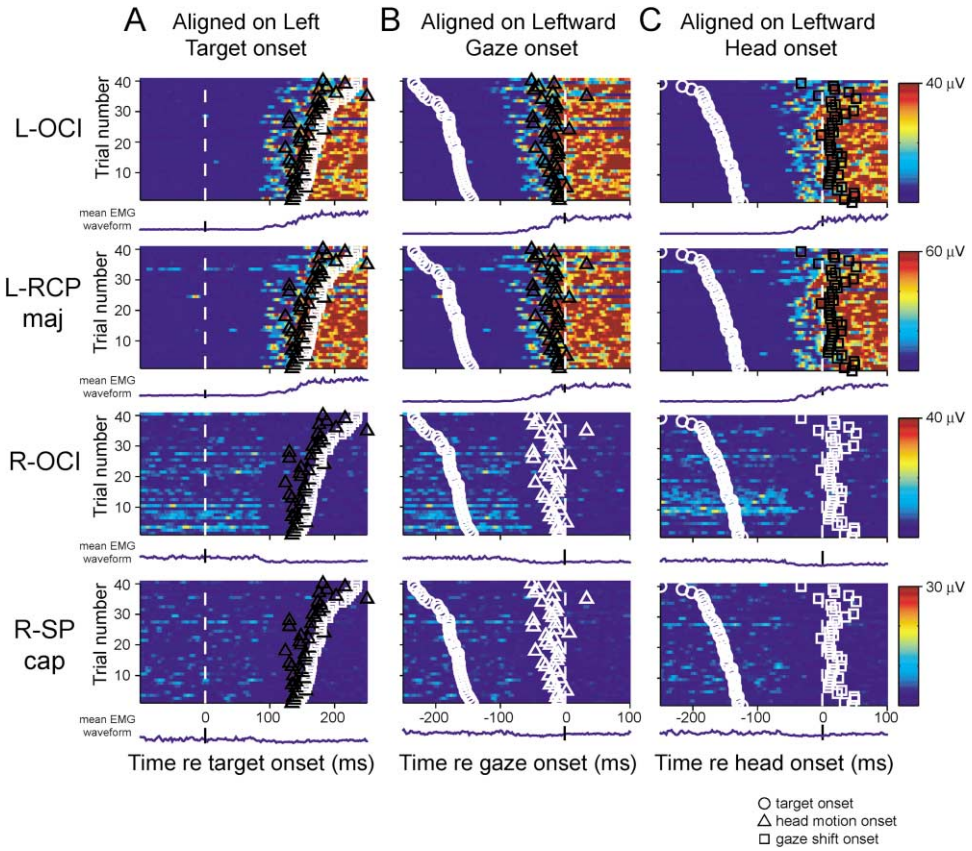


Figure 5. Neck EMG during Eye-Head Gaze Shifts

Activity for L-OCI, L-RCP maj, R-OCI, and R-SP cap during eye-head gaze shifts to targets located 90° to the left (rightward data is not shown for simplicity). Same format as Figures 1C and 1D, with data aligned on target presentation (A), gaze shift onset (B), and head motion onset (C; trials are sorted by head motion reaction time). The small symbols in the various subplots denote target presentation (circles), head motion onset (triangles), or gaze shift onset (squares); different colors are used to improve resolution. Note that head motion onset usually led gaze shift onset. Bars on mean EMG waveform plots aligned on target presentation (A), gaze shift onset (B), or head movement onset (C) denote 20 μV for L-OCI and L-RCP maj, 10 μV for R-OCI, and 5 μV for R-SP cap.

A number of observations argue against these possibilities. First, numerous studies recording directly from extraocular muscle motoneurons have never observed time-locked bursts of activity emitted in response to visual target presentation (Fuchs and Luschei, 1970; Robinson, 1970; Van Gisbergen et al., 1981; Fuchs et al., 1988), even using visual stimuli similar to ours (Ling et al., 1999; Sylvestre and Cullen, 1999; Cullen et al., 2000). Second, the biomechanics of eye motion are such that recruitment of as little as 2–3 motor units or firing changes on the order of 5 Hz are sufficient to move the eyes by 1° (Goldberg et al., 1998), hence recordings of eye position are very sensitive indicators of changes in the output of extraocular muscle motoneuron pools. Indeed, the 20 ms duration of the neck EMG bursts aligned on target presentation approximates the burst duration emitted by extraocular muscle motoneurons for a 5° saccade (Schiller, 1970). Because the eyes were perfectly stable leading up to saccade initiation, it is highly unlikely that lateralized recruitment of extraocular muscles were being generated. Finally, recordings have also established that antagonist muscle motoneurons decrease firing only immediately prior to and during saccades (Schiller, 1970; Robinson, 1970; Fuchs and Luschei, 1970), and thus cocontraction of extraocular muscles also cannot explain our results. Together, these results strongly suggest that, with the exception of express saccades, the eye plant does not receive the time-locked, lateralized orienting command delivered to the head plant after visual target presentation.

A likely neural mechanism for such selective gating of an orienting command involves both the intermediate and deep layers of the superior colliculus (dSC) and the brainstem omni-pause neurons (OPNs). The dSC has long been implicated in mediating orienting in many species (Ingle, 1973) and is thought to act via tectal projections to the brainstem saccadic generator in monkeys and humans (Munoz et al., 2000). However, OPNs exert a tonic inhibition over the saccadic burst generator and cease firing only immediately prior to saccade onset (Scudder et al., 2002). Contemporary models of eye-head gaze control incorporate dual pathways downstream from the dSC, one of which drives the head premotor circuitry without being gated by the OPNs (Guitton et al., 1990; Galiana and Guitton, 1992; Phillips et al., 1995; Goossens and van Opstal, 1997; Freedman, 2001) (whether the dSC is in a gaze feedback loop, or whether a common drive downstream from the dSC is

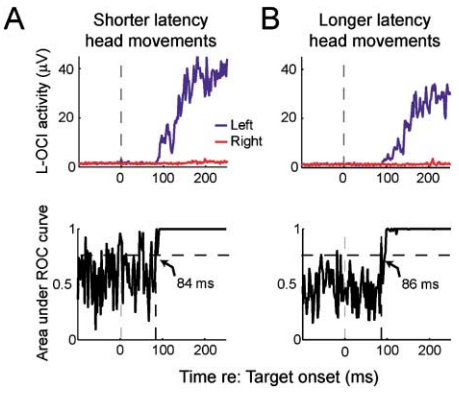


Figure 6. Analysis of Neck EMG for Short and Long Latency Head Movements

Comparison of mean neck EMG waveforms and area under the ROC curve for L-OCI, for data segregated based on whether the reaction time for head motion onset was less than (A) or greater than (B) the median leftward head movement reaction time (154 ms).

issued to both the eyes and head are not addressed by the current results). Such dual pathways presumably endow the brainstem circuitry downstream from the dSC with the flexibility to drive the eyes and head separately, and while such architecture could account for our results, no model has ever predicted time-locked lateralized patterns of neck EMG in response to visual target presentation.

Despite the neuromechanical delays inherent to head motion, a fundamental characteristic of the eye-head gaze shifting system is that the onset of head motion can lag, be synchronous with, or lead eye motion during gaze shifts (Figure 5; Fuller, 1992; Corneil and Munoz, 1999; Herst et al., 2001). In extreme cases head orienting can proceed in one direction even if the ensuing gaze shift goes in the other (Corneil and Munoz, 1999). Recent results demonstrate that low levels of stimulation current in the dSC can drive neck EMG and/or head movements without gaze shifts (Pélisson et al., 2001; Corneil et al., 2002a, 2002b), strengthening the notion that dSC neurons can engage head movements at low levels of activity and eye-head gaze shifts at high levels of activity. The results presented in this paper constitute the strongest evidence that naturally occurring patterns of dSC activity can selectively engage the low-threshold drive to the head.

A number of other observations are consistent with our contention that visual target presentation recruits a low-threshold tectorecticulospinal pathway independent of OPN inhibition. The inverse relationship between neck EMG magnitude and the ensuing reaction time for ipsilateral saccades (Figure 4) resembles a similar relationship between the magnitude of dSC activity in response to visual targets and the ensuing reaction time for ipsilateral saccades (Dorris et al., 2002), although the relationship observed here is weaker. Such an inverse relationship was not observed for saccades in the opposite direction, since only the contralateral dSC emits a burst in response to visual target presentation. Further, some OPNs display an increase in activity time-locked to visual target presentation (Everling et al., 1998), presumably increasing the inhibition of the saccadic burst generator without affecting the excitability of head premotor elements. Finally, feline reticulospinal neurons in areas receiving tectal projections and identified tectoreticulospinal neurons also display bursts of activity time-locked to visual target presentation (Grantyn, 1989; Guitton and Munoz, 1991; Isa and Naito, 1995).

How then could the CNS use the dual pathways emanating from the dSC to generate the highly labile patterns of eye-head coordination during gaze shifts? We suggest that the selective gating of an orienting command issued by the dSC reflects a neural solution that preferentially delivers an orienting command to the head while the oculomotor system is engaged in deciding to commit to a gaze shift. While the phasic component of neck EMG following visual target presentation is undoubtedly too short to overcome the head's inertia, the ensuing tonic levels of lateralized neck EMG can drive head movements prior to gaze shifts (Figure 5). During such head movements, the VOR keeps gaze stable. While the gain of the VOR is suppressed during eye-head gaze shifts (Roy and Cullen, 1998), it is important to stress that the selective gating we have described in this paper occurs on a fundamentally different timescale, occasionally leading gaze shift onset by upwards of 150 ms. Persistent, lateralized patterns of neck EMG also have important kinetic consequences, preventing lengthening contractions of neck muscles antagonistic to the head turn and possibly priming premotor and motoneuronal pools to augment the overall force output of the head plant during ensuing gaze shifts.

Importantly, in addition to high-frequency bursts of activity that accompany sensory or motor events, dSC neurons can also display persistent levels of low-frequency activity in advance of target presentation when target location is predictable (Glimcher and Sparks, 1992; Basso and Wurtz, 1997; Dorris and Munoz, 1998). Increasing target predictability also leads to head motion prior to gaze shift onset (Bizzi et al., 1972; Zangemeister and Stark, 1982), supporting our hypothesis that low-frequency activity within the dSC can selectively engage the low-frequency tectoreticulospinal pathway to move the head independent of gaze shifts.

Our data do not address the converse question of how large amplitude gaze shifts are generated without any contribution of the head, although three alternatives are readily apparent. On one hand, the absence of head motion could occur simply because neck muscles are not recruited. Alternatively, the absence of head motion may not infer the absence of lateralized neck muscle recruitment (Corneil et al., 2002b) but may result if lateralized recruitment is dampened enough so that it cannot overcome the head's inertia. Finally, the absence of head motion may result from cocontraction of both agonist and antagonistic muscles. Recording neck EMG during large gaze shifts with varying head contributions will discriminate these alternatives and provide insights into the premotor circuits controlling neck muscle recruitment.

In conclusion, our results suggest that selective gating of a descending orienting command issued by the dSC is a fundamental mechanism used in the neural control of eye-head gaze shifts. The characteristic flexibility of

eye-head gaze shifts may be implemented in part by well-understood oculomotor circuits via the integration of such selective gating with cognitive signals that influence low-frequency activity within the dSC. While at first we were quite surprised that the brain permits an orienting command to access the head well in advance of gaze shift onset, such a strategy in retrospect appears quite efficient and may be a conserved strategy for other types of movements. For example, recent results examining eye-hand coordination describe the activation of lateralized limb EMG 20–80 ms in advance of saccade onset (Gribble et al., 2002). Although a proximal-to-distal sequence of muscle recruitment is observed in many postural and reaching tasks (Jeannerod, 1988), the strategy to selectively deliver a movement command to a more inertial segment may be pertinent particularly for multisegmental motion that includes an ocular component, given both the biomechanics of eye motion and the behavioral consequences of spurious or inappropriate eye movements.

## Experimental Procedures

### Neck Muscle Electromyography

All experimental procedures were approved by the Queen's University Animal Care Committee in compliance with the guidelines of the Canadian Council on Animal Care. In two monkeys, bipolar EMG electrodes were implanted chronically in up to 12 neck muscles, and electromyography (EMG) signals and eye, head, and gaze position signals were recorded at 500 Hz from monkeys who were either permitted to make head movements or not. The monkeys sat in a customized primate chair that restricted torso rotation to approximately $\pm 10°$. These procedures are described in more detail in our previous work (Corneil et al., 2001, 2002a, 2002b). Conditioning of the EMG signals attenuated peak-to-peak raw voltages by a factor of about 10. Neck EMG activity is reported for three muscles known to be involved in horizontal head turns (OCI, RCP maj, and SP cap; Figure 1B; Corneil et al., 2001). Other neck muscles involved in vertical head motion (biventer cervicis and complexus) or vigorous head turns (sternocleidomastoid) (Corneil et al., 2001) did not display target-aligned patterns of EMG activity.

### Gap Task

Monkeys generated saccades or eye-head gaze shifts in a gap task in an otherwise completely dark room (Figure 1A). Visual stimuli were bright red LEDs (4.7 cd/m$^2$) or back-projected red laser spots (8.4 cd/m$^2$). The monkeys first looked at a central fixation point (FP) for an interval chosen randomly among 800, 975, 1150, 1325, or 1500 ms. The FP then disappeared, and the monkeys were required to keep looking at the location of the extinguished FP for 200 ms, after which a target was randomly presented at one of two diametrically opposite peripheral positions. The monkeys had to look to the target within 500 ms, and a liquid reward was given if they maintained their eye position at the target for a further 500 ms. Target eccentricity ranged between 5° and 35° when the head was restrained and between 15° and 90° when the head was unrestrained. The radial angle of the target was never more than 45° from the horizontal meridian. The onset of saccadic eye movements or gaze shifts was determined by a velocity threshold of 50°/s and the onset of head movements by a velocity threshold of 10°/s. Velocities were determined from the position traces using a low-pass finite impulse response filter (Usui and Amidror, 1982) with the $-3$ dB cutoff set at 45.9 Hz.

### Receiver-Operating Characteristic Analysis

Receiver-operating characteristic (ROC) analysis was used to determine when after target presentation neck EMG discriminated target laterality, modifying a method previously used to analyze neural data (Bradley et al., 1987). Briefly, EMG waveforms from individual trials were segregated by the side of the target presentation relative to the side of recording and then smoothed with a 5-point (10 ms) running average. For every sample (2 ms) between 300 ms before and 300 ms after target presentation, we calculated a ROC curve using these smoothed EMG waveforms for the ipsilaterally or contralaterally presented target and then integrated the area under the ROC curve. This area metric represents the probability that an ideal observer could discern the side of target presentation based on the differential distribution of neck EMG activity at that point in time. A value of 0.5 indicates that an ideal operator would perform at chance, whereas a value of 1.0 indicates that the operator would perform perfectly. We defined the discrimination time (the time at which the EMG activity informed reliably about target location) as the time after target presentation when the ROC area surpassed 0.75 and remained above this level for at least 5 of the next 8 samples (i.e., 10 of the next 16 ms). Our results were not affected by selecting different threshold criteria or smoothing window widths, as the ROC area typically attained stable values near 1.0 whenever target-aligned neck EMG bursts were observed.

### Identification of Express Saccades

In previous publications out of our laboratory, the upper bound of express saccade reaction times for monkeys was determined to be 120 ms (Dorris et al., 1997; Paré and Munoz, 1996). This upper bound was determined using dim experimental stimuli, and we have since found the response latencies of visual-related responses within the dSC to be highly dependent on stimulus intensity (A.H. Bell and D.P.M., unpublished observations), consistent with previous reports (McPeek and Schiller, 1994). Accordingly, we examined the distributions of saccadic reaction times generated by the two monkeys used in this study, and based on the bimodality of these distributions, we adopted an upper bound for express saccades of 100 ms. The lower bound of express saccades was determined to be 70 ms, as saccades with reaction times less than 70 ms were equally as likely to be generated toward or away from the target, and were hence classified as anticipatory. Trials with anticipatory saccades were rare (1.3% of all saccades) and were not subjected to further analyses.

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