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Neuromuscular consequences of reflexive covert orienting

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Visual stimulus presentation activates the oculomotor network without requiring a gaze shift. Here, we demonstrate that primate neck muscles are recruited during such reflexive covert orienting in a manner that parallels activity recorded from the superior colliculus (SC). Our results indicate the presence of a brainstem circuit whereby reflexive covert orienting is prevented from shifting gaze, but recruits neck muscles, predicting that similarities between SC and neck muscle activity should extend to other cognitive processes that are known to influence SC activity.

Presentation of a visual stimulus, even in the absence of gaze shifts, induces widespread transient visual responses in the frontal-parietal-collicular network[1]. These responses are carried by neurons in the intermediate and deep layers of the SC that project to the brainstem premotor circuits that control eye and head movements[2,3]. Brainstem omnipause neurons (OPNs) inhibit the saccadic-burst generator circuit during this transient visual response, preventing the execution of saccadic gaze shifts[4,5]. It is widely believed that OPNs do not gate head movements in a similar manner (**Fig. 1a**). Consistent with this, transient visual responses have been observed on neck muscles following visual stimulus presentation[6]. Correlates of reflexive covert orienting have been identified in the transient visual responses in the SC[7], leading to the hypothesis that reflexive covert orienting signals may also be present on neck muscles. If true, this would call into question the very nature of reflexive covert orienting by demonstrating recruitment in the motor periphery.

To test this, we recorded electromyographic (EMG) activity from neck muscles in monkeys carrying out a cueing task (**Supplementary Methods** and **Supplementary Fig. 1** online; the University of Western Ontario Animal Use Subcommittee approved all experiments). Subjects looked to a target regardless of the presence of a visual cue that may be flashed at the 'same' or 'opposite' location before target presentation (subjects were trained not to look at the cue, **Supplementary Figs. 1** and **2** online). If the cue and target were presented at the same location in relatively quick succession (for example, 50-ms cue-target onset asynchrony, CTOA), monkeys looked to the target sooner than they did when the cue was presented at the opposite location. This effect has been termed attentional capture[8]. In contrast, if the CTOA is increased to 200 ms, saccades to the target are delayed compared with when the cue is presented at the opposite location. This has been termed inhibition of return (IOR)[9,10].

We studied the patterns of neck EMG during the cueing paradigm (**Fig. 1**; both monkeys in this study produced attentional capture and IOR, **Fig. 1e**,**g**). We recorded EMG activity from obliquus capitis inferior (OCI), a small suboccipital muscle that turns the head[6] (we observed similar results on other turning muscles; **Supplementary Fig. 3** online), and focus first on the consequences of cue and target presentation on OCI activity in a head-restrained monkey (**Fig. 1b–d**). We noted several important changes in neck EMG activity.

First, cue presentation (the time between two vertical dashed lines in **Fig. 1b–d**) led to a short-latency ($\sim$50–60 ms) phasic increase in the activity of the OCI muscle ipsilateral to the cue and to an accompanying short-latency decrease in the activity of the OCI contralateral to the cue (first phase following cue presentation in **Fig. 1b–d**). These visual responses increased in magnitude for larger stimulus eccentricities (**Supplementary Fig. 4** online).

Second, following the initial cue-related response, EMG activity increased to a more sustained level (second phase following cue presentation in **Fig. 1c**,**d**), which commenced about 100 ms following cue presentation and persisted for a subsequent 200 ms. This post-cue plateau was increased on the OCI muscle ipsilateral to the cue and decreased on the OCI muscle contralateral to the cue. These post-cue plateaus also increased in magnitude for larger stimulus eccentricities (**Supplementary Fig. 4**).

Third, target presentation produced transient visual responses on neck muscles (vertical gray bars in **Fig. 1b–d**; the subsequent increase in EMG activity relates to saccade execution). Both cue location and CTOA had a strong influence on the magnitude of this target-related transient response. At the 200-ms CTOA, where IOR occurred, target onset that followed cue presentation at the same location elicited virtually no identifiable transient visual response (**Fig. 1c**, vertical gray bar). The absence of a transient visual response is particularly stunning given the elevated activity on the muscle before target onset. In sharp contrast, EMG activity on the ipsilateral OCI muscle increased robustly on opposite and control trials. We observed these transient visual responses in both same and opposite trials at the 600-ms CTOA (**Fig. 1d**), demonstrating that the diminution of a transient visual response in same trials observed at the 200-ms CTOA was only temporary. At the 50-ms CTOA (**Fig. 1b**), the transient visual responses were exaggerated on same trials compared with both control and opposite trials.

We quantified the transient visual EMG responses to target presentation with a metric *ΔEMG* (inset of **Fig. 1f** and **Supplementary Methods**). The *ΔEMG* curves mirrored the changes in saccadic reaction

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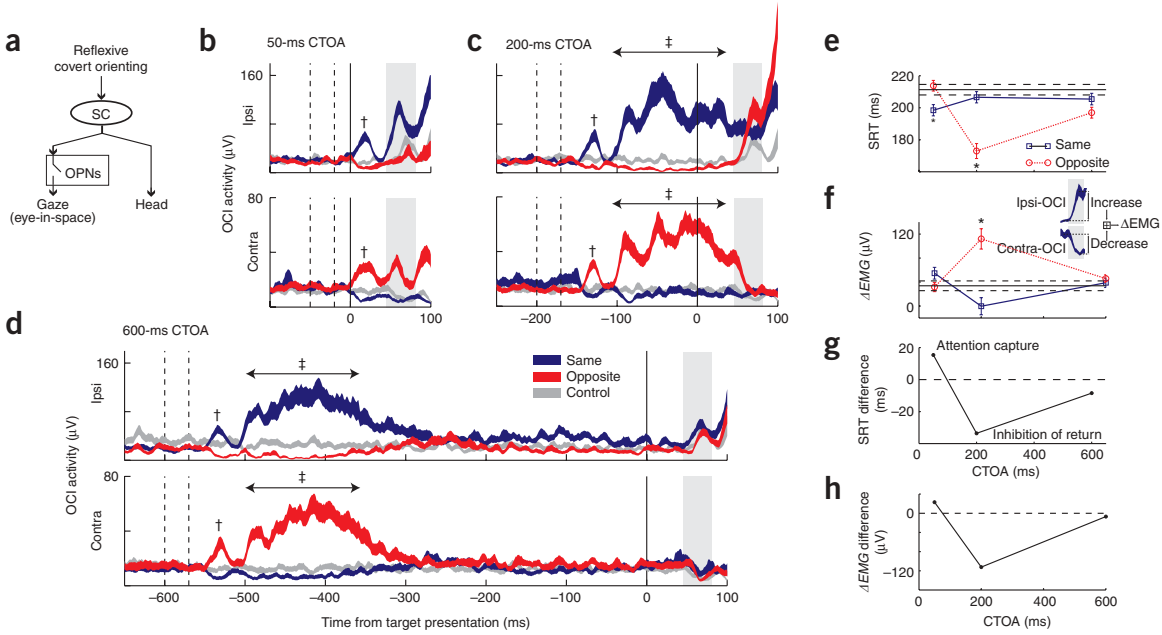

Figure 1 Neck EMG recorded during the cueing task, with the head restrained. (**a**) Simplified depiction of the brainstem circuit downstream of the SC. (**b**–**d**) EMG activity recorded from contralateral and ipsilateral OCI muscles from one head-restrained monkey, aligned to target presentation 27° right (contours subtend s.e.m.). Data segregated into the 50-ms (**b**), 200-ms (**c**) and 600-ms (**d**) CTOA conditions. Cue duration spans the interval between dashed vertical lines. The vertical gray bars denote the transient EMG responses related to target presentation. † in **b**–**d** denotes the phasic EMG response following cue presentation; ‡ in **c**,**d** denotes the more sustained level of EMG activity beginning 100 ms after cue presentation. (**e**) Mean (± s.e.m.) head-restrained SRTs as a function of CTOA and cue location (same session). Mean SRTs for control trials are shown with the solid horizontal line (dashed lines show s.e.m.). A two-way ANOVA of SRT across CTOA and cue location demonstrated significant ($P < 0.05$) effects of CTOA and cue location, and an interaction between CTOA and cue location. (**f**) *ΔEMG* (the sum of the ipsilateral muscle increase plus the contralateral muscle decrease, see inset) as a function of cue location and CTOA for the same and opposite conditions. A two-way ANOVA of *ΔEMG* across CTOA and cue location demonstrated significant effects of cue location and an interaction between CTOA and cue location. Asterisks in **e** and **f** denote SRT *ΔEMG* observations that were significantly different at the given CTOA (Bonferroni-corrected *post hoc t*-test, $P < 0.05$). (**g**) SRT difference ($SRT_{opposite} - SRT_{same}$) as a function of CTOA. (**h**) ΔEMG difference across CTOA, calculated as $\Delta EMG_{same} - \Delta EMG_{opposite}$ for comparison with SRT data illustrated in **g** (larger ΔEMG values accompany shorter SRTs).

time (SRT) as a function of CTOA and cue location (contrast **Fig. 1e**,**f**). At the 200-ms CTOA, we observed shorter SRTs and greater *ΔEMG* values in the opposite compared with the same condition. We computed a difference between the *ΔEMG* values across cueing conditions, calculating the same-opposite values at each CTOA. The overall shape of the *ΔEMG* difference curve (**Fig. 1h**) was markedly similar to that of the SRT difference curve (**Fig. 1g**), with the lowest values being observed at the 200-ms CTOA that was associated with maximal IOR. We repeated this experiment at eccentricities ranging between 10–35° in two monkeys and observed that the same qualitative patterns of neck EMG activity scaled across all eccentricities (**Supplementary Figs. 5** and **6** online for data at 10° and for data across all eccentricities, respectively).

Because the transient visual EMG responses occurred before the saccade, we sought to determine whether the *ΔEMG* metric would be a reliable predictor of the ensuing SRT on a given trial. In almost all conditions, we observed significant negative correlations between *ΔEMG* and SRT (**Supplementary Fig. 7** online). These relationships resemble those reported previously between neuronal activity in the SC and SRT in a cueing task[7]. Thus, the tectoreticulospinal system is a likely candidate for relaying the visual response onto neck muscles[2,3].

We observed the same patterns of SRT and neck EMG with the head unrestrained (**Fig. 2** and **Supplementary Fig. 8** online). Here we focused on the 600-ms CTOA (**Fig. 2a**), as this provides the largest temporal separation between cue and target presentation. We observed a very small movement of the head toward the cue (~1–2° in amplitude, reaching a peak velocity of ~10° s^{-1}), demonstrating that the neck EMG recruitment following cue presentation caused small head motion. However, gaze remained stable because a compensatory vestibulo-ocular reflex (VOR) drove the eyes in the opposite direction (**Fig. 2a** and **Supplementary Fig. 8**).

The observations presented here show that reflexive covert orienting imparts neuromuscular consequences at the head. The cueing procedure, which has been a mainstay in attentional research for nearly 30 years, clearly induced neuromuscular effects that have previously gone unnoticed as a result of head restraint and the preference for smaller target eccentricities. In the oculomotor system, OPNs inhibited reflexive covert orienting signals from influencing motion of the visual axis (**Fig. 1a**), ensuring a stable retinal image[5]. Such gating may represent a solution that is unique to the movements of the visual axis. Other effectors, such as the head, trunk and limbs, are characterized by more complex biomechanics (for example, larger inertia) and are compensated for by reflexes (for example, the VOR), and hence do not require the discrete gating imposed by OPNs.

The pattern of neck EMG activity reported here is remarkably similar to the profiles of SC activity recorded during the exact same task[7]. Although we have shown here that neck muscle EMG correlates with reflexive covert orienting, we speculate that the mechanism that we propose (**Fig. 1a**) could generalize to nonreflexive forms of covert

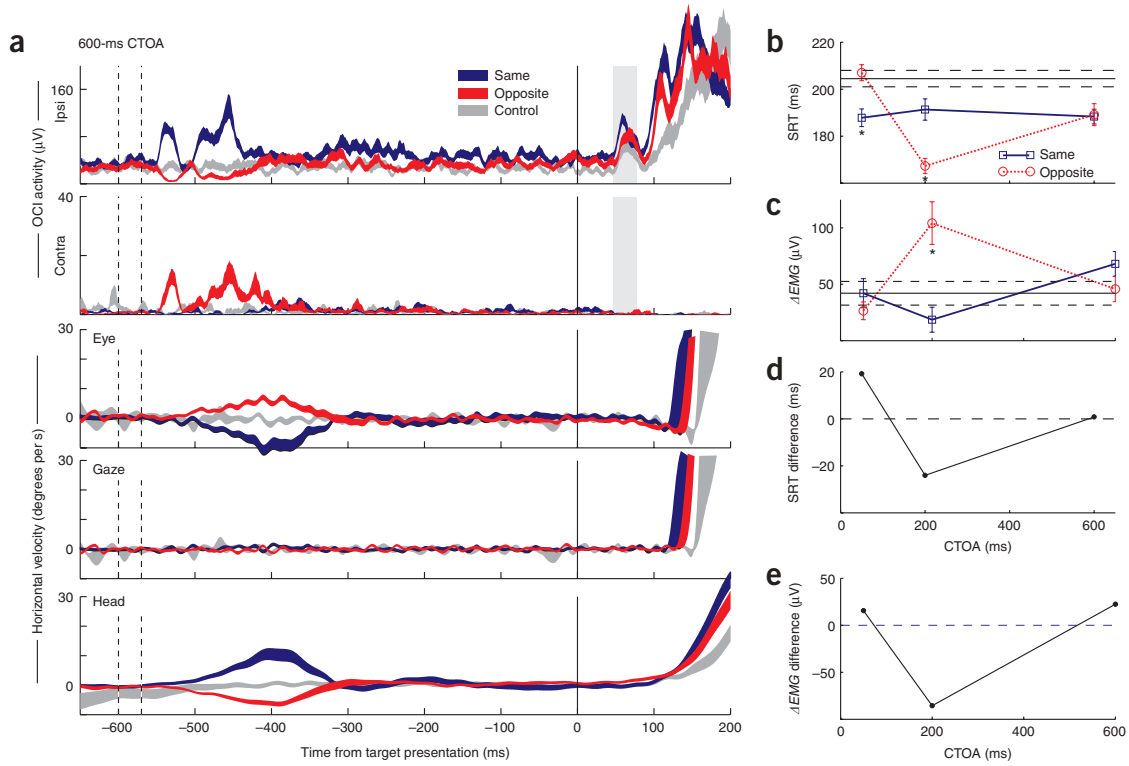

Figure 2 Neck EMG recorded during the cueing task, with the head unrestrained. (**a**) EMG and instantaneous-velocity data recorded head-unrestrained from ipsilateral and contralateral OCI for a target located 27° right for the 600-ms CTOA (**Supplementary Fig. 8** for the 50- and 200-ms CTOA conditions). The data is presented as in **Figure 1d**, with additional plots of mean horizontal eye, gaze and head velocity (contours subtend s.e.m.). Upward deflections denote rightward movements. Cue presentation led to a slight head movement toward the cue that was compensated for by a VOR eye movement in the opposite direction, ensuring gaze stability. (**b**–**e**) Plots of SRT and *ΔEMG* for the same and opposite conditions and the SRT and *ΔEMG* difference as a function of CTOA. The data is presented as in **Figure 1e**–**h**. For SRT, a two-way ANOVA demonstrated a significant effect of CTOA and a interaction between CTOA and cue location ($P < 0.05$). For *ΔEMG*, a two-way ANOVA demonstrated a significant interaction between CTOA and cue location ($P < 0.05$).

orienting that alter SC activity[7,11]. In support of this, correlates of other high-level cognitive processes, such as motor preparation and motivation-related to reward, can be recorded from the SC[12,13] and neck muscles[14,15].

Note: Supplementary information is available on the Nature Neuroscience website.

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AUTHOR CONTRIBUTIONS

B.D.C. and D.P.M. designed the experiments and co-wrote the paper. B.D.C., B.B.C. and T.A. collected and analyzed the experimental data. B.D.C., T.A. and S.L.C. implanted the neck muscle electrodes. B.B.C., T.A. and S.L.C. provided editorial comments throughout the writing process.

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