A Within Trial Measure of the Stop Signal Reaction Time in a Head-Unrestrained Oculomotor Countermanding Task

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Goonetilleke SC, Doherty TJ, Corneil BD. A within trial measure of the stop signal reaction time in a head-unrestrained oculomotor countermanding task. J Neurophysiol 104: 3677–3690, 2010. First published October 20, 2010; doi:10.1152/jn.00495.2010. The countermanding paradigm provides a framework for studying inhibitory control. Although no observable behavior is elicited on successfully cancelled stop signal trials, the duration of the stop process, the stop signal reaction time (SSRT), can be estimated using the assumptions of the race model (Logan and Cowan 1984). Estimation of the SSRT provides a metric for movement cancellation that has been used, for example, to quantify control deficits following brain damage (Dimitrov et al. 2003) and the effects of pharmacologicals on children with attention deficit hyperactivity disorder (Tannock et al. 1995; see Verbruggen and Logan 2008 for review). SSRT estimates are also useful neurophysiologically in assessing whether a given area is involved in the immediate control of action or not (Hanes et al. 1998; Paré and Hanes 2003; Stuphorn et al. 2000).

A number of methodologies have been proposed to estimate the SSRT. Two of the most common, the mean and the integration methods, derive SSRT estimates via calculations involving the reaction time (RT) distributions on control trials without a stop signal and the inhibition function describing response probability as a function of the delay of the stop signal relative to the target (Logan 1994). SSRTs can also be estimated statistically using maximum likelihood methods (Kornyl et al. 2003) or the density functions of the RTs from control and noncancelled stop signal trials and the inhibition function (Colonius 1990; see Band et al. 2003 for detailed description of these methods). Walton and Gandhi (2006) recently developed a novel psychophysical means of SSRT estimation by evoking blinks via air puffs. Delivering air puffs to the eye temporarily disengages the inhibition of the oculomotor burst generator. By timing the delivery of air puffs to the peri-saccade interval, Walton and Gandhi (2006) inferred the timing of movement cancellation via the presence or absence of saccades accompanying blinks. Although statistically or behaviorally based SSRT estimates are fairly consistent, attesting to the general robustness of the race model, such estimates require the integration of results over many trials. To our knowledge, it has not been possible to empirically measure processes on an individual cancelled stop signal trial; this makes intuitive sense as successful cancellation implies the absence of movement. Such a measure, if it could be obtained, would be valuable in a number of settings. First, it could serve as a regressor in functional imaging studies to help differentiate whether there are multiple types of movement cancellation (see Verbruggen and Logan 2008 for review). Second, it would be valuable in clinical settings, where an increased variability in SSRT is one of the indicators of dysfunction (Schachar and Logan 1990). Third, it would be
studies in studying inhibition as activity from a candidate neuron would necessarily have to precede the within-trial measure by a consistent interval to be involved in movement cancellation.

The oculomotor system provides one of the best systems for studying movement cancellation as eye movements are easily measured and readily paired with neurophysiological techniques. Experiments are typically run head-restrained with subjects either generating or canceling saccades. We have recently investigated how humans countermand eye-head gaze shifts, and in addition to either successfully canceling a gaze shift or not, subjects also produced a third movement sequence where the head moved toward the target even though gaze remained stable due to a compensatory vestibulo-ocular reflex (Cornel and Elsley 2005). On such trials, the head initially received an orienting command that was subsequently withdrawn or arrested in mid-flight. Here we record neck muscle activity in humans, focusing on the activity of splenius capitis (SPL), a large muscle known to be involved in head turning and active braking in monkeys and humans (Corneil et al. 2001; Zangemeister and Stark 1981). We show that these brief head movements are actively braked by selective recruitment of the antagonist SPL. Importantly, the timing of such recruitment correlated well with statistically based estimates of the gaze SSRT. Because gaze remained stable during such trials (i.e., the stop process "won" the race), we propose that the activation of the antagonistic neck muscle is driven by the oculomotor stop process. To our knowledge, this finding constitutes the first empirical, within-trial measure of the oculomotor stop process.

Some results have been reported previously in abstract form (Goonetilleke et al. 2008).

**METHODS**

Eight subjects (3 female, mean age = 27 yr) participated in the experiment. None reported any neurological deficits. All procedures were approved by the University Research Ethics Board for Health Science Research at the University of Western Ontario and were in accordance with the Declaration of Helsinki. Subjects gave informed consent and were aware that they could terminate testing at any time. Two subjects (S1 and S8) were the authors and hence were knowledgeable about the specific goals of the experiment. Their results did not differ from the remaining subjects who were naïve to the experimental goals.

**Countermanding task**

Subjects performed an oculomotor variant of the countermanding task with the head unrestrained as described in greater detail elsewhere (Cornel and Elsley 2005). Briefly, this task required subjects to look to peripheral visual targets on most trials (termed control trials, constituting 70% of all trials) but to try to maintain fixation on the central fixation point (FP) on stop signal trials (30% of all trials) on presentation of a stop signal. All stimuli in this experiment consisted of light-emitting diodes (LEDs) positioned 1.2 m in front of the subject at a height of 1.2 m to lie on the horizontal meridian from the subject’s perspective. One centrally positioned LED served as the central FP, and two peripheral LEDs placed at 60° either to the left or right served as the targets. The visual angle of the peripheral targets was selected to be well outside of the oculomotor range for humans (Guitton and Volle 1987; Stahl 1999), hence target-directed gaze shifts required a contribution of the head.

Both control and stop signal trials began with illumination of the central FP for 1–1.5 s. This FP was then extinguished for 200 ms prior to illumination of either the right- or leftward target. The inclusion of this 200 ms gap expedites both saccadic RTs (Munoz et al. 2000) and SSRTs (Stevenson et al. 2009). Subjects looked to the peripheral target on control trials. On stop signal trials, the central FP was re-illuminated; this served as the stop signal that instructed subjects to try not to look to the peripheral target. We varied the stop signal delay (SSD; Fig. 1B) between the illumination of the target and the stop signal among six different intervals spanning a 200 ms range. A fixed set of SSDs were customized for each subject so that the probability of movement spanned the minimum and maximum of the inhibition functions. All subjects completed three blocks of 200 trials (600 trials in total). Target direction (left or right), trial type (control or stop signal), and SSD were pseudorandomized by a customized LabView program (National Instruments) to ensure the appropriate proportion of the various trial conditions.

Each subject performed a series of practice trials prior to data collection to familiarize themselves to the task. Subjects were instructed to look as quickly and as accurately as possible to the central FP on presentation of the stop signal.

**Data collection and analysis**

Six dimensional head movements were recorded at a rate of 4 kHz using a passive infrared measurement system, the MotionMonitor tracker (Innovative Sports Training, Chicago, IL). Subjects wore a custom head band on which three reference markers were mounted on a Perspex triangle. The space in which the subjects were located was

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**FIG. 1.** A: depiction of a simple race model that proposes that there is an accumulation of two processes to a threshold, a go and stop process which determine whether a response is generated or suppressed, respectively. Depending on the stop signal delay (SSD) and the rate of rise of these 2 processes, determine whether a response is generated or suppressed, respectively. De-
first calibrated, and then the reference markers were assigned to the center of mass of the head using a centroid system. The location of the markers in space was monitored by four digital real-time cameras (Motion Analysis, Santa Rosa, CA). Horizontal eye movements were measured using bitemporal DC electrooculography (EOG) with electrodes placed at the outer canthus of the eyes (Carl 1993) and were filtered and amplified with a P122 AC/DC preamplifier (Grass Instruments, Warwick, RI). The horizontal eye position signal was then low-pass filtered (100 Hz), amplified, and digitized at a rate of 4 kHz onto the MotionMonitor.

Electromyographic (EMG) activity of SPL at the level of the C2/C3 vertebrae was detected bilaterally using intramuscular fine-wire “needle” electrodes (30 mm, 27 gauge, Motion Lab Systems, Baton Rouge, LA; Fig. 2). SPL is a dorsal neck muscle which plays a major role in ipsilateral horizontal head rotations as well as a subsidiary role in cervical extension (Mayoux-Benhamou et al. 1997). Intramuscular recordings were utilized as previous research has shown that surface recordings of SPL are susceptible to cross-talk from adjacent dorsal neck muscles (Mayoux-Benhamou et al. 1995). EMG needle electrodes were inserted under the guidance of real-time ultrasound imaging (Sonoace Pico, Universal Medical Systems, Bedford Hills, NY), following an approach described elsewhere (Blouin et al. 2007). During insertion, the fasciae surrounding SPL was clearly visible, and the electrode was always placed below the surface fascial plane. The insertion needle was removed after visual confirmation of electrode location, and the electrodes remained within SPL. The placement of the electrode in SPL was further confirmed by observing strong EMG activity in response to slight ipsilateral head rotation and the absence of activity in response to shoulder shrugs or contralateral head rotation (André-Deshays et al. 1988). A disposable self-adhesive Ag/AgCl electrode (Blue Sensor, Run Technologies, Laguna Hills, CA) was adhered to the subject’s left forearm and was utilized as the ground electrode. EMG data were recorded with a Myopac Jr (Run Technologies; low-pass filter modified to 2 kHz). The EMG data were amplified and sampled at 4 kHz and digitized with a 16 bit A-D converter by the MotionMonitor system.

All aspects of the experiment were controlled at a rate of 1 kHz by a customized Labview program that executed in real-time on a PXI box (National Instruments). Off-line analyses were performed using customized Matlab (The Mathworks) programs. Off-line analyses consisted first of downsampling all positional data from 4 to 1 kHz.

Horizontal gaze position was constructed by adding the EOG signal to the horizontal head position signals. EMG data were full-wave rectified and then bin-integrated into 1-ms bins.

Movement onsets and offsets were identified by a computer algorithm that detected crossings of velocity thresholds (50°/s for gaze and 10°/s for head). These marks were used as guides for the placement of interactive marks by an analyst within a customized graphical user interface (GUI) written in Matlab. Movement amplitudes and peak velocities were extracted for movements bounded by these marks. The on- and offset of EMG activity was identified by another algorithm based on when the activity exceeded baseline (average EMG activity in the 500 ms preceding target onset) by 3 SD for 20 data points over a period of 30 data points. These marks could also be adjusted by the analyst, and the peak magnitude of EMG activity between these bounds was also extracted. This interface also permitted the exclusion of trials if classified as anticipatory or too slow due to lack subject alertness (RTs <80 or >800 ms, respectively) (Coenil and Munoz 1996), if the subject looked in the wrong direction opposite to the target, or if there were excessive levels of background EMG activity. Less than 5% of trials had to be excluded using these criteria.

R E S U L T S

As reported previously (Coenil and Elsley 2005), subjects produced three types of movement sequences on stop signal trials (see Fig. 3, B–D, far left column, for representative single-trial examples from subject S2). In one movement sequence, subjects completely cancelled any motion of either the gaze axis or the head toward the target (a cancelled eye-head gaze shift; e.g., Fig. 3B). In another movement sequence, subjects were unable to suppress an eye-head gaze shift toward the target (a noncancelled eye-head gaze shift; e.g., Fig. 3C). In a third movement sequence, subjects successfully cancelled a gaze shift toward the target but generated a small and brief head movement toward the target; a VOR movement of the eye-in-head ensured gaze stability during the head-only error (e.g., Fig. 3D). Importantly, we never observed a complementary eye-only error sequence that consisted of a saccadic eye movement in the absence of head motion.

The presence of head-only movements is reflected in inhibition functions for each subject (Fig. 4). We derived separate inhibition functions for the head (with or without gaze shifts) and gaze as they showed different propensities to move. The inhibition functions for all subjects are consistent with the race model as the probability of making an erroneous movement increased at longer SSDs. Also, consistent with the findings of Coenil and Elsley (2005), head movement inhibition functions are shifted to the left when compared with gaze movement inhibition functions; this emphasizes there was a greater likelihood of a head movement than a gaze shift at a given SSD. The difference between the two inhibition functions indicates the probability of a head-only error at any given SSD. Consistent with the findings of Coenil and Elsley (2005), head-only errors mainly occurred at intermediate SSDs.

These movement sequences are consistent with a standard race model with a slight modification that the head can begin moving when the go process exceeds a lower threshold (Coenil and Elsley 2005) (see the schematics in Fig. 5 for the proposed activation of the go and stop processes for each of the 3 main movement sequences observed during stop trials). To analyze the average SSD associated with each movement sequence, we rank ordered the SSDs for each subject with a
rank of one indicating the shortest SSD. Across the different movement sequences, the average rank SSD was longest for noncancelled movements, intermediate for head-only movements, and shortest for cancelled movements. These observations are consistent with the framework of the race model where longer SSDs result in a greater probability of movement as subjects have more time to initiate a movement before they are instructed to try and cancel the movement (Fig. 5).

We further subdivided noncancelled gaze shifts into those where the gaze axis went all the way to completion (termed completed) and those where the gaze axis stopped short of the target (termed truncated). This distinction is important because our subsequent analyses focus on the presence or absence of activity in agonist and antagonist neck muscles and on the timing of such activity. We do not describe antagonist neck muscle activity during truncated noncancelled gaze shifts in the analysis below because, to put it in race model terms, the GO process initially wins on such trials before being superseded in mid-flight by the STOP process. Initiation of a gaze shift is also associated with a series of brain stem events that are not present on head-only trials, including the temporary attenuation of the VOR and the engagement of the saccadic burst generator (see Scudder et al. 2002 for review). Our paradigm requested that subjects either generate or attempt to suppress large gaze shifts that, if completed, are >100 ms long. This prolonged duration provides an interval in which subjects can cancel an on-going gaze shift in mid-flight. The presence of truncated gaze shifts suggest that gaze shifts are not completely ballistic (e.g., Corneil et al. 1999; Ramakrishnan et al. 2010) and that the processing of the stop signal continues after the initiation of the gaze shift. However, because we are explicitly interested in neck muscle recruitment as a function of the control of the gaze axis, we have derived our measures of antagonist muscle recruitment exclusively from trials with head-only errors to avoid confounds associated with gaze shift initiation.

![Graphical representation of the movement sequences and muscle activity](image_url)
The frequency of these movement sequences is shown in Table 1 on a per-subject basis. Overall, gaze shifts were cancelled on 50% of all STOP signal trials, with head-only errors occurring on 25% of all STOP signal trials (mean ± SD: 25 ± 7%, range: 10–34%).

Neck muscle activity during control and stop signal trials

We now turn to the patterns of SPL recruitment accompanying the various movement sequences; recall that SPL is an ipsilateral head turner, meaning that right-SPL or left-SPL acts as an agonist or antagonist for rightward head turns, respectively. The activity of both muscles is shown in Fig. 3 for a representative subject (S2) following rightward target presentation for both control and stop signal trials. The patterns of neck muscle recruitment described in the following text were observed in all subjects.

On control trials when the subject made a successful eye-head gaze shift to the target, we observed a strong burst of EMG activity on the agonist right-SPL muscle ~50 ms prior to head motion (Fig. 3A). Importantly, there was very little activity of the antagonist left-SPL muscle during the rightward head turn. The absence of such antagonist muscle activity suggests that there is no active braking of the head from this muscle; instead, the end of head motion was associated with a decrease in the activity of the agonist right-SPL muscle with residual activity following the head movement maintaining the head’s eccentric position. The absence of antagonist muscle recruitment during regularly paced eye-head gaze shifts is consistent with reports of SPL activity in humans (Mayoux-Benhamou et al. 1997; Takebe et al. 1974) and from reports in monkeys that have recorded from SPL and other neck muscles during horizontal head turns (Corneil et al. 2001; Lestienne et al. 1995). Active braking by antagonist neck muscles (including SPL) is observed during rapid head turns in both humans and monkeys (Corneil et al. 2001; Lestienne et al. 1995; Zangemeister and Stark 1981).

The pattern of neck muscle activity observed on stop signal trials depended on the generated movement sequence. We observed a complete absence of neck muscle activity on both agonist and antagonist muscles on cancelled stop signal trials where neither the gaze nor head moved toward target (Fig. 3B). In contrast, the profile of neck muscle activity observed on completed noncancelled stop signal trials resembled that observed on control trials: erroneous head movements were preceded by agonist muscle activation, and the antagonist muscle remained silent (Fig. 3C). On such trials, the recruitment of the antagonist muscle after the noncancelled eye-head gaze shift returned the head back to the central FP.
An erroneous movement was also observed on truncated non-cancelled trials to the target. Such antagonist muscle recruitment during an erroneous movement occurred very quickly by robust activation of the antagonist muscle, which occurred followed very quickly by robust activation of the antagonist muscle. However, such agonist activation was cancelled on any of the movement profiles observed on cancelled or completed or non-cancelled stop signal trials (Fig. 3D). Head-only errors, as expected, were preceded by activation of the agonist muscle. If antagonist neck muscle recruitment is serving as an active braking pulse, then its timing should be related to the onsets of the stop signal. In Fig. 7, we present the latency of antagonist muscle recruitment during an erroneous movement relative to the time of stop signal presentation for all head-only movements for all subjects (recall S2 was our representative subject). Within each subplot, trials are sorted by decreasing SSD (○, target onset; |, stop signal onset; □, the onset of antagonist muscle recruitment), and the histogram shows the antagonist muscle latencies relative to stop signal onset. The mean antagonist muscle latency was 180 ± 26 ms (range: 149–217 ms). The breadth and shape of the distributions of these varied substantially across subjects, being narrow in S1, S3, and S5, and broad within S4, S6, and S8. S2, and to a lesser degree S7, displayed a bimodal latency distribution; most observations fell within a first mode −150 ms, while fewer observations fell within a more broad mode centered ~300 ms.

Antagonist muscle activity as an active braking pulse

The selective recruitment of the antagonist neck muscle during erroneous head motion suggests that such activity may serve as an active braking pulse to arrest (or even reverse) head motion in mid-flight. If so, then the magnitude of the antagonist muscle activity should scale with the kinematics of the head-only movement as a larger braking pulse would be required to stop a larger or faster head movement. We therefore compared the peak of antagonist muscle activity during the head-only error (normalized to the average peak activity when the muscle served as an agonist on control trials in the other direction) to the amplitude of the head-only error (such normalization enables pooling data across bilateral muscles within a subject) for each head-only error trial. Seven of the eight subjects showed a significant positive correlation between the peak level of antagonist muscle activity and the amplitude of the head-only error (Fig. 6A). We also compared the peak antagonist muscle activity to the peak velocity of the head-only error, and again observed significant positive correlations in seven of eight subjects (Fig. 6B). These positive correlations are consistent with the notion that antagonist muscle recruitment serves as an active braking pulse, as larger and faster head-only errors required greater levels of antagonist muscle recruitment.

Onset of antagonist muscle recruitment is best associated with the time of stop signal presentation

We next investigated the timing of antagonist neck muscle recruitment. If antagonist neck muscle recruitment is serving as an active braking pulse, then its timing should be related to the onset of the stop signal. In Fig. 7, we present the latency of antagonist neck muscle activity relative to the time of stop signal presentation for all head-only movements for all subjects (recall S2 was our representative subject). Within each subplot, trials are sorted by decreasing SSD (○, target onset; |, stop signal onset; □, the onset of antagonist muscle recruitment), and the histogram shows the antagonist muscle latencies relative to stop signal onset. The mean antagonist muscle latency was 180 ± 26 ms (range: 149–217 ms). The breadth and shape of the distributions of these varied substantially across subjects, being narrow in S1, S3, and S5, and broad within S4, S6, and S8. S2, and to a lesser degree S7, displayed a bimodal latency distribution; most observations fell within a first mode ~150 ms, while fewer observations fell within a more broad mode centered ~300 ms.

TABLE 1. **Response sequence probabilities on stop trials for all subjects**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Number of stop trial</th>
<th>No Gaze Shift</th>
<th>Gaze Shift Non-cancelled</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Cancelled</td>
<td>Head Only</td>
</tr>
<tr>
<td>S1</td>
<td>155</td>
<td>33 (0.21)</td>
<td>37 (0.24)</td>
</tr>
<tr>
<td>S2</td>
<td>165</td>
<td>35 (0.21)</td>
<td>56 (0.34)</td>
</tr>
<tr>
<td>S3</td>
<td>171</td>
<td>61 (0.36)</td>
<td>35 (0.20)</td>
</tr>
<tr>
<td>S4</td>
<td>171</td>
<td>88 (0.51)</td>
<td>43 (0.25)</td>
</tr>
<tr>
<td>S5</td>
<td>175</td>
<td>61 (0.35)</td>
<td>39 (0.22)</td>
</tr>
<tr>
<td>S6</td>
<td>168</td>
<td>46 (0.27)</td>
<td>17 (0.10)</td>
</tr>
<tr>
<td>S7</td>
<td>177</td>
<td>83 (0.47)</td>
<td>31 (0.18)</td>
</tr>
<tr>
<td>S8</td>
<td>140</td>
<td>16 (0.11)</td>
<td>38 (0.27)</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>35 (0.23)</td>
<td>37 (0.25)</td>
</tr>
</tbody>
</table>

The number of observations for each movement sequence are followed by the probabilities in parentheses.
Examination of the timing of antagonist muscle recruitment and its relationship to the stop signal

We also examined whether the timing of antagonist muscle recruitment was more closely related to the onset of the target or the stop signal. We first compared the variance of the antagonist muscle latency aligned to the onset of either the target or the stop signal. For all eight subjects, the variance of the antagonist latency was less when this measure was aligned to the stop signal rather than the target onset ($t_{7} = 3.46, P = 0.011$).

We then examined whether the antagonist latency depended in any way on the SSD. To do this, we extracted the mean antagonist muscle latency on a per-subject, per-SSD basis (for this analysis, there had to be at least 2 head-only errors at a given SSD), and analyzed the results as a function of SSD (Fig. 8A). This analysis revealed no significant trend in the latency of the antagonist muscle recruitment across the different SSDs in six of eight subjects (1-way ANOVA for each subject; S2 and S3 showed a significant decrease in antagonist muscle latency with increasing SSD $F = 2.33$ and $3.12, P = 0.05$ and 0.02, respectively; Fig. 8A). These results are further consistent with the notion that antagonist muscle recruitment serves as an active braking signal generated in response to the stop signal, regardless of the timing of the stop signal relative to the target.

Although there was no statistically significant trend in the antagonist latency across SSDs in most subjects, the ranges of SSDs did differ in our sample. We therefore collapsed our data after rank-ordering the SSDs from shortest to longest. This analysis revealed a general trend of a decrease in the latency of the antagonist burst with increasing SSD (Fig. 8B), although this trend did not reach significance (1-way ANOVA). The correlation coefficients for each subject are shown in the figure, indicating that larger and faster head-only errors were associated with larger bursts of antagonist muscle activity.

**FIG. 6.** Comparison of the normalized antagonist EMG activity to the amplitude (A) and the peak velocity (B) of the head-only error on each head-only STOP signal trial. Most subjects show a significant positive correlation between the measures indicating that larger and faster head-only errors were associated with larger bursts of antagonist muscle activity. Correlation co-efficients for each subject are also shown (*) indicating a correlation which was statistically significant at the alpha level of 0.05.
ANOVA of antagonist burst across SSD; $P = 0.16$). This general trend is consistent with previous reports in the literature than shorter SSRTs tend to occur at shorter SSDs (Logan and Cowan 1984). To analyze this, we calculated the SSRTs at a given SSD via the integration method (see next section), and observed a similar trend for shorter SSRTs at longer SSDs. This trend also approached but did not reach significance (Fig. 8B; $P = 0.17$).

**Comparison of antagonist muscle latencies to SSRTs**

We now turn to the implications of our results to the race model. As mentioned in the Introduction, the assumptions inherent to the race model permit estimation of the duration of the stop process via calculation of SSRTs. The stop process is initiated on presentation of the stop signal and wins the race against the go process on successfully cancelled trials. We have provided evidence that the timing of antagonist muscle latency is most closely associated with the onset of the stop signal, and on head-only trials when a gaze shift is successfully cancelled (meaning that the stop process won the race on these trials). Following this reasoning, is it possible that recruitment of the antagonist muscle is a manifestation of the stop process?!

To answer this question, we derived the SSRT for both gaze shifts and head movements. We estimated SSRTs via the integration method and the mean method (Hanes and Schall 1995; Logan 1994). Because the integration and mean methods yield equally valid estimates for both gaze and head SSRTs, we combined these estimates to yield an average SSRT for each subject for both the gaze and head (see Table 2).

![Fig. 7. Schematic showing the onset of antagonist EMG activity (■) in relation to stop signal onset (‖) for all subjects across both directions and for each SSD. ○, target onset. Below each plot is a histogram showing the distribution of reaction times of the antagonist burst for each subject. The mean and SD of the antagonist latency in relation to the onset of the stop signal is also shown for each subject.](image-url)
A number of past and recent findings have concluded that there may be multiple forms of stopping, depending on how close the movement is to being initiated at the time of cancellation (Aron and Verbruggen 2008; De Jong et al. 1995). Briefly, there is good evidence from manual movements of the existence of central and peripheral stopping processes and that the durations of these stop processes may vary independently of one another. Based on this, canceling a gaze shift following a head-only movement (from which the antagonist muscle latencies are derived) may involve a peripheral stopping process, whereas fully canceling any motion of the gaze or the head may involve a different central stopping process. If this was to be the case, then perhaps the correlation between gaze SSRT and antagonist muscle latencies arises from the inclusion of head-only trials in the gaze inhibition functions. To investigate this, we recomputed gaze SSRTs for each subject after excluding head-only errors and compared these derived SSRTs with the antagonist muscle latencies; note that in this analysis, the trials from which gaze inhibition functions are calculated are different from the trials from which the antagonist muscle latencies are measured. As shown in Fig. 9C, a strong correlation persists between gaze SSRTs derived without head-only errors and antagonist muscle latencies \(r = 0.91, P = 2 \times 10^{-3}\). Thus even if it was the case that there are different central and peripheral stopping processes, this result demonstrates that such processes are not independent in the oculomotor system. As will be clarified in the DISCUSSION, we support an alternative mechanism whereby the antagonist muscle latency represents a peripheral expression of the central stopping process.

Although the antagonist muscle latencies correlated well with SSRT, on average antagonist muscle latencies are ~50 ms longer. However, SSRTs and antagonist muscle latencies are derived from a different subset of SSDs. Specifically, perhaps antagonist muscle latencies arise preferentially from trials with a shorter subset of SSDs than those used to calculate SSRTs and hence are slightly longer (recall that antagonist latencies and SSRTs tend to decrease for longer SSDs; Fig. 8B). To address this, we derived SSRTs via the integration method at those SSDs where we also observed at least three head-only errors and plotted the average antagonist latency as a function of the derived SSRT (Fig. 9D; in this analysis, multiple values could be extracted from a given subject). Even after constraining our data in this way, the antagonist muscle latency remained well correlated with SSRT.

We then compared these SSRT estimates to the antagonist muscle latencies measured from individual head-only error trials. We observed a very strong correlation between antagonist muscle latency and gaze SSRTs on a subject-by-subject basis, meaning that subjects with longer SSRTs tended to have longer antagonist latencies (Fig. 9A, \(r = 0.96\)); this result demonstrates that 92% of the variance in antagonist latencies across subjects can be explained by the variance in gaze SSRTs across subjects. For all subjects, gaze SSRT estimates were lower than the antagonist muscle latencies by ~50 ms on average (51 ± 12 ms; range: 26–66 ms). Antagonist muscle latencies also correlated well with estimates of the head SSRT (Fig. 9B, \(r = 0.81\)), and although head SSRTs were generally less than the antagonist muscle latencies, the difference between these measures was more variable than that seen for gaze SSRTs (19 ± 22 ms; range: −11 to 51 ms).

We also estimated the variance of the antagonist burst to determine how well it compared with other methods that estimate the variance of the stop process. We used the maximum likelihood model described in Corneil and Elsley (2005) because this model incorporates a lower threshold for initiating head movements, consistent with our findings. The results of this analysis show that the variance estimated using this model correlated well with the variance observed in the antagonist latency (paired \(t\)-test, \(t = -5.15, P = 1 \times 10^{-3}\)).

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**TABLE 2.** Estimates of gaze and head stop signal reaction times (SSRTs) and the latency of the antagonist burst

<table>
<thead>
<tr>
<th>Subject</th>
<th>Gaze SSRTs, ms</th>
<th>Head SSRTs, ms</th>
<th>Antagonist Latency, ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>100</td>
<td>151</td>
<td>152</td>
</tr>
<tr>
<td>S2</td>
<td>170</td>
<td>205</td>
<td>194</td>
</tr>
<tr>
<td>S3</td>
<td>139</td>
<td>175</td>
<td>192</td>
</tr>
<tr>
<td>S4</td>
<td>163</td>
<td>202</td>
<td>217</td>
</tr>
<tr>
<td>S5</td>
<td>85</td>
<td>103</td>
<td>152</td>
</tr>
<tr>
<td>S6</td>
<td>134</td>
<td>168</td>
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<td>S8</td>
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</tr>
<tr>
<td>Mean</td>
<td>129 ± 31</td>
<td>162 ± 33</td>
<td>180 ± 26</td>
</tr>
</tbody>
</table>

Means are ± SD.
reflecting the efferent lag in the head movement system. The initiation of antagonist muscle activity did not appear on trials when head motion was cancelled or on trials when the eye-head gaze shifts landed on target. The correlation between the latency of the antagonist burst and the gaze SSRT measured using the integration method after excluding trials where a head-only error occurred. The correlation between the antagonist latency and the gaze SSRT measured using the integration method at SSDs was $\geq 3$ head-only errors were observed. Each point indicates a unique combination of antagonist latency and SSRT measured across all subjects. The correlation co-efficients and corresponding $P$ values for these correlations are shown for each comparison.

We have described the patterns of neck muscle activity in humans as they attempt to countermand eye-head gaze shifts. We focused in particular on neck muscle activity during head-only errors as these sequences represent a situation where a target-directed head movement is initiated even though gaze remains stable. During head-only errors, which were originally described in Corneil and Elsley (2005), antagonist neck muscles produced a burst of activity that increased during larger and faster erroneous head movements. Such antagonist muscle activity did not appear on trials when head motion was completely cancelled or on trials when the eye-head gaze shifts landed on target. The initiation of antagonist muscle activity related most closely to the onset of the stop signal rather than the target, correlating well with estimates of the duration of the concurrent response processing or response conflict that invariably arises on stop trials. Aspects of our data are incon-

Alternative explanations for antagonist neck muscle activity

A number of central processes are engaged during stop trials, some of which could result in the recruitment of antagonist neck muscles. For example, could our results be due to the concurrent response processing or response conflict that invariably arises on stop trials? Aspects of our data are incon-
sistent with both interpretations. First, if the concurrent processing of both the go and stop signal were to percolate through to the motor periphery, then one could expect simultaneous activation of both agonist and antagonist neck muscles. As shown in Fig. 3, such co-activation was never observed. Second, if antagonist neck muscle activity arises from response conflict, then such neck muscle activity should appear on every stop trial. Neurophysiological correlates of the go and stop process are present on every stop trial regardless of outcome (Hanes et al. 1998; Paré and Hanes 2003). Instead our data clearly show that antagonist neck muscle activity arises only on a subset of stop trials (Fig. 3). Third, the gradation of antagonist neck muscle activity with the kinematics of the erroneous head movement (Fig. 6) is also hard to reconcile with response conflict, which presumably is either present or not. Finally, the latency of antagonist neck muscle activity relative to stop signal presentation, which averaged 180 ms in our sample, is very short when one considers the efferent lag of the head movement system (~20 ms in monkeys; Elsley et al. 2007), and likely longer in humans considering longer conduction distances). Thus whatever cortical process initiates antagonist neck muscle activity must begin ~160 ms after stop signal presentation, which is shorter than the peak of N200 event-related potentials considered a signal of conflict (Enriquez-Geppert et al. 2010).

Although the presentation of the stop signal can initiate multiple processes, including response conflict and concurrent response processing, we suggest that such processes do not necessarily influence the motor periphery. It is only in the special circumstance where the head has already started to move that the presentation of the stop signal also leads to the recruitment of antagonist neck muscle activity. The scaling of antagonist neck muscle activity with the kinematics of the erroneous head movement suggests that such activity serves the functional role of actively braking the head. From this perspective, the antagonist neck muscle activity results from an error-correcting mechanism. It seems logical to surmise that the correction of the head error was initiated by the oculomotor error-correcting mechanism. It seems logical to surmise that the correction of the head error was initiated by the oculomotor error-correcting mechanism. It seems logical to surmise that the correction of the head error was initiated by the oculomotor error-correcting mechanism. It seems logical to surmise that the correction of the head error was initiated by the oculomotor error-correcting mechanism. It seems logical to surmise that the correction of the head error was initiated by the oculomotor error-correcting mechanism.
propose that oculomotor go and stop process interact briefly very late in the race, with the stop process potently inhibiting the go process. The prevalence of mid-flight corrections we observed, during both head-only errors and noncancelled truncated eye-head gaze shifts, attests to the persistence and potency of the stop process even after a movement has begun.

**How is the oculomotor stop process integrated into head movement control?**

The selective recruitment of antagonist muscles on head-only errors, but not on cancelled stop trials without head motion, leads to the question of the underlying neural mechanisms. Recordings in the FEF and SC of head-restrained monkeys have determined that the timing of the silencing of movement-related neurons and the simultaneous re-activation of fixation-related neurons is appropriate to mediate the control of saccades (Hanes et al. 1998; Paré and Hanes 2003). However, it is unlikely that either of these mechanisms could directly recruit the antagonist muscle activity observed on head-only errors. Increasing levels of activity of movement-related neurons in the SC is correlated with increasing levels of agonist, not antagonist, neck muscle activity (Rezvani and Corneil 2008). Moreover, stimulation in the rostral SC or the lateral FEF (near fixation-related neurons) never evokes a profile of neck muscle activity resembling active braking but instead evokes weak agonist muscle recruitment associated with small-amplitude gaze shifts (Corneil et al. 2002a; Elsley et al. 2007; Guitton and Mandl 1978; Roucoux et al. 1980). Stimulation of the omnipause neurons (OPNs) that directly inhibit the saccadic burst circuitry also fails to actively cancel an ongoing head motion (Gandhi and Sparks 2007). Thus it appears unlikely that the recruitment of antagonist neck muscle activity by the stop process is mediated by oculomotor fixation circuits.

Instead we speculate that the stop process engages with other premotor head circuits in parallel with the engagement of oculomotor fixation circuits. At the current time, very little is known about how head movements are cancelled or truncated in mid-flight, and it is an open question whether similar central mechanisms are even engaged (the absence of antagonist activity on cancelled trials without head movements attests to different peripheral strategies). The successful cancellation of limb movements engages a variety of areas in a fronto-basal ganglia circuit (see Verbruggen and Logan 2008 for review), and it is possible that such circuits may also be involved in canceling or truncating head movements. Another candidate is the precentral cortex lying posterior to the FEF in the monkey. This area is labeled bilaterally following the injection of trans-synaptic retrograde tracers into neck muscles (Billig and Strick 2009), and stimulation of this area provokes defensive movements that include an ipsilaterally directed head movement (Boulanger et al. 2009; Graziano et al. 2002) in contrast to the contralaterally directed head movement evoked from the nearby FEF. Regardless of the precise mechanism, there are strong correlations between the timing of antagonist muscle recruitment and the SSRT estimates of the oculomotor stop process (Fig. 9), and similar trends in how these measures change across SSD (Fig. 5). These results imply that the timing of completion of the oculomotor stop process can be indexed via measurements of neck muscle recruitment on a within-trial basis.

**Implications for the study of countermanding**

The countermanding paradigm and the race model have become increasingly popular in both behavioral and neurophysiological settings because they provide a framework for investigating movement cancellation. We suggest that recordings of neck muscle activity could be deployed in both settings as a more objective measure of the latency of the stop process. For example, in behavioral experiments in humans, the distribution of the latencies of antagonist neck muscle activity provide both a measure of the mean value (comparable to estimates of SSRTs) and a direct measure of its variance (which is not attainable with current estimates of SSRTs). Such measures could then be compared, for example, across different types of stop signals, or across different clinical populations or treatment modes.

In a neurophysiological setting, latencies of antagonist neck muscle recruitment could provide a new type of boundary condition for assessing whether a given area is directly involved in movement cancellation. Currently, the determination of a candidate area is based on whether the divergence of neural activity prior to cancelled or noncancelled stop trials (termed the neural SSRT) precedes the behavioral SSRT. Both neural and behavioral SSRTs are calculated across a large sample of trials and hence do not directly provide insights into the trial-by-trial fluctuations in the stop process. Such insights could come from the addition of neck muscle activity; logically, neural activity would then have to precede the antagonist neck muscle latencies on every trial (presumably by a relatively fixed latency) to be directly involved in movement cancellation.

Two issues may limit the feasibility of employing measurements of neck muscle activity in behavioral and neurophysiological settings. First, given the anatomy of the neck, the intramuscular recording techniques we have used to ensure proper electrode placement are both invasive and involved. Our rationale for using intramuscular electrodes was to avoid cross-talk from nearby muscles (Mayoux-Benhamou et al. 1995). In retrospect we speculate that surface recordings of neck muscle activity may be sufficient. Sternocleidomastoid (SCM) is a contralateral head turning muscle that lies quite close to the surface of the ventral neck, whereas other muscles near SPL are not involved in head turns. Both SCM and SPL are commonly recorded with surface electrodes (Zangemeister and Stark 1981), and potential concerns of cross-talk from muscles such as trapezius could be alleviated somewhat by restricting the task to the horizontal plane. Moreover, if the main experimental measure is the timing of muscle activation, which specific muscle(s) the signal originated from is less important. It remains to be confirmed that active braking on neck muscles can be recorded via surface techniques during an countermanding task, but we note that previous research has utilized surface electrodes to record active braking on SPL during rapid head turns (Zangemeister and Stark 1981).

A second issue relates to the relatively low yield of head-only errors. In our subject pool, head-only errors were produced on ~20% of stop trials, which themselves comprised only 30% of the total trial count. This yield could essentially be
doubled by including neck muscle activity from truncated noncancelled stop trials (Table 2). Although we chose not to include such trials in our comparative analysis of antagonist neck muscle latency with SSRTs, because the go process technically wins the race on such trials, the stop process is still engaged to arrest the trial in mid-flight. While we note that other studies have reported truncated movements previously in other tasks (Boucher et al. 2007b; Ramakrishnan et al. 2010), we observed a much higher incidence in this study. Presumably, the use of larger amplitude gaze shifts (which last longer) provides more opportunity for mid-flight movement cancellation. Thus appropriate experimental design can increase the number of trials in which the stop process is expressed in the motor periphery.

Finally, we emphasize again that our results depend critically on eye-head gaze shifts representing a form of nested control, enabling instances where the lower (head) component can begin to move even though the uppermost (gaze) component remains stable. We speculate that the insights that we have made may extend to other motor systems, providing they involve nested control. Chief among these would be the study of multi-segmental pointing or grasping, where the accuracy of the overall goal depends on the combined actions at multiple underlying segments. Such a scenario may enable instances where movements at the lowest segments (such as the shoulder) are initiated and cancelled prior to overt movement of the distal-most effector. To our knowledge, whereas many studies have investigated the control of manual movements (occasionally reporting subtle recruitment of agonist muscles on otherwise cancelled movements) (Boucher et al. 2007b; De Jong et al. 1990), most of these studies have investigated relative simple manual movements, such as a key press or a button squeeze, that do not involve nested control. Moreover, few if any have additionally considered the recruitment of antagonist muscles. Our results from the oculomotor system suggest that this may be a fruitful avenue of investigation.

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Disclosures

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