

Validation of a within-trial measure of the oculomotor stop process

Samanthi C. Goonetilleke,¹ Jeffrey P. Wong,¹ and Brian D. Corneil^{1,2,3}

¹Department of Physiology and Pharmacology, Western University, London, Ontario, Canada; ²Department of Psychology, Western University, London, Ontario, Canada; and ³The Brain and Mind Institute, Robarts Research Institute, London, Ontario, Canada

Submitted 27 February 2012; accepted in final form 10 May 2012

Goonetilleke SC, Wong JP, Corneil BD. Validation of a within-trial measure of the oculomotor stop process. *J Neurophysiol* 108: 760–770, 2012. First published May 16, 2012; doi:10.1152/jn.00174.2012.—The countermanding (or stop signal) task requires subjects try to withhold a planned movement upon the infrequent presentation of a stop signal. We have previously proposed a within-trial measure of movement cancellation based on neck muscle recruitment during the cancellation of eye-head gaze shifts. Here, we examined such activity after either a bright or dim stop signal, a manipulation known to prolong the stop signal reaction time (SSRT). Regardless of stop signal intensity, subjects generated an appreciable number of head-only errors during successfully cancelled gaze shifts (compensatory eye-in-head motion ensured gaze stability), wherein subtle head motion toward a peripheral target was ultimately stopped by a braking pulse of antagonist neck muscle activity. Both the SSRT and timing of antagonist muscle recruitment relative to the stop signal increased for dim stop signals and decreased for longer stop signal delays. Moreover, we observed substantial variation in the distribution of antagonist muscle recruitment latencies across our sample. The magnitude and variance of the SSRTs and antagonist muscle recruitment latencies correlated positively across subjects, as did the within-subject changes across bright and dim stop signals. Finally, we fitted our behavioral data with a race model architecture that incorporated a lower threshold for initiating head movements. This model allowed us to estimate the efferent delay between the completion of a central stop process and the recruitment of antagonist neck muscles; the estimated efferent delay remained consistent within subjects across stop signal intensity. Overall, these results are consistent with the hypothesis that neck muscle recruitment during a specific subset of cancelled trials provides a peripheral expression of oculomotor cancellation on a single trial. In the DISCUSSION, we briefly speculate on the potential value of this measure for research in basic or clinical domains and consider current issues that limit more widespread use.

countermanding; eye-head coordination; neck muscles

CONTEXTUAL CONTROL in a dynamic environment occasionally requires the abrupt cancellation or alteration of an impending movement. This aspect of contextual control can be studied in the laboratory via a countermanding (or stop signal) paradigm, which requires that subjects engaged in a response task occasionally try to cancel this movement upon the infrequent presentation of a stop signal. This paradigm enables estimation of the stop signal reaction time (SSRT), or the time needed to cancel the impending action (Colonius 1990; Logan and Cowan 1984). As reviewed elsewhere (Verbruggen and Logan 2008), the SSRT can serve as a benchmark to differentiate neural processes directly related to movement cancellation from those related to the adaptive adjustment of behavior

(Hanes et al. 1998; Paré and Hanes 2003; Stuphorn et al. 2010), a metric for regression in imaging studies (Li et al. 2006) and as a means to assess inhibition or impulsivity in clinical and developmental studies (Floden and Stuss 2006; Schachar et al. 2004). Because the SSRT is estimated rather than objectively measured, it is not possible to directly assess the variability of stopping, even though this measure may be particularly relevant in clinical domains.

Our recent work has examined how humans countermand large, head-unrestrained gaze shifts (Corneil and Elsley 2005). In this task, subjects may either successfully or unsuccessfully cancel an eye-head gaze shift (Fig. 1C, *left* and *middle*); these two sequences are the analogous to cancelled and noncancelled saccades made with the head restrained (Hanes and Carpenter 1999). Releasing the head permits the expression of a third sequence consisting of a head-only movement to the target while gaze remains stable due to compensatory eye motion (Fig. 1C, *right*). Such head-only movements represent an intermediary type of error reflecting the nested nature of the eye within a mobile head; although gaze remains stable (hence subjects successfully cancelled the gaze shift), the head begins to orient to the target. Head-only movements are uniquely associated with a selective burst of antagonist neck muscle activity that actively brakes the head (Goonetilleke et al. 2010). Across subjects, the timing of this antagonist burst relative to the stop signal (the antagonist muscle latency) correlated positively with estimates of the SSRT, even though antagonist muscle latencies were ~50 ms longer. These observations are consistent the hypothesis that antagonist muscle recruitment arises as a peripheral manifestation of oculomotor cancellation, with longer antagonist muscle latencies arising from the efferent delay from cancellation of the oculomotor program to the onset of antagonist muscle recruitment. If true, this hypothesis suggests that expressions of oculomotor cancellation are available within a single trial.

The goal of this study was to test this hypothesis across a manipulation known to influence the SSRT: the intensity of a visual stop signal. Since dimmer stop signals prolong and increase the variance of SSRTs (Morein-Zamir and Kingstone 2006), our hypothesis predicted a similar influence on the latencies of antagonist muscle recruitment. The collection of a large data set also allowed us to examine the distribution and variance of antagonist muscle recruitment latencies across our sample and across the manipulation of stop signal intensity. Finally, given that we had a data set of both SSRTs and antagonist muscle recruitment latencies for both dim and bright stop signals, we could also compare within-subject changes in these values. Consistent with our hypothesis, our results em-

Address for reprint requests and other correspondence: B. D. Corneil, The Brain and Mind Institute, Robarts Research Institute, London, ON, Canada N6A 5K8 (e-mail: bcorneil@uwo.ca).

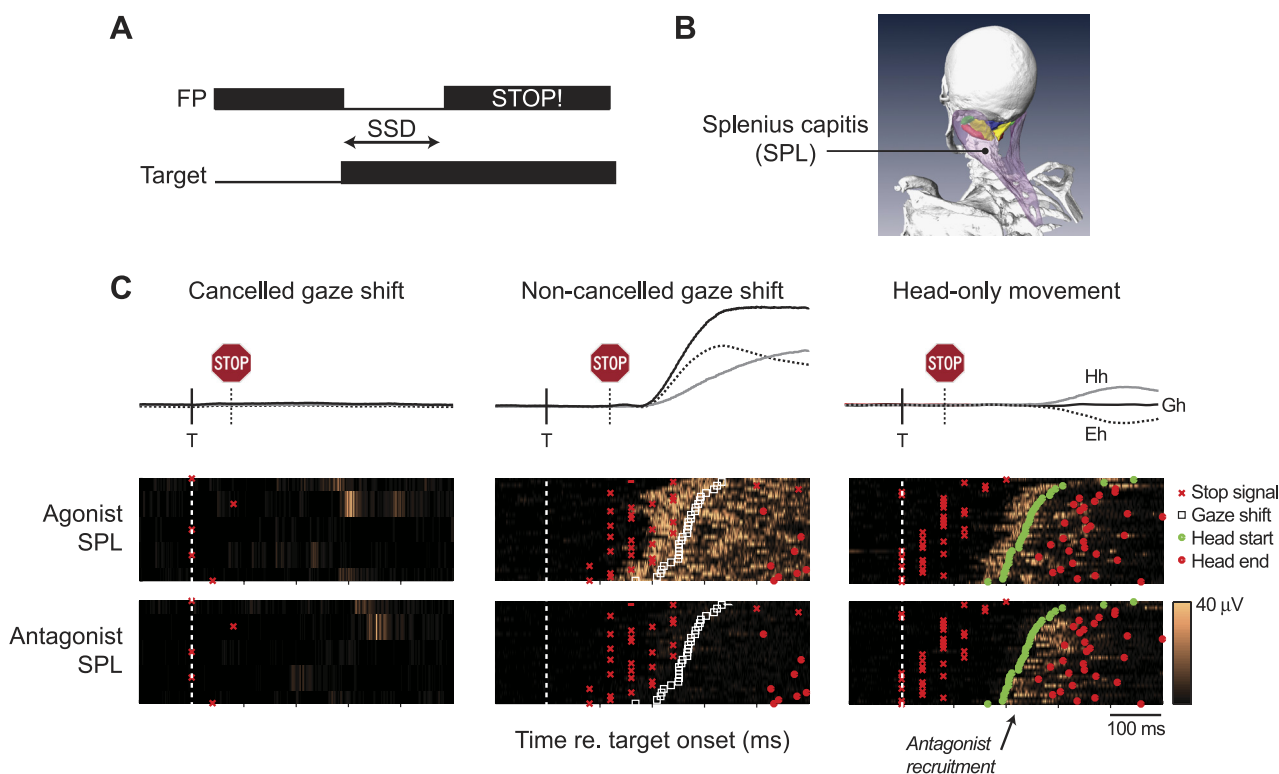


Fig. 1. *A*: depiction of timing of a stop trial, in which the reillumination of the central fixation point (FP) after target presentation cues the subject to try to cancel the impending eye-head gaze shift. SSD, stop signal delay, or time between target and stop signal presentation. *B*: neck muscle recruitment was measured bilaterally from splenius capitis (SPL), a large and relatively superficial neck muscle involved in ipsilateral head turns. *C*: representative examples of the three sequences observed during stop trials (taken from *subject 8*), showing the coordination of horizontal eye-in-head (Eh), head-in-space (Hh), and eye-in-space (Gh), and bilateral SPL recruitment (the agonist SPL serves to move the head toward the target). Note how the eye-in-head counterrotation during head-only errors ensures gaze stability; hence, head-only errors are a subtype of successfully cancelled gaze shifts. For the subplots showing neck muscle activity, each row represents data from a different trial, aligned on the target onset and ordered when possible by the movement onset. Note that there was antagonist muscle recruitment selectively during head-only errors (arrow in *C*).

phasized a close relationship between estimates of SSRTs and objective measures of antagonist muscle latencies.

METHODS

Eight human subjects (3 women and 5 men; mean age: 27 yr) participated in the experiment. Experimental procedures were approved by the University Research Ethics Board for Health Science Research of the University of Western Ontario and were in accordance with the Declaration of Helsinki (1967). Subjects gave informed written consent and were aware they could terminate testing at any time. No subjects reported any neurological or musculoskeletal deficits, and all had normal or corrected to normal vision. The three authors (*subjects 1, 4, and 6*) were knowledgeable about the specific experimental objectives, but their results did not differ from remaining subjects who were naïve to experimental goals.

Countermanding task. Subjects performed an oculomotor countermanding task (Fig. 1*A*) with their head unrestrained, as described in greater detail elsewhere (Corneil and Elsley 2005; Goonetilleke et al. 2010). Briefly, the primary task (no-stop trials) required subjects to generate gaze shifts from a central fixation point (presented for between 1 and 1.5 s) to the peripheral targets located 60° to the left and right. Target eccentricity was selected to be well outside the oculomotor range for humans (Guitton and Volle 1987; Stahl 1999); thus, gaze shifts required coordinated movements of the eyes and head. On a subset of trials (stop trials; 30% of all trials), a stimulus directly above the central fixation point was illuminated after target presentation. The interval between the target onset and stop signal was termed the stop signal delay (SSD). All stimuli were light-emitting

diodes (LEDs). When presented, the intensity of the stop signal was either bright (110 cd/m²) or dim (20 cd/m²) with equal probability. Subjects were instructed to try to withhold a gaze shift to the target upon presentation of the stop signal and were not given any instruction about eye-head coordination on either trial type or any feedback on performance.

All subjects completed 6 blocks of 204 trials each (1,224 trials in total per subject), with short breaks in between blocks. All experimental factors (target direction, trial type, stop signal intensity) were pseudorandomly presented with a block of trials by a customized LabView program that controlled the experiment at a rate of 1 kHz (National Instruments). This program also varied the SSD using an adaptive staircasing algorithm so that subjects cancelled gaze shifts on ~50% of all stop signal trials. This algorithm manipulated the ease of stopping by adjusting the SSD based the outcome of the previous stop signal trial, increasing (decreasing) the SSD by either 40 or 80 ms if the previous stop signal trial was successfully (unsuccessfully) cancelled, respectively. Separate staircasing algorithms ran for bright or dim stop signals. Both were initialized at 80 ms and ranged between 0 ms (i.e., target and stop signal presented simultaneously) and 600 ms (no subject reached this maximum SSD).

Data collection and analysis. As previously described (Goonetilleke et al. 2010), we recorded horizontal eye movements via bitemporal electrooculography, head movements via an infrared tracking system, and the electromyographic (EMG) recruitment of splenius capitis (SPL; Fig. 1*B*), a neck muscle known to contribute to horizontal head turns in the ipsilateral direction. EMG recordings were made via in-house intramuscular fine wire needle electrodes inserted at the level of the C4/C5 vertebrae [electrodes consisted of seven-stranded stain-

less steel wire (A-M Systems, Sequim, WA) threaded into a 30-mm 25-gauge cannula (Kendal Monoject, Mansfield, MA)]. Because we sought to characterize SPL recruitment across a population of multiple motor units, we used two monopolar electrodes staggered by ~ 5 mm parallel to the long axis of the muscle fibers. Electrode placement was confirmed by strong recruitment during small ipsilateral head rotation and the absence of recruitment to shoulder shrugs or contralateral head rotation. EMG data were recorded with a Myopac Jr (Run Technologies, Mission Viejo, CA; low-pass filter modified to 2 kHz). All EMG and position data were sampled at 4 kHz and digitized with a 16-bit converter onto a MotionMonitor system (Innovative Sports Training, Chicago, IL).

All offline analyses were performed in Matlab. EMG data were full wave rectified and then bin integrated into 1-ms bins, and horizontal eye and head rotation was downsampled to 1 kHz and then added together to yield horizontal gaze position. Movement onsets and offsets were detected by a computer algorithm whenever motion crossed velocity thresholds ($50^\circ/\text{s}$ for gaze and $10^\circ/\text{s}$ for head). These marks were used as guides for the placement of interactive marks by an analyst within a graphical user interface. Movement amplitudes and peak velocities were extracted between the onset and offset of these marks. The analyst also marked the onset and offset of muscle recruitment. Guide marks for the onset (offset) of muscle recruitment were placed when muscle activity exceeded 3 SD (or returned within 1 SD) of the mean EMG activity during the 200 ms preceding target presentation; these marks could also be interactively moved by the analyst. The behavior on a given stop trial was divided into one of three categories (Fig. 1; see also RESULTS). On cancelled trials, neither the gaze axis nor head moved. On noncancelled trials, both the gaze axis and head moved in the direction of the target. On head-only errors, the head moved toward the target, but gaze remained stable due to counterrotation of the eye within the head. Because gaze remained stable during head-only errors, such trials are a subtype of cancelled gaze shifts.

Some analyses related to movement cancellation (e.g., construction of inhibition functions expressing the rate of movement probability as a function of SSD) required a minimum of 10 repeats of a given combination of SSD and stop signal intensity. Trials were excluded if movements began within <80 or >800 ms, proceeded away from the peripheral target, or exhibited abnormal profiles of muscle recruitment related to, for example, shifts in body position. Less than 5% of all trials were excluded with these criteria. Details of other statistical analyses are provided in the RESULTS.

RESULTS

Behavior and neck muscle recruitment during the attempted cancellation of gaze shifts. As previously reported (Corneil and Elsley 2005; Goonetilleke et al. 2010), subjects generated three movement sequences when attempting to cancel large impending gaze shifts: they either fully cancelled motion of both the eyes and head ($12.5 \pm 7.8\%$ of all stop signal trials; Fig. 1C, left), generated a coordinated eye-head gaze shift to the target ($50.5 \pm 6.0\%$ of all stop signal trials; Fig. 1C, middle), or generated a head-only movement where the head moved toward the target but a compensatory eye-in-head movement ensured stability of the gaze axis ($37.0 \pm 10.1\%$ of all stop signal trials; Fig. 1C, right). Note that the first two sequences are complementary to the cancelled or noncancelled saccades made in a head-restrained study of saccade countermanding (Hanes and Carpenter 1999) but that the third movement sequence is predicated on the head being free to move. Note as well that gaze remains stable during the cancelled and head-only movement sequences, meaning that subjects successfully countermanded gaze shifts. The propensity of these movement sequences did

not differ systematically with either a bright or dim stop signal (paired *t*-test of propensity across intensity, $P > 0.2$ for all comparisons). We never observed a type of eye-only error (i.e., a target-directed eye movement without an accompanying head movement), likely because the targets were well beyond the oculomotor range.

Our more recent work (Goonetilleke et al. 2010) assessed the bilateral recruitment of SPL during these three movement sequences. As previously reported, and regardless of stop signal intensity, each sequence was associated with a unique spatio-temporal profile of SPL recruitment (shown for a representative subject in Fig. 1C). Bilateral SPL recruitment was absent on the majority of successfully cancelled trials (Fig. 1C, left). Strong agonist and negligible antagonist SPL recruitment accompanied noncancelled eye-head gaze shifts that attained the target (Fig. 1C, middle) and during eye-head gaze shifts on no-stop signal trials (not shown). Finally, although strong agonist SPL recruitment accompanied head-only errors, this movement sequence also featured consistent and robust recruitment of antagonist SPL (Fig. 1C, right, arrow). Such antagonist muscle recruitment occurred during the erroneous head motion to the target and hence is a lengthening contraction that serves to actively brake the head. Our previous work (Goonetilleke et al. 2010) showed that the magnitude of antagonist muscle recruitment correlated with the metrics and kinematics of erroneous head motion, with larger antagonist muscle activity accompanying larger and/or faster head movements. Here, we were particularly interested in the timing of antagonist muscle recruitment and how it relates to the SSRT.

Prolonged movement cancellation for dimmer stop signals. As expected based on previous results (Morein-Zamir and Kingstone 2006), subjects required more time to stop an impending gaze shift when presented with a dim stop signal. This result was attested to by the leftward shift in the inhibition functions that expressed gaze shift probability as a function of SSD (Fig. 2A), i.e., a dim stop signal had to be presented earlier than a bright stop signal to result in an equal probability of successful cancellation. Note that all inhibition functions appeared fairly symmetrical, ranging between less than about 0.2 for the shorter SSDs to above 0.9 for the longer SSDs; this means that the dynamic adjustment of the SSDs was successful in getting subjects to cancel gaze shifts on approximately half of all stop trials. Finally, note from Fig. 2A that although the inhibition functions shifted left for all subjects for dim stop signals, the amount of the shift varied substantially, ranging from a very small shift for *subject 2* (differing only at the longer SSDs) to larger shifts of ~ 75 ms for *subject 4*.

Figure 2B shows the proportion of head-only errors as a function of SSD for both dim and bright stop signals. Head-only errors were equally likely for bright and dim stop signals (paired *t*-test, $t_7 = 1.1$, $P = 0.3$), being generated on average in $38.1 \pm 10.6\%$ (mean \pm SD; range: 25.5–55.4%) or $35.8 \pm 10.4\%$ (range: 22.1–48.4%) of stop trials with a bright or dim stop signal, respectively. Head-only errors tended to occur at the early and intermediate SSDs within the range of SSD. Recall that head-only errors are a subtype of a successfully cancelled gaze shifts; in some subjects, essentially all of the successfully cancelled gaze shifts at a given SSD were head-only errors (e.g., *subject 1* at the lower SSDs). Thus, although we gave no explicit instructions to encourage head-only errors, they are a common sequence that provides a considerable yield

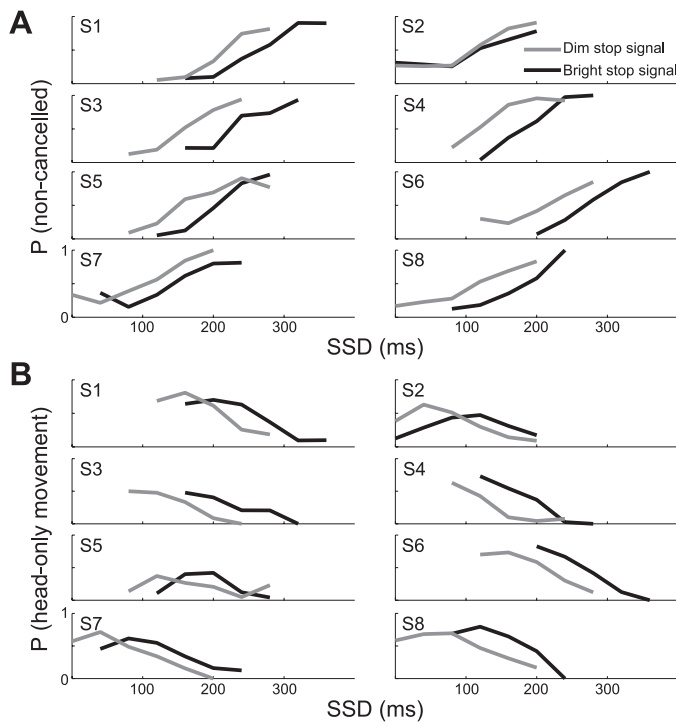


Fig. 2. Plots of the proportion of noncancelled gaze shifts (A; also known as inhibition functions) and proportion of head-only errors (B) as a function of SSD. Functions were plotted separately for dim and bright stop signals.

to examine how muscle recruitment varies across different stop signal intensity.

Using the inhibition functions and distribution of reaction times on no-stop signal trials, we calculated the SSRT for gaze shifts using both the integration and mean method as well as the average of these two methods (Table 1) (see Goonetilleke et al. 2010 for further details). SSRTs were significantly longer for dim (154 ± 17.3 ms) versus bright stop signals (102 ± 32.2 ms, paired t -test, $t_7 = 6.32$, $P < 10^{-4}$), indicating that subjects required more time to stop a planned gaze shift with a dim stop signal.

Antagonist muscle latency: alignment, distribution, and relationship with SSD. Every head-only error provides an opportunity to measure the timing of the recruitment of the antagonist muscle and, we hypothesized, the cancellation of an oculomotor program. One prediction of this hypothesis is that the timing of antagonist muscle recruitment should be better

aligned to the onset of the stop signal rather than the peripheral target. Across our sample, the coefficient of variation for the timing of neck muscle recruitment was significantly lower when aligned to the stop signal rather than the target, regardless of stop signal intensity (bright stop signals: paired t -test, $t_7 = 5.15$, $P = 0.001$; dim stop signals: paired t -test, $t_7 = 4.79$, $P = 0.002$).

Figure 3 shows the timing of antagonist muscle recruitment for every head-only movement observed in our sample (recall data from *subject 8* was shown in Fig. 1C). Based on our analysis of the coefficient of variation, we aligned these data relative to the stop signal (within each subplot, trials are stacked in terms of decreasing SSD so that the SSD for a given trial is the time between the circle on the left and the white vertical line). Above each plot in Fig. 3 is a histogram showing the distribution of antagonist muscle recruitment latencies for each subject. We observed substantial variation in the shape, breadth, and magnitude in this measure, with the latency distribution appearing unimodal in some subjects and bimodal in others. Across our sample, the average median antagonist muscle latency for dim stop signals was 216 ± 42 ms (range: 169–294 ms) versus 173 ± 51 ms for bright stop signals (range: 123–266 ms; see also Table 1). Thus, as with the SSRT, the latency of antagonist muscle recruitment increased significantly for dimmer stop signals (paired t -test, $t_7 = 7.68$, $P < 10^{-4}$).

The race model provides a conceptual framework that produces testable predictions about how behavior should change with different SSDs. One prediction is that the SSRT should decrease for longer SSDs (Logan and Cowan 1984); if the stop process varies stochastically, cancellation at longer SSDs only arises on trials when movement cancellation proceeds more quickly, since there is less time available for stopping. We therefore looked at the relationship of both the SSRT (estimated by the integration method at a given SSD) and the antagonist muscle latency across SSD (since the range of SSDs differed for each subject, SSDs were rank ordered from shortest to longest). This analysis was done separately for dim (Fig. 4A) or bright (Fig. 4B) stop signals, with either the SSRT or antagonist muscle latency normalized to the value at the shortest SSD before being pooled across subjects. For both dim and bright stop trials, both SSRTs and antagonist muscle latencies decreased with increasing SSDs, consistent with the

Table 1. Estimates of gaze SSRTs and the median latency of the antagonist burst for both dim and bright stop signals

	Dim Stop Signal				Bright Stop Signal			
	Mean method, ms	Integration method, ms	Average SSRT, ms	Antagonist latency, ms	Mean method, ms	Integration method, ms	Average SSRT, ms	Antagonist latency, ms
Subject 1	128	132	130	210	76	81	78	174
Subject 2	153	204	178	294	155	187	171	266
Subject 3	162	191	177	243	84	121	102	176
Subject 4	137	159	148	191	72	92	82	133
Subject 5	192	140	166	180	100	120	110	131
Subject 6	131	154	143	192	60	85	72	149
Subject 7	122	170	146	248	121	115	118	230
Subject 8	131	161	146	169	62	106	84	123
Mean	145	164	154	216	91	113	102	173

SSRT, stop signal reaction time.

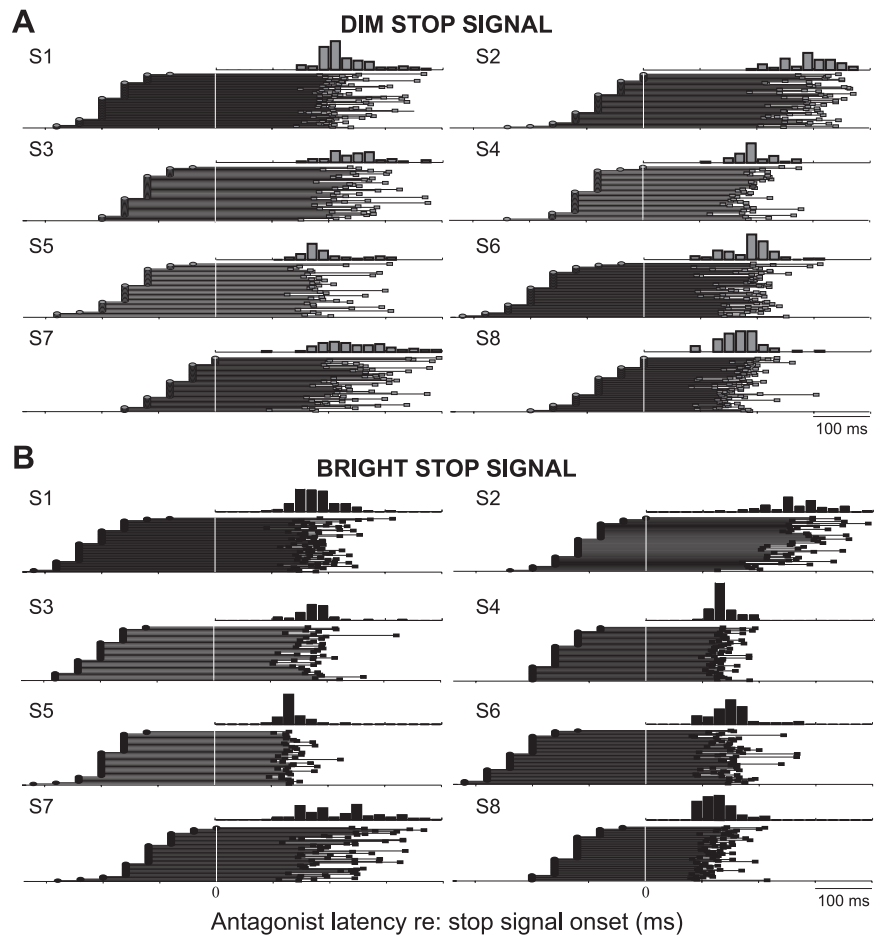


Fig. 3. Distributions of antagonist muscle latencies, relative to the onset of a dim (*A*) or bright (*B*) stop signal. In each subplot, each line in the lineplot represents a single observation, running from the target onset (circle on the left), aligned on the stop signal onset (white vertical line), and ending at the onset of antagonist muscle recruitment (square on the right).

predictions of the race model ($P < 0.001$ for regressions in Fig. 4, *A* and *B*).

Correlations between the duration and variance of SSRTs and antagonist muscle latencies. We now turn to the relationship between SSRTs and antagonist muscle recruitment across and within subjects. As shown in our previous work (Goonetilleke et al. 2010), although SSRTs were ~ 60 – 70 ms shorter than antagonist muscle latencies (consistent with an efferent delay in conveying a signal to the motor periphery), these measures were positively correlated across our sample, meaning that subjects with a shorter SSRT tended to also have shorter antagonist muscle latencies (Fig. 5*A*; each subject contributed two points to this plot, one from each stop signal intensity, $r = 0.76$, $P < 0.001$). We also observed a positive correlation between estimates of the variance of the SSRT (obtained from the integration method) with the interquartile range obtained from the antagonist muscle response ($r = 0.51$, $P = 0.04$; Fig. 5*B*). Thus, when our data were pooled across our sample of subjects and stop signal intensities, both the magnitudes and variances of SSRTs and antagonist latencies were related; subjects with longer and more variable SSRTs tended to have longer and more variable antagonist muscle latencies.

As a final test of our hypothesis that antagonist recruitment latencies reflect the stop process, we examined how both the antagonist latencies and SSRTs changed within subjects across stop signal intensity. As shown above, although both the antagonist latencies and SSRTs increased for dim stop signals,

the magnitude of such increases varied substantially across subjects (see Table 1). Figure 5*C* shows the magnitudes of such intensity-dependent increases for the antagonist muscle latency as a function of that for the SSRT and demonstrated a positive correlation ($r = 0.83$, $P = 0.01$); thus, subjects with larger intensity-dependent changes in SSRTs tended to have larger increases in the antagonist latency. This result demonstrates that a manipulation that influences SSRT in a subject-dependent fashion exerts a similar influence on antagonist muscle latencies, providing further evidence for a tight relationship between these two measures.

Estimating the efferent delay for antagonist muscle recruitment during head-only errors. Despite the relationships between SSRTs and antagonist latencies, antagonist latencies are substantially longer than SSRTs (Table 1 and Fig. 5*A*). From a conceptual standpoint, this difference is not surprising; SSRTs estimate the duration of a central process, whereas antagonist latencies are measured in the motor periphery. If our hypothesis is true, antagonist latencies arise from the sum of the duration of central process (estimated by the SSRT) and the time it takes to convey the outcome of this process to the motor periphery (the efferent delay for antagonist muscle recruitment). We modeled the countermanding of eye-head gaze shifts and used this model to estimate the efferent delay during head-only errors. In particular, we were interested in 1) whether a single architecture can replicate the observed inhibition functions and the proportion of head-only errors across SSD and stop signal intensity and 2) whether the within-subject efferent delays

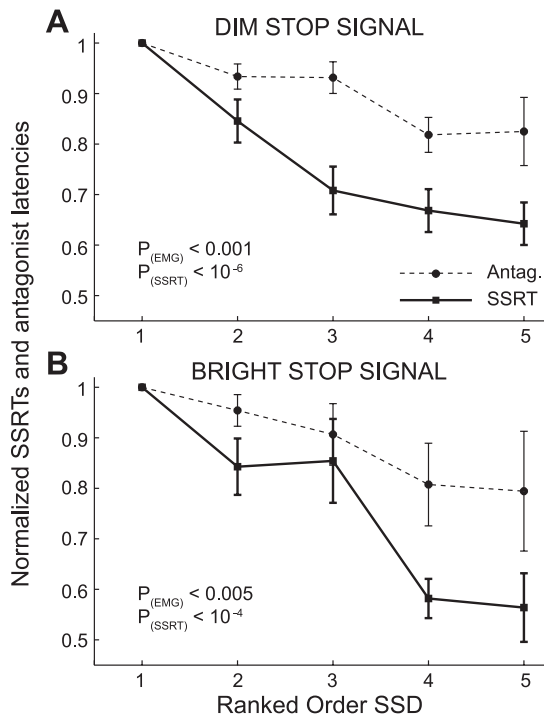


Fig. 4. Test of the race model. The race model predicted that measures related to the duration of movement cancellation decrease with increasing SSD, since less time is available for cancellation on such trials. For both dim (A) and bright (B) stop signals, both the SSRT (calculated at a given SSD via the integration method) and antagonist muscle latency followed this prediction. Both measures had to be normalized within a subject before being pooled across our sample; this was done by normalizing both measures to that observed at the shortest SSD and then rank ordering SSDs. EMG, electromyograph. Error bars show SEs.

estimated using this model are the same for bright and dim stop signals.

Our model is shown in Fig. 6A. In this model, which we have previously proposed (Corneil and Elsley 2005), behavior is determined by the outcome of a race between an “oculomotor” go and stop process. Head-only errors occur on successfully cancelled trials when the go process exceeded a lower “head” threshold before movement cancellation. This lower head threshold is consistent with neurophysiological observa-

tions (for a review, see Corneil 2011) and the absence of gaze errors without head movements. This model also incorporates ballistic intervals for gaze shifts and head movements that comprise the time between the go process exceeding the gaze or head threshold and the onset of the respective movement. These ballistic intervals themselves comprise the efferent delay between the commitment to move and agonist muscle activation and the biomechanical coupling time from muscle activation to movement onset.

To model behavior and estimate parameters, we adapted the maximum likelihood technique presented previously (Kornylo et al. 2003) for eye-head gaze shifts (see *model 2* in Corneil and Elsley 2005 for full details). Briefly, the go and stop processes are modeled to vary independently about a mean rate (μ) with a SD (σ). We first estimated rate parameters for the go process, based on the gaze and head reaction times from no-stop trials, candidate gaze and head ballistic intervals, and a candidate head threshold. Candidate ballistic intervals were subtracted from the observed reaction times to derive oculomotor reaction times, and oculomotor reaction times from head movements were further divided by the fraction of the head threshold. The oculomotor reaction times estimated from observed gaze and head reaction times were then combined into a reaction time distribution. Rates were determined from the reciprocal of this reaction time distribution, based on maximum likelihood estimates [i.e., (μ, σ) obtained via the “mle” function in Matlab].

Using the estimated rates of the go process and candidate head threshold, we then produced predicted inhibition functions for gaze and head movements by racing this go process against a candidate stop processes (this was done separately for dim and bright stop signals). Different SSDs delay the onset of the stop process relative to the go process. The predicted proportion of gaze shifts was the intersection between the cumulative rate distribution from the shifted stop process and the inverted cumulative rate distribution of the go process (Kornylo et al. 2003). The predicted proportion of head movements was obtained in a similar manner, after first shifting go rates to higher values to account for the lower head threshold.

We used a nonlinear minimization technique (“fminsearch” in Matlab) to determine the best parameters for the model that

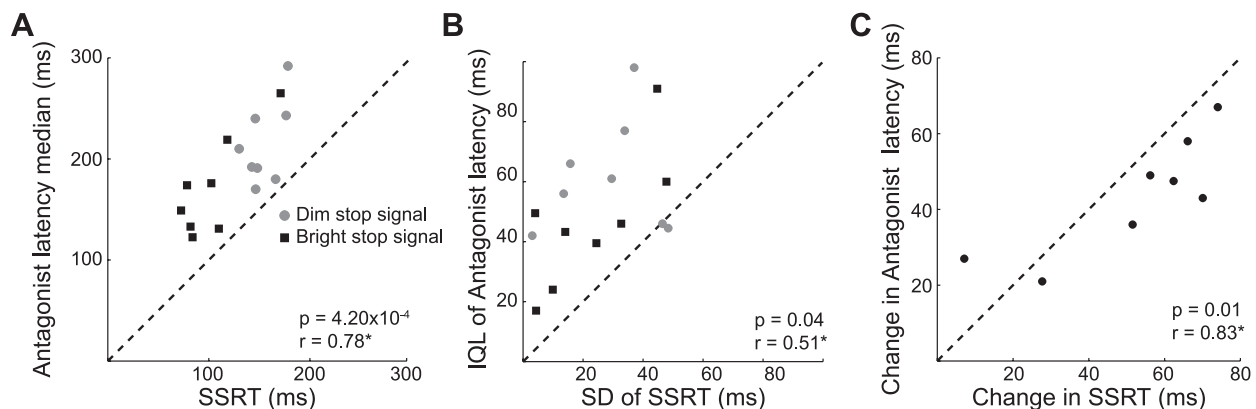


Fig. 5. Relationship between SSRT and antagonist muscle latency. A and B: across-subject comparison of the median antagonist muscle latency versus SSRT (A) or the interquartile spread of the antagonist muscle latency versus the SD of the SSRT estimated from the integration method (B). In both A and B, each square comes from a different subject, with each subject providing a point for both the dim and bright stop signal. C: within each subject, we calculated the change in the antagonist latency for bright versus dim stop signals and plotted this as a function of the change in the SSRT for bright versus dim stop signals. The diagonal dashed lines are lines of unity.

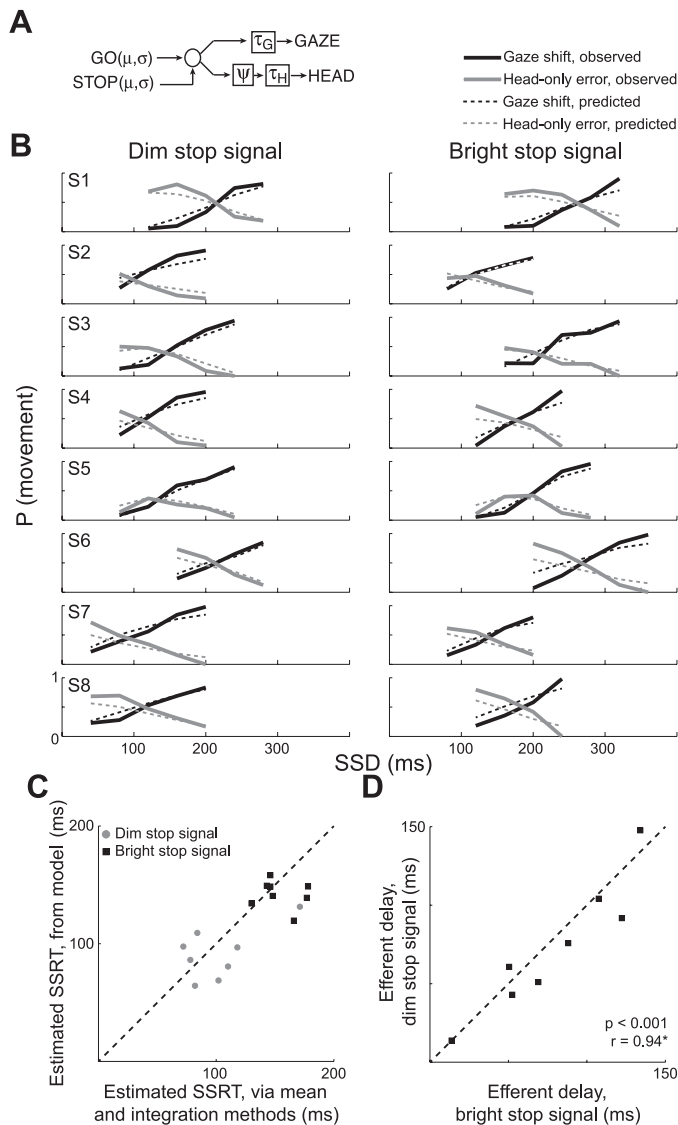


Fig. 6. Proposed race model architecture for the countermanding of eye-head gaze shifts and comparison with behavior. *A*: this model assumed that gaze shifts and head movements are controlled by a single race of “oculomotor” go and stop processes, each of which vary about mean rate μ with standard deviation σ . The gaze and head branches contain a ballistic interval (τ) that is not under inhibitory control. The head branch is activated at a different threshold (Ψ) than the gaze branch; if $\Psi < 1$, then the head branch has a lower threshold than the gaze branch. *B*: comparison of observed (solid lines) and predicted (dashed lines) inhibition functions for gaze shifts (black lines) and the proportion of head-only errors versus SSD for dim and bright stop signals. *C*: plot of the estimated SSRT obtained by averaging the mean and integration methods versus the SSRT estimated by the model. *D*: subject-by-subject plot of the efferent delay for antagonist muscle recruitment for dim versus bright stop signals.

minimize the difference between the expected and observed gaze and head inhibition functions for both bright and dim stop signals (for some subjects, we fitted only the middle portion of the inhibition function with a smooth transition, excluding short or long SSDs). Overall, the minimization processes involved the estimation of nine parameters [the rate parameters (μ, σ) for the go and stop processes for dim and bright stop signals, the ballistic intervals for gaze shifts and head movements, and the head threshold]. The parameters for the best fit are shown for all subjects in Table 2. The estimated ballistic

interval for gaze shifts averaged 33.3 ± 13.0 ms (range: 17.0–54.5 ms). The estimated ballistic interval for head movements averaged 112.6 ± 13.4 ms (range: 93.3–128.3 ms). The head threshold averaged 0.63 ± 0.10 (range: 0.50–0.80), meaning that the threshold for activating head movements was less than gaze shifts for all subjects.

Figure 6*B* shows a comparison of the observed inhibition function for gaze shifts and the proportion of head-only errors versus SSD to that predicted by our model. Across our sample and both stop signal intensities, the model shown in Fig. 6*A* did a reasonable job describing movement propensity across the range of SSDs. The SSRTs predicted by this model (i.e., the inverse of the mean rate of the stop process) also agreed well with those estimated by averaging the results of the mean and integration methods (paired *t*-test, $t_{15} = 1.8$, $P = 0.08$; Fig. 6*C* and Table 2).

Having derived the parameters for the model shown in Fig. 6*A*, we could then use them to estimate the duration of the efferent delay between the completion of the STOP process and the onset of antagonist muscle recruitment. Note that this measure differs from the ballistic interval for head movements, which runs from when the go process exceeds the head threshold to the onset of head motion. Because we estimated independent stop processes for bright and dim stop signals, we could derive independent estimates of the efferent delay for antagonist muscle recruitment within a given subject and determine whether they agree. To do this, at each SSD for both dim and bright stop signals, we raced independently selected go and (delayed) stop processes against each other (1,000 iterations) and determined the completion time for those stop processes where the go process had exceeded the head threshold, since this produces head-only errors. The estimated efferent delay was then calculated as the difference between these completion times and the observed antagonist latencies. For dim stop signals, the efferent delay for antagonist muscle recruitment was 73.4 ± 41.3 ms (range: 13.7–147.7 ms; Table 2). For bright stop signals, the efferent delay for antagonist muscle recruitment was 79.7 ± 40.8 ms (range: 13.8–134.0 ms; Table 2). As shown in Fig. 6*D*, these measures were positively correlated ($r = 0.94$, $P < 0.001$), meaning that subjects with low efferent delays with bright stop signals had a similarly low efferent delay for dim stop signals. Moreover, the efferent delays for bright and dim stop signals did not differ significantly (paired *t*-test, $t_7 = 1.2$, $P = 0.27$).

A number of aspects of this modeling effort deserve emphasis. First, the estimation of the nine parameters for the model was done exclusively based on the reaction times of gaze shifts and head movements and the gaze and head inhibition functions; the timing of neck muscle recruitment did not factor into this estimation in any way. Second, the estimation of the rates for the stop processes for dim and bright stop signals were independent. Finally, although there was substantial variation in the estimations of the efferent delay for antagonist muscle recruitment across our sample, these estimations fell within a physiological range and never obtained negative values. Overall, this modeling effort reinforces the plausibility that antagonist muscle recruitment arises from the completion of the stop process on a subset of successfully cancelled trials.

Table 2. Maximum likelihood estimates for parameters of the race model and the behavior predicted from this model

	Estimated parameters						Predicted behavior			
	Go rate	Stop rate (dim)	Stop rate (bright)	Ballistic interval (gaze), ms	Ballistic interval (head), ms	Threshold of the head pathway	SSRT (dim), ms	SSRT (bright), ms	Effective delay (dim), ms	Effective delay (bright), ms
Subject 1	2.92 ± 0.62	7.44 ± 0.22	11.59 ± 1.12	17	116	0.63	134	86	76	88
Subject 2	3.98 ± 1.22	6.72 ± 1.32	7.60 ± 0.41	55	124	0.56	148	131	148	134
Subject 3	3.50 ± 0.69	7.19 ± 0.16	14.53 ± 1.47	48	125	0.75	139	68	104	108
Subject 4	4.12 ± 1.02	7.11 ± 0.36	15.54 ± 1.95	26	102	0.66	140	64	51	69
Subject 5	3.58 ± 0.61	8.35 ± 0.53	12.39 ± 0.82	33	98	0.80	119	80	61	50
Subject 6	2.98 ± 0.72	6.69 ± 0.18	10.25 ± 1.06	28	128	0.59	149	97	43	52
Subject 7	4.41 ± 1.40	6.73 ± 0.30	10.31 ± 0.61	38	93	0.59	148	96	92	122
Subject 8	3.80 ± 0.93	6.31 ± 0.94	9.15 ± 0.76	21	115	0.50	158	109	14	14
Mean	3.66 ± 0.52	7.07 ± 0.62	11.42 ± 2.67	33 ± 13	113 ± 13	0.63 ± 0.10	142 ± 12	92 ± 22	73 ± 41	80 ± 41

Rate parameters have units of Hz and are given as means ± SD. The threshold of the head pathway is a fraction and has no units.

DISCUSSION

The countermanding, or stop signal, paradigm remains a mainstay for research into movement cancellation. This task provides a means to estimate how long it takes to cancel a movement (the SSRT) based on the theoretical framework of the race model (Logan and Cowan 1984). This advantage also presents a problem: movement cancellation implies the absence of something that can be observed directly and accordingly must be estimated indirectly. A number of methods for estimating movement cancellation have been proposed (Colnius 1990; Kornyló et al. 2003; Logan and Cowan 1984; Walton and Gandhi 2006). When the head is free to move, a subset of successfully cancelled gaze shifts are accompanied by small head movements to the target (Corneil and Elsley 2005). Neck muscle recruitment during such head-only trials both launches and then actively brakes erroneous head motion, and we have proposed the recruitment of the antagonist muscle, in particular, is a direct manifestation of oculomotor cancellation (Goonetilleke et al. 2010). As shown here, antagonist muscle recruitment latencies correlated well with traditional measures of movement cancellation (the SSRT) both within and across subjects when the intensity of a visual stop signal was varied. The opportunity to directly measure movement cancellation on a within-trial basis also enables observation of the distribution and variance of the antagonist muscle latency. As shown in Fig. 3, we observed substantial heterogeneity in these measures across our sample of subjects; such observations demonstrate one example of the utility of measurements of neck muscle activity, as they would not have been possible via traditional estimates of SSRTs.

A bottom-up perspective on oculomotor control for large gaze shifts. Key to our hypothesis is that head movements are initiated before the commitment for a large gaze shift. Head-only errors arise because of such staggered initiation, providing instances wherein head movements are both started and stopped even though gaze remains stable by virtue of compensatory vestibular reflexes (Corneil and Elsley 2005). Our previous research on the countermanding of eye-head gaze shifts has shown that such sequences are common, being expressed on ~20% of all stop signal trials (Corneil and Elsley 2005; Goonetilleke et al. 2010). The proportion of head-only errors was higher in the current study (~35%), likely because the staircasing procedure for adaptively determining SSDs prefer-

entially samples the intermediate SSDs at which head-only errors are more common (Fig. 2).

Far more is known about the oculomotor mechanisms for starting versus stopping head movements during large eye-head gaze shifts. At the 60° eccentricity used here, neck EMG began far in advance of the gaze shift (e.g., Fig. 2). Other behavioral studies have suggested that eye-in-head motion must be made in light of anticipated head motion (Tweed et al. 1998), emphasizing a bottom-up perspective to recruitment that reflects the nested nature of the eye within a mobile head. Neurophysiological evidence has demonstrated that levels of oculomotor activity insufficient to recruit saccadic gaze shifts can produce neck muscle recruitment and head-only movements that are counteracted by compensatory eye movements, similar to the head-only errors observed here in humans (Corneil et al. 2010; Corneil et al. 2002b; Rezvani and Corneil 2008). In humans, transcranial magnetic stimulation of the frontal eye fields (FEF) can evoke neck muscle responses without saccades (Goonetilleke et al. 2011). We and others have argued that the brainstem mechanisms that tightly govern saccadic gaze shifts do not exert a similar level of inhibition on premotor head circuits (for a review, see Corneil 2011).

The neural mechanisms recruited to actively arrest head motion (i.e., those recruiting the antagonist neck muscles) remain unknown. Neural correlates of oculomotor cancellation have been reported in the superior colliculus (SC) and FEF, consisting of a decrease of firing in saccade-related neurons and a simultaneous increase in the activity of fixation-related neurons (Hanes et al. 1998; Paré and Hanes 2003). We view it as unlikely that either of these mechanisms could directly recruit antagonist neck muscles, even if they may be occurring contemporaneously. Electrical stimulation in the vicinity of fixation-related neurons (i.e., in the rostral SC or lateral FEF) is associated with little or no neck muscle recruitment, and when such recruitment is evoked, it appears on the agonist rather than antagonist muscles (Corneil et al. 2002a; Elsley et al. 2007). The direct contribution of other oculomotor areas also appears unlikely. Stimulation of omni-pause neurons (OPNs) also does not consistently decelerate on-going head motion (Gandhi and Sparks 2007). Signals recorded in the supplementary eye fields (SEF) arise too late to be directly signal movement cancellation (Stuphorn et al. 2010), and evidence favors a role for this area in the adaptive control of

behavior rather than strict inhibition (Stuphorn and Schall 2006). Furthermore, SEF stimulation, like stimulation of the FEF and SC, also recruits a contralateral head-turning synergy that scales with gaze shift magnitude and never recruited anything resembling movement cancellation (Chapman et al. 2012).

Although further work is needed, we suggest that the mechanisms that actively brake on-going head motion reside outside of well-studied oculomotor areas. Numerous results have suggested that a frontobasal ganglia circuit, which includes the inferior frontal cortex and dorsomedial frontal cortex and subthalamic nucleus, may comprise the neural circuit for stopping manual movements (for a review, see Aron 2011). Electrical stimulation in areas of the precentral cortex can evoke ipsilaterally directed head movements during defensive responses that differ from the contralaterally directed head movements evoked from the FEF and SEF (Boulanger et al. 2009; Graziano et al. 2002); such a circuit may provide a substrate for arresting the head in midflight. Neural substrates for the cancellation of limb movements have also been found in the supplementary and presupplementary motor areas (Chen et al. 2010; Scangos and Stuphorn 2010) and to a greater degree in the dorsal premotor cortex (Mirabella et al. 2011). The study by Mirabella and colleagues (2011) in particular describes two classes of neurons whose activity either decreased or increased before the SSRT; the latter may relate the activation of antagonist muscles, although this remains to be investigated.

While correlates of limb cancellation have been demonstrated in the frontal cortex, the extrapolation of these findings to our results must be done cautiously. The cancellation of eye and hand movements can be uncoupled to a much higher degree than eye-head gaze shifts (Boucher et al. 2007; Logan and Irwin 2000), suggesting that the cancellation of eye and hand movements are not obligatorily coupled. In contrast, although the control of an inertial structure like the head may resemble that of the limb in some aspects, orienting head movements are an essential component of large gaze shifts controlled by the oculomotor system. Further, even though subjects were not given any specific instruction on how to control the head, they never generated the eye-only errors, which would have been predicted if the control of gaze and head orienting was independent. We favor the interpretation that cancellation of an oculomotor program is a single process than can manifest differentially in the cephalomotor periphery depending on whether a head movement has been initiated or not. Although this is speculation, it is consistent with the strong relationships we observed between antagonist muscle latencies and the SSRT across a variety of experimental manipulations and analyses.

Head-only movements as a partial response in the oculomotor system. The strict segregation between cancelled and non-cancelled trials is perhaps best exemplified in the saccadic system when studied with the head restrained. Saccades are easily detected, and there is no evidence for ocular motility or extraocular muscle recruitment during cancelled trials (Godlove et al. 2011). Reports have differed on whether small- to medium-sized (less than $\sim 10\text{--}15^\circ$ in amplitude) noncancelled saccades can be hypometric or not (Colonius et al. 2001; Godlove et al. 2011; Hanes and Schall 1995; Ozyurt et al. 2003; Paré and Hanes 2003), although large noncancelled gaze shifts can certainly be arrested in midflight (Corneil and Elsley

2005; Goonetilleke et al. 2010). On the whole, it appears that the processes preceding saccade initiation have a ballistic component: once initiated, a rapid saccade will be launched, even if the later part of the trajectory can be modified. In contrast, the classification of response types in limb or hand movements is not as straightforward. Many aspects of movement kinematics, muscle recruitment, or force profiles suggest that inhibition can be expressed at essentially any stage along the continuum from motor preparation through execution (De Jong et al. 1990; Ko et al. 2012; McGarry and Franks 1997, 2003; McGarry et al. 2000; Osman et al. 1986; Scangos and Stuphorn 2010; van Boxtel et al. 2001). In the skeletomotor system, therefore, the “point of no return” may be therefore a conceptual notion without any underlying neural substrate. In contrast in the oculomotor system, we speculate that the point of no return is embodied in the brain stem circuit via the potent inhibition of OPNs on the saccadic burst generator (Scudder et al. 2002), highlighting a fundamental difference between the oculomotor and skeletomotor systems (Godlove et al. 2011). According to our proposition that head orienting escapes OPN inhibition (effectively allowing a lower threshold for initiating orienting head movements, as conceptualized in Fig. 6A), head-only errors can therefore be interpreted as a partial response characteristic of the head-unrestrained oculomotor system. What is particularly unique about this type of partial response is that it is occurring within a nested motor system: given that subjects successfully countermanded the gaze shift on head-only errors, the initiation and active cancellation of head movements occurred prior to the point of no return of the gaze axis.

If one accepts that the cancellation of head-only movements arises from the oculomotor stop process, then every head-only movement provides an opportunity to empirically assess the stop process. As recently noted (Godlove et al. 2011), partial responses in the oculomotor system could be a powerful tool for understanding motor control in saccadic tasks. Although partial responses do not appear to be present in a proxy of extraocular EMG activation via electroencephalography (Godlove et al. 2011), we suggest that head-only movements, and more precisely the underlying profile of neck muscle recruitment, provides exactly this type of measure. Since such activity is recorded in the motor periphery, one must acknowledge the efferent delay, which we suggest is the main contributor to the difference between SSRTs and antagonist muscle latencies ($\sim 60\text{--}70$ ms in this study). The variability in the estimated efferent delay across subjects may arise at least in part from the functional or histochemical heterogeneity of SPL motor units recorded in a given subject. We did not tightly constrain the location of recording within SPL, and there is some evidence for a differential distribution of muscle fiber type throughout a number of dorsal neck muscles, including the SPL (Richmond et al. 1999, 2001). The morphometry and functional recruitment of the SPL can also vary substantially across different humans (Blouin et al. 2007; Kamibayashi and Richmond 1998; Keshner et al. 1989); hence, it is possible that recordings accessed different muscle compartments.

Potential utility of antagonist muscle latencies and issues limiting widespread use. We have previously speculated on the potential utility of antagonist muscle latencies for neurophysiological research (Goonetilleke et al. 2010); neck EMG measures could provide a precise boundary condition for determin-

ing whether or not a given neurophysiological signal is directly involved in movement cancellation. Neck EMG measures could prove useful in basic or applied research domains. In particular, the ability to directly measure the variance and assess the distribution of a proxy of oculomotor stopping may provide new metrics suitable for developmental studies or comparisons across drug therapies. Applied studies of developmental or clinical populations frequently obtain >250 trials to estimate SSRT (e.g., Armstrong and Munoz 2003; Aron et al. 2003; Enticott et al. 2008; Schachar and Logan 1990); given that head-only movements occur on ~10% of all trials total, the yield of head-only movements should be sufficiently high to permit assessment of variance and distributions.

Currently, a few issues limit more widespread use of neck EMG measurements studies of oculomotor cancellation. First, neck muscle motoneurons are the final common path of numerous descending systems, and this diversity has to be recognized for appropriate experimental design and interpretation. As an example, our first iteration of this experiment involved a manipulation of stop signal modality, since SSRTs are longer with auditory versus visual stop signals (Cabel et al. 2000; Stevenson et al. 2009). However, our preliminary data showed far shorter antagonist muscle latencies to auditory stop signals than expected based on the changes in SSRTs. In retrospect, we realized that the antagonist neck muscle latencies were confounded by the acoustic startle reflex, which can interact with voluntary motor programs (Siegmund et al. 2001; Valls-Sole et al. 1997).

Another issue is the use of intramuscular fine wire needle electrodes. Our choice of this recording technique stemmed from concerns that surface EMG recordings of neck muscles involved in head turning can be unreliable and susceptible to cross-talk from other neck muscles (Mayoux-Benhamou et al. 1995). While intramuscular recording techniques were an appropriate choice for the goals of this study and our sample of healthy volunteers, the inherent invasiveness would likely limit the size of the sample in clinical and developmental studies. In this regard, we note that the development of high-density surface EMG recordings may provide a fruitful alternative to intramuscular recording techniques (Drost et al. 2006; Zwarts and Stegeman 2003). Whether such recordings can reliably assess antagonist recruitment latencies remains to be determined.

In conclusion, neck muscle recruitment during a subset of successfully cancelled gaze shifts appears to provide a novel marker of the cancellation of an oculomotor program. What is particularly unique about this measure is its availability within a single trial. This may allow for direct assessment of the variability and distribution of stopping latencies at an unprecedented resolution and could be particularly relevant in clinical populations.

ACKNOWLEDGMENTS

The authors thank Dr. D. Irwin and Dr. P. Medendorp for comments on an earlier version of the manuscript.

GRANTS

This work was supported by Canadian Institutes of Health Research Operating Grant MOP 93796 and a Discovery Grant from the Natural Sciences and Engineering Research Council of Canada.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: S.C.G., J.P.W., and B.D.C. conception and design of research; S.C.G. and J.P.W. performed experiments; S.C.G. and B.D.C. analyzed data; S.C.G., J.P.W., and B.D.C. interpreted results of experiments; S.C.G., J.P.W., and B.D.C. prepared figures; S.C.G. and B.D.C. drafted manuscript; S.C.G., J.P.W., and B.D.C. edited and revised manuscript; S.C.G., J.P.W., and B.D.C. approved final version of manuscript.

REFERENCES

- Armstrong IT, Munoz DP.** Inhibitory control of eye movements during oculomotor countermanning in adults with attention-deficit hyperactivity disorder. *Exp Brain Res* 152: 444–452, 2003.
- Aron AR.** From reactive to proactive and selective control: developing a richer model for stopping inappropriate responses. *Biol Psychiatry* 69: e55–68, 2011.
- Aron AR, Fletcher PC, Bullmore ET, Sahakian BJ, Robbins TW.** Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nat Neurosci* 6: 115–116, 2003.
- Blouin JS, Siegmund GP, Carpenter MG, Inglis JT.** Neural control of superficial and deep neck muscles in humans. *J Neurophysiol* 98: 920–928, 2007.
- Boucher L, Stuphorn V, Logan GD, Schall JD, Palmeri TJ.** Stopping eye and hand movements: are the processes independent? *Percept Psychophys* 69: 785–801, 2007.
- Boulanger M, Bergeron A, Guitton D.** Ipsilateral head and centring eye movements evoked from monkey premotor cortex. *Neuroreport* 20: 669–673, 2009.
- Cabel DW, Armstrong IT, Reingold E, Munoz DP.** Control of saccade initiation in a countermanning task using visual and auditory stop signals. *Exp Brain Res* 133: 431–441, 2000.
- Chapman BB, Pace MA, Cushing SL, Corneil BD.** Recruitment of a contralateral head turning synergy by stimulation of monkey supplementary eye fields. *J Neurophysiol* 107: 1694–1710, 2012.
- Chen X, Scangos KW, Stuphorn V.** Supplementary motor area exerts proactive and reactive control of arm movements. *J Neurosci* 30: 14657–14675, 2010.
- Colonius H.** A note on the stop-signal paradigm, or how to observe the unobservable. *Psychol Rev* 97: 309–312, 1990.
- Colonius H, Ozyurt J, Arndt PA.** Countermanning saccades with auditory stop signals: testing the race model. *Vision Res* 41: 1951–1968, 2001.
- Corneil BD.** Eye-head gaze shifts. In: *The Oxford Handbook of Eye Movements*, edited by Liversedge SP, Gilchrist ID, Everling S. Oxford: Oxford Univ. Press, 2011, p. 303–322.
- Corneil BD, Elsley JK.** Countermanning eye-head gaze shifts in humans: marching orders are delivered to the head first. *J Neurophysiol* 94: 883–895, 2005.
- Corneil BD, Elsley JK, Nagy B, Cushing SL.** Motor output evoked by subsaccadic stimulation of primate frontal eye fields. *Proc Natl Acad Sci USA* 107: 6070–6075, 2010.
- Corneil BD, Olivier E, Munoz DP.** Neck muscle responses to stimulation of monkey superior colliculus. I. Topography and manipulation of stimulation parameters. *J Neurophysiol* 88: 1980–1999, 2002a.
- Corneil BD, Olivier E, Munoz DP.** Neck muscle responses to stimulation of monkey superior colliculus. II. Gaze shift initiation and volitional head movements. *J Neurophysiol* 88: 2000–2018, 2002b.
- De Jong R, Coles MG, Logan GD, Gratton G.** In search of the point of no return: the control of response processes. *J Exp Psychol Hum Percept Perform* 16: 164–182, 1990.
- Drost G, Stegeman DF, van Engelen BG, Zwarts MJ.** Clinical applications of high-density surface EMG: a systematic review. *J Electromyogr Kinesiol* 16: 586–602, 2006.
- Elsley JK, Nagy B, Cushing SL, Corneil BD.** Widespread presaccadic recruitment of neck muscles by stimulation of the primate frontal eye fields. *J Neurophysiol* 98: 1333–1354, 2007.
- Enticott PG, Oglloff JR, Bradshaw JL.** Response inhibition and impulsivity in schizophrenia. *Psychiatry Res* 157: 251–254, 2008.
- Floden D, Stuss DT.** Inhibitory control is slowed in patients with right superior medial frontal damage. *J Cogn Neurosci* 18: 1843–1849, 2006.

- Gandhi NJ, Sparks DL.** Dissociation of eye and head components of gaze shifts by stimulation of the omnipause neuron region. *J Neurophysiol* 98: 360–373, 2007.
- Godlove DC, Garr AK, Woodman GF, Schall JD.** Measurement of the extraocular spike potential during saccade countermanding. *J Neurophysiol* 106: 104–114, 2011.
- 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.
- Goonetilleke SC, Gribble PL, Mirsattari SM, Doherty TJ, Corneil BD.** Neck muscle responses evoked by transcranial magnetic stimulation of the human frontal eye fields. *Eur J Neurosci* 33: 2155–2167, 2011.
- Graziano MS, Taylor CS, Moore T.** Complex movements evoked by microstimulation of precentral cortex. *Neuron* 34: 841–851, 2002.
- Guittan D, Volle M.** Gaze control in humans: eye-head coordination during orienting movements to targets within and beyond the oculomotor range. *J Neurophysiol* 58: 427–459, 1987.
- Hanes DP, Carpenter RH.** Countermanding saccades in humans. *Vision Res* 39: 2777–2791, 1999.
- Hanes DP, Patterson WF, Schall JD.** Role of frontal eye fields in countermanding saccades: visual, movement, and fixation activity. *J Neurophysiol* 79: 817–834, 1998.
- Hanes DP, Schall JD.** Countermanding saccades in macaque. *Vis Neurosci* 12: 929–937, 1995.
- Kamibayashi LK, Richmond FJ.** Morphometry of human neck muscles. *Spine* 23: 1314–1323, 1998.
- Keshner EA, Campbell D, Katz RT, Peterson BW.** Neck muscle activation patterns in humans during isometric head stabilization. *Exp Brain Res* 75: 335–344, 1989.
- Ko YT, Alsford T, Miller J.** Inhibitory effects on response force in the stop-signal paradigm. *J Exp Psychol Hum Percept Perform* 38: 465–477, 2012.
- Kornylo K, Dill N, Saenz M, Krauzlis RJ.** Cancelling of pursuit and saccadic eye movements in humans and monkeys. *J Neurophysiol* 89: 2984–2999, 2003.
- Li CS, Huang C, Constable RT, Sinha R.** Imaging response inhibition in a stop-signal task: neural correlates independent of signal monitoring and post-response processing. *J Neurosci* 26: 186–192, 2006.
- Logan GD, Cowan WB.** On the ability to inhibit thought and action: a theory of an act of control. *Psychol Rev* 91: 295–327, 1984.
- Logan GD, Irwin DE.** Don't look! Don't touch! Inhibitory control of eye and hand movements. *Psychon Bull Rev* 7: 107–112, 2000.
- Mayoux-Benhamou MA, Revel M, Vallee C.** Surface electrodes are not appropriate to record selective myoelectric activity of splenius capitis muscle in humans. *Exp Brain Res* 105: 432–438, 1995.
- McGarry T, Franks IM.** A horse race between independent processes: evidence for a phantom point of no return in preparation of a speeded motor response. *J Exp Psychol Hum Percept Perform* 23: 1533–1542, 1997.
- McGarry T, Franks IM.** On the nature of stopping an earlier intended voluntary action. *Motor Control* 7: 155–198, 2003.
- McGarry T, Inglis JT, Franks IM.** Against a final ballistic process in the control of voluntary action: evidence using the Hoffmann reflex. *Motor Control* 4: 469–485, 2000.
- Mirabella G, Pani P, Ferraina S.** Neural correlates of cognitive control of reaching movements in the dorsal premotor cortex of rhesus monkeys. *J Neurophysiol* 106: 1454–1466, 2011.
- Morein-Zamir S, Kingstone A.** Fixation offset and stop signal intensity effects on saccadic countermanding: a crossmodal investigation. *Exp Brain Res* 175: 453–462, 2006.
- Osman A, Kornblum S, Meyer DE.** The point of no return in choice reaction time: controlled and ballistic stages of response preparation. *J Exp Psychol Hum Percept Perform* 12: 243–258, 1986.
- Ozyurt J, Colonius H, Arndt PA.** Countermanding saccades: evidence against independent processing of go and stop signals. *Percept Psychophys* 65: 420–428, 2003.
- Paré M, Hanes DP.** Controlled movement processing: superior colliculus activity associated with countermanded saccades. *J Neurosci* 23: 6480–6489, 2003.
- Rezvani S, Corneil BD.** Recruitment of a head-turning synergy by low-frequency activity in the primate superior colliculus. *J Neurophysiol* 100: 397–411, 2008.
- Richmond FJ, Singh K, Corneil BD.** Marked non-uniformity of fiber-type composition in the primate suboccipital muscle obliquus capitis inferior. *Exp Brain Res* 125: 14–18, 1999.
- Richmond FJ, Singh K, Corneil BD.** Neck muscles in the rhesus monkey. I. Muscle morphometry and histochemistry. *J Neurophysiol* 86: 1717–1728, 2001.
- Scangos KW, Stuphorn V.** Medial frontal cortex motivates but does not control movement initiation in the countermanding task. *J Neurosci* 30: 1968–1982, 2010.
- Schachar R, Logan GD.** Impulsivity and inhibitory control in normal developmental and childhood psychopathology. *Dev Psychol* 26: 710–720, 1990.
- Schachar RJ, Chen S, Logan GD, Ornstein TJ, Crosbie J, Ickowicz A, Pakulak A.** Evidence for an error monitoring deficit in attention deficit hyperactivity disorder. *J Abnorm Child Psychol* 32: 285–293, 2004.
- Scudder CA, Kaneko CS, Fuchs AF.** The brainstem burst generator for saccadic eye movements: a modern synthesis. *Exp Brain Res* 142: 439–462, 2002.
- Siegmund GP, Inglis JT, Sanderson DJ.** Startle response of human neck muscles sculpted by readiness to perform ballistic head movements. *J Physiol* 535: 289–300, 2001.
- Stahl JS.** Amplitude of human head movements associated with horizontal saccades. *Exp Brain Res* 126: 41–54, 1999.
- Stevenson SA, Elsley JK, Corneil BD.** A “gap effect” on stop signal reaction times in a human saccadic countermanding task. *J Neurophysiol* 101: 580–590, 2009.
- Stuphorn V, Brown JW, Schall JD.** Role of supplementary eye field in saccade initiation: executive, not direct, control. *J Neurophysiol* 103: 801–816, 2010.
- Stuphorn V, Schall JD.** Executive control of countermanding saccades by the supplementary eye field. *Nat Neurosci* 9: 925–931, 2006.
- Tweed D, Haslwanter T, Fetter M.** Optimizing gaze control in three dimensions. *Science* 281: 1363–1366, 1998.
- Valls-Sole J, Valldeoriola F, Tolosa E, Nobbe F.** Habituation of the auditory startle reaction is reduced during preparation for execution of a motor task in normal human subjects. *Brain Res* 751: 155–159, 1997.
- van Boxtel GJ, van der Molen MW, Jennings JR, Brunia CH.** A psychophysiological analysis of inhibitory motor control in the stop-signal paradigm. *Biol Psychol* 58: 229–262, 2001.
- Verbruggen F, Logan GD.** Response inhibition in the stop-signal paradigm. *Trends Cogn Sci* 12: 418–424, 2008.
- Walton MM, Gandhi NJ.** Behavioral evaluation of movement cancellation. *J Neurophysiol* 96: 2011–2024, 2006.
- Zwarts MJ, Stegeman DF.** Multichannel surface EMG: basic aspects and clinical utility. *Muscle Nerve* 28: 1–17, 2003.