A within trial measure of the stop signal reaction time in a head-unrestrained oculomotor countermanding task

Abbreviated title: An empirical measure of the stop signal reaction time

Samanthi C. Goonetilleke¹,³, Timothy J. Doherty⁴, Brian D. Corneil¹,²,³

¹CIHR Group in Action and Perception, ²Departments of Physiology & Pharmacology
³Psychology, ⁴Clinical Neurological Sciences
University of Western Ontario,
London, Ontario, Canada N6A 5C1

Correspondence should be addressed to:
Brian D. Corneil, Centre for Brain and Mind,
Robarts Research Institute, London, Ontario, Canada N6A 5K8.
(519) 663-5777, ext 24132. FAX: (519) 931-5233. E-mail: bcorneil@uwo.ca

Number of figures: 9
Number of Tables: 2
Number of pages: 37
Words in abstract: 294

Keywords: Countermanding, eye-head co-ordination, neck muscles, stop signal reaction time (SSRT)
Abstract

The countermanding (or stop-signal) task, which requires the cancellation of an impending response upon the infrequent presentation of a stop signal, enables study of the contextual control of movement generation and suppression. Here we present a novel and empirical measure of the time needed to cancel an impending gaze shift by recording neck muscle activity during a head-unrestrained oculomotor countermanding paradigm. On a subset of STOP signal trials, subjects generated small head movements towards a target even though gaze remained stable due to a compensatory vestibular ocular reflex (VOR). On such trials, we observed a burst of antagonist neck muscle activity during the small head-only error. Such antagonist neck muscle activity served as an active braking pulse, as its magnitude scaled with the kinematics of the head-only error. This activity was selective for trials in which the head was arrested in mid-flight, and did not appear on trials without a stop signal, on non-cancelled STOP signal trials when the gaze shift was completed, or on STOP signal trials without head motion. Importantly, the timing of this antagonist activity related best to the onset of the stop signal (lagging it by ~180 ms), and strongly correlated with behavioural estimates of the time needed to cancel a movement (the stop-signal reaction time). These results are consistent with the notion that such selective antagonist neck muscle activity arises as a peripheral expression of the oculomotor STOP process that successfully cancelled the gaze shift. Studying movement cancellation within nested systems like the head-unrestrained gaze shifting system offers a unique opportunity for investigating underlying neural mechanisms, as the overall goal (i.e., to cancel a gaze shift) can be achieved despite motion of other components; on such individual trials, the oculomotor STOP process is expressed as an active braking pulse.
Introduction

To interact in one’s environment there are often instances where it is necessary to cancel an impending movement that is no longer required. Movement cancellation has been studied experimentally using a countermanding paradigm that requires subjects try to withhold a movement upon the intermittent presentation of a stop signal (termed STOP signal trials). Performance on such trials is conceptualised to be determined by a race between two processes: a GO process initiated upon target presentation, and a STOP process initiated upon stop signal presentation. Whether subjects successfully cancel the movement or not is determined by which process reaches threshold first (Fig. 1A).

The countermanding paradigm provides a framework for studying inhibitory control. Although no observable behaviour 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 pharmaceuticals 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 (Kornylo et al. 2003), or the density functions of the RTs from CONTROL and non-cancelled STOP signal trials and the inhibition function (Colonius, 1990; see Band, Molen and Logan (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 of absence of saccades accompanying blinks. Although statistically- or behaviourally-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 useful for neurophysiological 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 cancelling saccades. We have recently investigated how humans countermand eye-head gaze shifts, and in addition to either successfully cancelling 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 (Corneil 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 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 = 27 years) 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 (Corneil and Elsley 2005). Briefly, this task required subjects to look to peripheral visual targets on most trials (term...
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) upon 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 rightward or leftward target. The inclusion of this 200 ms gap expedites both saccadic reaction times (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 amongst six different intervals spanning a 200 ms range. A fixed set of SSDs were used customised 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 pseudo-randomized 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 familiarise themselves to the task. Subjects were instructed to look as quickly and as accurately as possible to the peripheral target with whatever combination of eye and head movement was natural for them, but to try and maintain fixation on the central FP upon 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 first calibrated and then the reference markers were assigned to the centre of mass of the head using a centroid system. The location of the markers in space was monitored by four Digital Realtime Cameras (Motion Analysis, Santa Rosa, CA). Horizontal eye movements were measured using bi-temporal DC electrooculography (EOG) with electrodes placed at the outer canthi 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 C4/C5 vertebrae was detected bilaterally using intramuscular fine-wire “needle” electrodes (30mm, 27ga 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 utilised 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 Inc, 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 subject’s left forearm and was utilized as the ground electrode. EMG data were recorded with a Myopac Jr (Run Technologies, Laguna Hills, CA; 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 kHz 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 onset and offset of EMG activity was identified by another algorithm based on when the activity exceeded baseline (average EMG activity in the 500ms preceding target onset) by three standard deviations for 20 consecutive 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 > 800ms, respectively) (Corneil 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.

RESULTS

As reported previously (Corneil and Elsley 2005), subjects produced three types of movement sequences on STOP signal trials (see leftmost column in Fig. 3B-D 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 non-cancelled 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 axis 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 Corneil and Elsley (2005), head movement inhibition functions are shifted to the left when compared to gaze movement inhibition functions, which 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 Corneil 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 (Corneil and Elsley 2005; see the schematics in Fig. 5 for the proposed activation of the GO and STOP processes for each of the three main movement sequences observed during STOP trials). To analyse 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 non-cancelled movements, intermediate SSDs 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 non-cancelled 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 non-cancelled gaze shifts in the analysis below since, 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 brainstem events that are not present on head-only trials, including the temporary attenuation of the vestibulo-ocular reflex 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 over 100 ms long. This prolonged duration provides an interval in which subjects can cancel an ongoing 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.

The frequency of these movement sequences is shown in Table 1 on a per-subject basis. Overall, gaze shifts were cancelled on approximately 50% of all *STOP* signal trials, with head-only errors occurring on approximately 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 below 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 which 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 non-cancelled 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 antagonist muscle after the non-cancelled eye-head gaze shift returned the head back to the central FP.

The profiles of neck muscle activity during head-only errors stand in contrast to those observed during either CONTROL trials, or on completed or non-cancelled STOP signal trials (Fig. 3D). Head-only errors, as expected, were preceded by activation of the agonist muscle. However, such agonist activation was followed very quickly by robust activation of the antagonist muscle, which occurred during the erroneous head only movement to
the target. Such antagonist muscle recruitment during an erroneous movement was also
observed on truncated non-cancelled STOP signal trials, but we will focus on head-only
ers for the reasons described above. We stress that these patterns of muscle activity
were consistent across our sample; we never observed antagonist muscle recruitment on
cancelled STOP signal trials, during completed non-cancelled STOP signal trials, or
during CONTROL trials. Antagonist muscle recruitment during a head movement only
occurred when head motion was arrested in mid-flight. We also never observed
simultaneous activation of the agonist and antagonist muscle on any of the movement
profiles observed on STOP trials in any subject.

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 (normalised 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 out 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 of 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.

_______________________________________________________________________

INSERT FIGURE 6 HERE

________________________________________________________________________
The 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 (the circle denotes target onset, the vertical line denotes stop signal onset, and the square denotes 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 to 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 centred ~300 ms.

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 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 stop signal rather than 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 two head-only errors at a given SSD), and analysed 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 (one-
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 increased SSD (Fig.
8B), although this trend did not reach significance (one-way 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
analyse 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; \( p = 0.17 \)).

A comparison of antagonist muscle latencies to stop signal reaction times

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 stop signal reaction times (SSRT). The STOP
process is initiated upon 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). Since 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).

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 = 0.93); this result demonstrates that 87% 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 muscles 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 to other methods that estimate the variance of the STOP process. We used the maximum likelihood model described in Corneil and Elsley (2005) since 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 = 1×10^-3).
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 process, and that the durations of these stop processes may vary independently of one another. Based on this, cancelling a gaze shift following a head-only movement (from which the antagonist muscle latencies are derived) may involve a peripheral stopping process, whereas fully cancelling any motion of the gaze or the head may involve a different central stopping process. If this were 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 were 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 muscle 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 (r = 0.64, p = 2 × 10⁻³), but was still ~ 40 ms longer, possibly reflecting the efferent lag in the head movement system.

DISCUSSION

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 STOP process, and displaying trends consistent with the race model. Taken together, these observations suggest that antagonist neck muscle activity arises in response to presentation of the stop signal, serving the kinetic role of arresting the head movement in mid-flight via active braking. The selective appearance of antagonist muscle activity on trials where the gaze axis remained stable leads us to suggest that such antagonist muscle activity is a peripheral manifestation of the oculomotor STOP process.

One of the major advantages of the race model is that it allows estimation of processes that are unobservable, since the STOP process cancels motion on trials where it wins the race. However, the position of the gaze axis with the head-unrestrained is the sum of both
eye-in-head and head-in-space motion, since the eyes are nested within a mobile head.

The neural control of such nested systems is fundamentally bottom-up, in that the control of the upper-most component (eye-in-head) must account for the motion of the lower component (head-in-space) to accurately achieve the goal (eye-in-space; see also Tweed et al. (1999)). Close analysis of the timing of neck muscle recruitment during large eye-head gaze shifts demonstrates a bottom-up staggering of neuromuscular commands in many experimental conditions, with neck muscle recruitment leading eye muscle recruitment by intervals greater than 20 ms or more (Bizzi et al. 1971; Corneil et al. 2004; Zangemeister and Stark 1982). Such sequencing suggests that there should be instances where movement of a lower component has been initiated even though a commitment to move the upper-most component is ultimately withheld; head-only errors appear to represent such an instance.

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 inconsistent 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 shows 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 potential 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 STOP process, since the subjects successfully cancelled a gaze shift on such trials.

Numerous aspects of our results emphasize a close correlation between the antagonist neck muscle latency and the SSRT, consistent with this interpretation. Thus, although the functional role of the antagonist neck muscle activity is to actively brake the head, we propose that the timing of such recruitment provides a single-trial measure of when the commitment is made to cancel a developing, but still unexpressed at the level of the gaze axis, oculomotor program.

Neurophysiology of controlling eye-head gaze shifts

To our knowledge, ours is the first report suggesting that countermanding an eye-head gaze shift can produce quantifiable recruitment in the motor periphery. However, is the eye-head gaze shifting system a nested system, or could separate control of gaze and head orienting explain our results? If so, then the processes arresting head motion may not be linked to the oculomotor STOP process.
One of the main observations supporting a nested control structure is the absence of eye-only errors on STOP signal trials. If the control of gaze or head movements were determined by separate GO and STOP processes, then eye-only errors should have been observed on some STOP signal trials. The absence of eye-only errors in our task differs from that reported by Boucher et al. (2007b) during eye-hand countermanding, where errors could consist of eye-only, hand-only, or coordinated eye-hand movements. The generation of these various error types was one piece of evidence that led Boucher and colleagues (2007b) to suggest that the eye and hand were controlled independently.

We have suggested previously that head-only errors are generated when the oculomotor GO process exceeds a lower “head” threshold but the oculomotor STOP process wins the race to the gaze threshold. The notion that head movements are initiated at a lower threshold is consistent with other behavioural results in humans (Corneil and Munoz 1999; Ron and Berthoz 1991), and with a variety of neurophysiological results. For example, low-current stimulation of either the superior colliculus (SC) or frontal eye fields (FEF) can generate neck EMG activity and head movements without initiating gaze shifts (Corneil et al. 2010; Corneil et al. 2002a; b; Pélisson et al. 2001). Processes known to increase low-frequency preparatory activity within the SC also produce similar levels of neck muscle activity prior to gaze shifts (Corneil et al. 2008; Rezvani and Corneil 2008). Thus, developing oculomotor programs can drive head movements even while a decision to commit to a gaze shift is ongoing. Our results show that the cancellation of such developing programs is associated with selective recruitment of antagonist neck muscles.

Comparison of antagonist latencies and SSRTs, and the persistence of the STOP process

If the selective recruitment of antagonist neck muscle arises from the oculomotor STOP process, why then are antagonist latencies relative to the STOP signal longer than conventional SSRT estimates of the STOP process (Table 2)? Part of the difference likely arises from biomechanical differences in moving the eyes or head; for example, the head has a much greater inertia (Zangemeister and Stark 1981), and skeletal muscles take
longer to develop forces compared to extraocular muscles (Barmack et al. 1971; Botterman et al. 1986).

Additionally we speculate that head-only errors arise on a subset of trials where the STOP process is accumulating more slowly than on average, as this provides more time for the GO process to exceed the lower head threshold (see schematics in Fig. 5). Thus, we speculate that our measures of the antagonist burst latency are not derived from every single cancelled STOP signal trial (recall as well than an antagonist burst is not present on successfully cancelled STOP signal trials without head motion; Fig. 3). Thus, while we propose that the antagonist muscle burst arises from the STOP process, it is important to emphasize that it is not expressed on every successfully cancelled STOP signal trial.

It is also possible that the GO process, as it exceeds the head threshold, slows the accumulation of the STOP process. While such a speculation violates the race model’s assumption of independence between the GO and STOP process, it is consistent with recent behavioural and modelling results (Boucher et al. 2007a; Özyurt et al. 2003), and with mutually-antagonistic interactions of saccade- and fixation-related neurons within the oculomotor system (see Munoz et al. 2000 for review). The recent modelling efforts by Boucher and colleagues (2007a) 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 non-cancelled 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 cancelling or truncating head
movements. Another candidate is the precentral cortex lying posterior to the FEF in the
monkey. This area is labelled bilaterally following the injection of trans-synaptic
retrograde tracers into neck muscles (Billig and Strick 2010), 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 behavioural 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 behavioural 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 non-cancelled STOP trials (termed the neural SSRT) precedes the behavioural SSRT. Both neural and behavioural 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 behavioural 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 muscle can be recorded via surface techniques during an countermanding task, but we note that previous research has utilised 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 non-cancelled 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 amongst 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.
Acknowledgements

We thank JS Blouin and T Admans for assistance with the intramuscular insertion; K Barker for technical assistance; and T Wilson for providing assistance with anatomy.

GRANTS

This research was supported by an operating and group grant from the Canadian Institutes of Health and Research (CIHR), the Natural Sciences and Engineering Research Council and infrastructure support from the Canadian Foundation for Innovation. BD Corneil is a CIHR New Investigator.
1. A key assumption to this line of reasoning is that performance on STOP trials is determined by a nested race of an oculomotor GO process versus an oculomotor STOP, rather than separate races that control gaze or head movements independently. In this nested race scenario, described in further detail in Corneil and Elsley (2005), head-only errors occur when the GO process surpasses a lower threshold for initiating head movements even though the STOP process ultimately wins the race to suppress a gaze shift. As described in more detail in the Discussion, the absence of “eye-only” errors and a series of neurophysiological results support this nested race scenario.
Figure Legends

Figure 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 two processes, the movement is either generated or successfully withheld. B: Schematic representation of the task during a STOP signal trial. The fixation point (FP) was extinguished 200 ms prior to target presentation. The FP reappears after target presentation with a variable delay termed the stop signal delay (SSD), which signalled that the planned gaze shift should be cancelled.

Figure 2

CT image on the muscle of interest in purple; splenius capitis (SPL).

Figure 3

Positional (I) and intramuscular EMG activity (II and III) on CONTROL (A) and STOP trials (B-D) classified into the three main movement sequences observed. Magenta, eye (Eh); blue trace, head (Hh); cyan trace, gaze (Gh), with the red crosses on the STOP trials indicating the onset of the stop signal for that given trial. All data is from subject S2 for rightward target trials. Data is aligned to target onset (dashed vertical line). In the colour EMG plots each stacked row shows the EMG data from a single trial, ordered by gaze shift reaction time (white square) (A & C), or head movement reaction time (green solid circle) (D) with the red solid circle indicating the end of the head movement. The data in B is not ordered as cancelled trials are characterised by an absence of displacement of the movement traces. Below each colour plot is the average EMG data for that condition across all trials.

Figure 4

Inhibition functions for each subject showing the probability of gaze shifts (solid line) or head movements (either with or without a gaze shift, short dashed line) on STOP trials as
a function of SSD. The difference between head and gaze inhibition functions is also shown which represents the probability of a head only error (long dashed line).

**Figure 5**
The average rank SSD for each subject for the three types of movement sequences observed on STOP trials. Note that head-only errors occurred at the intermediate SSDs on average. The bottom of this figure shows a schematic representing the activation process of the STOP and GO processes for each of the movement classes with respect to the individual thresholds for the gaze and head.

**Figure 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-efficient for each subject is also shown with an asterisk indicating a correlation which was statistically significant at the alpha level of 0.05.

**Figure 7**
Schematic showing the onset of antagonist EMG activity (squares) in relation to stop signal onset (vertical line) for all subjects across both directions and for each SSD. Empty circles indicate target onset. Below each plot is a histogram showing the distribution of reaction times of the antagonist burst for each subject. The mean and standard deviation of the antagonist latency in relation to the onset of the stop signal is also shown for each subject.

**Figure 8**
(A) Average antagonist latency for all subjects at each SSD where more than two head-only errors were detected. Solid lines indicate that there is no difference in the latency of the antagonist burst across the SSDs, (one-way ANOVA p > 0.05), dashed lines indicate significant difference in the antagonist latency across the different SSDs (one-way ANOVA p < 0.05).
ANOVA P < 0.05). The degrees of freedom and F-statistic is also shown for each subject. (B) Shows the average trend across subjects for the antagonist latency and the estimated SSRT across a rank ordered SSD. Although there was no significant trend in either set of data both the antagonist latency and the SSRT measure show a similar trend of decreasing with increasing SSD.

Figure 9
Subject by subject correlations of the average latency of the antagonist neck muscle activity versus the gaze SSRT (A) or head SSRT (B). Each data point shows the data from an individual subject, with SSRTs averaged across both the mean and integration method. (C) Shows 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. (D) Shows the correlation between the antagonist latency and the gaze SSRT measured using the integration method at SSDs were at least three head-only errors were observed. Each point indicates a unique combination of antagonist latency and SSRT measure across all subjects. The correlation co-efficients and corresponding p-values for these correlations are shown for each comparison.
References


TABLE 1. Response sequence probabilities on STOP trials for all subjects.

The number of observations for each movement sequence are followed by the probabilities in brackets.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Number of STOP trial</th>
<th>Response Probability on STOP trials</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No Gaze Shift</td>
<td>Cancelled</td>
<td>Head-only</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gaze Shift</td>
<td>Completed</td>
<td>Truncated</td>
</tr>
<tr>
<td>s1</td>
<td>155</td>
<td>33 (0.21)</td>
<td>37 (0.24)</td>
<td>40 (0.26)</td>
</tr>
<tr>
<td>s2</td>
<td>165</td>
<td>35 (0.21)</td>
<td>56 (0.34)</td>
<td>32 (0.20)</td>
</tr>
<tr>
<td>s3</td>
<td>171</td>
<td>61 (0.36)</td>
<td>35 (0.20)</td>
<td>30 (0.18)</td>
</tr>
<tr>
<td>s4</td>
<td>171</td>
<td>88 (0.51)</td>
<td>43 (0.25)</td>
<td>8 (0.05)</td>
</tr>
<tr>
<td>s5</td>
<td>175</td>
<td>61 (0.35)</td>
<td>39 (0.22)</td>
<td>64 (0.37)</td>
</tr>
<tr>
<td>s6</td>
<td>168</td>
<td>46 (0.27)</td>
<td>17 (0.10)</td>
<td>72 (0.43)</td>
</tr>
<tr>
<td>s7</td>
<td>177</td>
<td>83 (0.47)</td>
<td>31 (0.18)</td>
<td>44 (0.25)</td>
</tr>
<tr>
<td>s8</td>
<td>140</td>
<td>16 (0.11)</td>
<td>38 (0.27)</td>
<td>50 (0.36)</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>35 (0.23)</td>
<td>37 (0.25)</td>
<td>43 (0.29)</td>
</tr>
</tbody>
</table>

Mean: 35 (0.23) 37 (0.25) 43 (0.29)
Table 2: Estimates of gaze and head 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>
<td>187</td>
</tr>
<tr>
<td>s7</td>
<td>99</td>
<td>139</td>
<td>149</td>
</tr>
<tr>
<td>s8</td>
<td>142</td>
<td>154</td>
<td>198</td>
</tr>
<tr>
<td>Mean</td>
<td>129 ± 31</td>
<td>162 ± 33</td>
<td>180 ± 26</td>
</tr>
</tbody>
</table>
I  Position

II  Agonist

III  Antagonist

CONTROL trials

A  

TARGET

Gh  Hh  Eh

Gaze  Head

40 μV

Head-only

SSD

100 ms

Time re. target onset (ms)

Goonetilleke et al. Fig. 3
Stop signal delay (ms)

Gaze
Head
Difference

P(movement)

S1
S2
S3
S4
S5
S6
S7
S8

Goonetilleke et al. Fig 4
Stop trial type

Rank order SSD

- Non-cancelled
- Head only
- Cancelled

Gaze threshold
Head threshold

Activation process

Goonetilleke et al. Fig 5
A

Normalized antagonist EMG activity

Head movement amplitude during head-only errors (degrees)

B

Normalized antagonist EMG activity

Peak velocity during head-only errors (degrees/sec)

Goonetilleke et al. Fig 6
Antagonist latency re: stop signal onset (ms)
A

Stop signal delay (ms)

Antagonist latency (ms)

S1; $F_{(3,32)} = 1.57$

S2; $F_{(6,48)} = 2.33; p = 0.05$

S3; $F_{(5,28)} = 3.12; p = 0.02$

S4; $F_{(5,37)} = 0.73$

S5; $F_{(5,33)} = 1.43$

S6; $F_{(5,8)} = 0.72$

S7; $F_{(5,32)} = 2.95$

S8; $F_{(5,32)} = 0.95$

B

Normalised SSRT and Antagonist latency

SSRTs

Antagonist latency

Rank order SSD

Goonetilleke et al. Fig 8
A

B

C

D

Goonetilleke et al. Fig 9