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RESEARCH ARTICLE

D.W.J. Cabel · I.T. Armstrong · E. Reingold
D.P. Munoz

Control of saccade initiation in a countermanding task using visual and auditory stop signals

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Abstract We examined inhibitory control in an oculomotor countermanding task, where the primary task required a saccadic eye movement be made to a target and a less-frequent secondary task required that the movement be halted. Previous studies have used a visual stimulus presented centrally on the fovea as the signal to stop or countermand a saccade. In these previous studies, there are at least two possible sources of saccadic inhibition: (1) sensory stimulation at the fovea can elicit a bottom-up mechanism, where a visual transient signal can delay or inhibit the developing saccade command; and (2) information based on the task instruction can be used to initiate a top-down mechanism to halt the movement. In the present study, we used both visual and auditory stop signals to test the hypothesis that the bottom-up mechanism is activated only after presentation of a foveal visual stop signal. Subjects were instructed first to look at a central spot and then to look to an eccentric visual target that appeared randomly to the left or right of center. On about one-third of the trials, a stop signal was presented. Three types of stop signals were used with equal probability: a broad-band noise burst (auditory), a central fixation spot (visual), and a combination of the auditory and visual stimuli (combined). Saccadic reaction time and stop-signal accuracy were used to calculate stop signal reaction time (SSRT), an estimate of the time required to inhibit the eye movement. Mean SSRT was longer for the auditory stop signals (201 ms) than for the signals with a foveal visual component (visual 113 ms; combined 91 ms). We conclude that a foveal visual stop signal in an oculomotor countermanding task changes the measure of inhibitory control to reflect not only inhibitory processes but also the sensory information afforded by stimulation at the fovea.

Key words Eye movement · Oculomotor countermanding · Saccade · Inhibitory control · Human

D.W.J. Cabel · I.T. Armstrong · D.P. Munoz (✉)
Department of Physiology, Queen's University, Kingston, Ontario, K7L 3N6, Canada
e-mail: doug@eyeml.QueensU.CA
Tel.: +1-613-5332111, Fax: +1-613-5336840

E. Reingold
Department of Psychology, University of Toronto, Toronto, Ontario, Canada

Introduction

Oculomotor countermanding is used to investigate saccade initiation and the inhibitory control of voluntary eye movements (Hanes and Schall 1995, 1996; Hanes et al. 1998; Hanes and Carpenter 1999). The primary task, presented more frequently, requires an eye movement to a peripheral target; reaction time (RT) is measured from target appearance to the onset of the movement. In addition, a less-frequent secondary task requires subjects to halt an impending movement whenever a stop signal is presented. The delay from target presentation until the appearance of the stop signal (stop-signal delay) is varied in intervals that span the primary-task RT distribution. The probability of successfully inhibiting an eye movement changes across stop-signal delay. When the stop signal appears soon after target appearance, subjects are able to inhibit a movement, but as the stop-signal delay increases, subjects are less likely to stop. The inhibition function, which is the change in the probability of inhibitory success as a function of stop-signal delay (Logan and Cowan 1984), is used to calculate two standardized measures: (1) the stop-signal reaction time (SSRT), an estimate of the time needed to cancel a response that is independent of the stop-signal delay; and (2) the slope of the normalized inhibition function. These standardized measures allow comparisons of inhibitory control across clinical populations (Schachar and Logan 1990) or under various experimental conditions (Hanes and Carpenter 1999).

In the oculomotor countermanding task, SSRT to a visual stop signal was found to be around 90 ms in monkeys (Hanes and Schall 1995) and ranged from 125 to 145 ms in humans (Hanes and Carpenter 1999). In these

previous studies, the stop signal was always presented to subjects visually at the fovea. The presentation of a visual foveal stop signal could elicit a stopping response in two ways. First, stopping depends on the instruction to the subject; the instruction motivates inhibition of the planned movement (top-down). Second, stopping may depend on the sensory response evoked by the sudden appearance of a visual stop stimulus (bottom-up); its appearance produces a visual transient response that could delay or inhibit the programming of an eye movement (Corneil and Munoz 1996; Walker et al. 1997; Findlay and Walker 1999; Olivier et al. 1999; Reingold and Stampe 1999).

To evaluate whether one mechanism for inhibitory control to a visual foveal stimulus in oculomotor countermanding is elicited primarily by a reflexive sensory mechanism, we also presented stop signals in another modality. If the stimulus-specific processes evoked by the foveal presentation alter the measures of oculomotor inhibitory control, we expect both SSRT and the normalized inhibition function to change when compared with measures found with a nonfoveal stop signal. Furthermore, such a changes should alter the slope of the standardized inhibition function (Logan and Cowan 1984).

Under identical instructions, we presented either an auditory stimulus, a central, foveal visual stimulus, or a combination of these stimuli as the secondary, stop-signal task trials within a block of primary task reaction time trials. This design allows us to compare the effectiveness of the nonfoveal auditory stimulus with the foveal visual stimulus. SSRT was significantly longer for auditory stimuli. Part of this work has been presented in abstract form (Cabel et al. 1999).

Materials and methods

Subjects

All experimental procedures were reviewed and approved by the Queen's University Human Research Ethics Board. Five na subjects between the ages of 22 and 25 years (two women) were informed of the general nature of the study and consented to participate. Subjects reported no visual, auditory, neurological, or psychiatric disorders and none reported taking any psychoactive medication.

Subjects were tested first in a modality intensity task, which lasted 30 min, followed by the countermanding task. Countermanding data were collected in five separate 45-min sessions distributed over 1 week. Subjects were debriefed only after the final session.

Apparatus

Visual displays were presented on a 17-inch Viewsonic 17PS monitor using an S3 VGA card. The visual display had a resolution of 640×480 pixels, with a frame rate of 60 Hz. Eye movement data were collected using a video-based eye-tracker (Eyelink, SR Research), which was mounted on the head of each subject with an adjustable headband. The eye-tracker uses infrared cameras to track the movements of the pupils, measuring pupil size and vertical and horizontal eye position with a spatial resolution of 0.005° and a sampling rate of 250 Hz. It also provides spatial information about head position for head-motion compensation. The eye-tracker system employed an Ethernet link between the eye-tracker and the display computers for real time saccade, gaze position, and button-press data transfer. The configurable acceleration and velocity thresholds were set to detect saccades greater than 0.15°. Responses were also collected from buttons pressed on a hand-held manipulandum; button pressing initiated a trial and, during the modality intensity task, marked subject responses.

Countermanding

Subjects sat 60 cm away from a display monitor. Each trial started with the presentation of a 1-cm white (1.40 cd/m^2; CIE coordinates: x=0.299, y=0.300) octagonal fixation spot centered on a black background. When ready, subjects initiated the trial with a button press; after a 500-ms delay, the fixation spot disappeared and a green target (0.72 cd/m^2; CIE coordinates: x=0.294, y=0.533) equal in size and shape to the fixation spot appeared randomly 5° to the left or right of center. Subjects were instructed to look to the eccentric target when it appeared unless a stop signal was presented. In the presence of a stop signal, the subjects were instructed to suppress the saccade to the eccentric target. Stop signals consisted of one of the following stimuli with equal probability: (1) a broad-band noise burst (440 Hz) emitted from a speaker located 2 m above the head of the subject; (2) a red visual stimulus of the same size and shape and in the location of the previous fixation signal; or (3) a combination of both the auditory and visual stop signals. The relative intensity of each stop signal was matched for each subject using a modality intensity test. The stop-signal presentation followed target appearance after delays that ranged from 0 to 350 ms in 50-ms steps (stop-signal delays). The stop signal and the eccentric target then remained illuminated for the duration of the trial.

Subjects completed five sessions consisting of four blocks of 128 trials each. Trials were randomly ordered within a block in the following proportion: no stop signal (GO trials; 62.5%), auditory stop signal (12.5%), visual stop signal (12.5%), and the combination of visual and auditory stop signals (12.5%). Eye-tracker data were recorded on each trial from the initial button press until 800 ms after target appearance.

Modality intensity

To ensure that the stop-signal stimuli across modalities produced consistent reaction times, the intensity of the stimuli were varied systematically in blocks designed to measure response time. Subjects were instructed to press a button after presentation of either a visual or an auditory stimulus. A white circular fixation point (1 cm diameter; 2.80 cd/m^2; CIE x=0.297, y=0.313) centrally located on a black display preceded each trial. A button press initiated each trial. After a randomized delay ranging from 25 to 200 ms in 25-ms steps, a visual or an auditory stimulus was presented for 100 ms, and the subject responded as quickly as possible with a button press. A total of 14 blocks of 32 trials each were used. Only one stimulus was varied in each block. The visual stimulus was a red disc the same size and shape as the fixation point presented at the same location as the fixation point using one of eight unique visual intensity levels. Intensity was varied in equal steps across the 256 internal levels of the RGB-controlled visual display. The auditory stimulus was delivered via a speaker, which was fixed 2 m above the subject's head and presented in one of six auditory intensity units at 440 Hz.

Data collected from each subject provided a RT distribution as a function of intensity level for both modalities (Fig. 1). The shortest mean RT was used to determine the intensity level of each presentation mode necessary to produce consistent performance in the countermanding task.

Data analysis

The five subjects provided a total of 8000 GO trials and 4800 stop trials, consisting of 1600 trials each of auditory, visual and com-

Table 1 Minimum button press reaction time (milliseconds) in the modality intensity task (data from Fig. 1), the difference between these minimum values, and the subsequent auditory and visual intensity levels used in the countermanding task for each subject

Subject	Auditory (ms)	Visual (ms)	Difference (ms)	Auditory Intensity (dB)	Visual Intensity (cd/m^2)
AB	210	240	30	77	0.80
JD	186	271	84	77	0.80
KL	173	218	45	79	1.07
KK	220	267	47	79	0.80
MH	213	260	47	77	1.07
Mean	200	251	51		

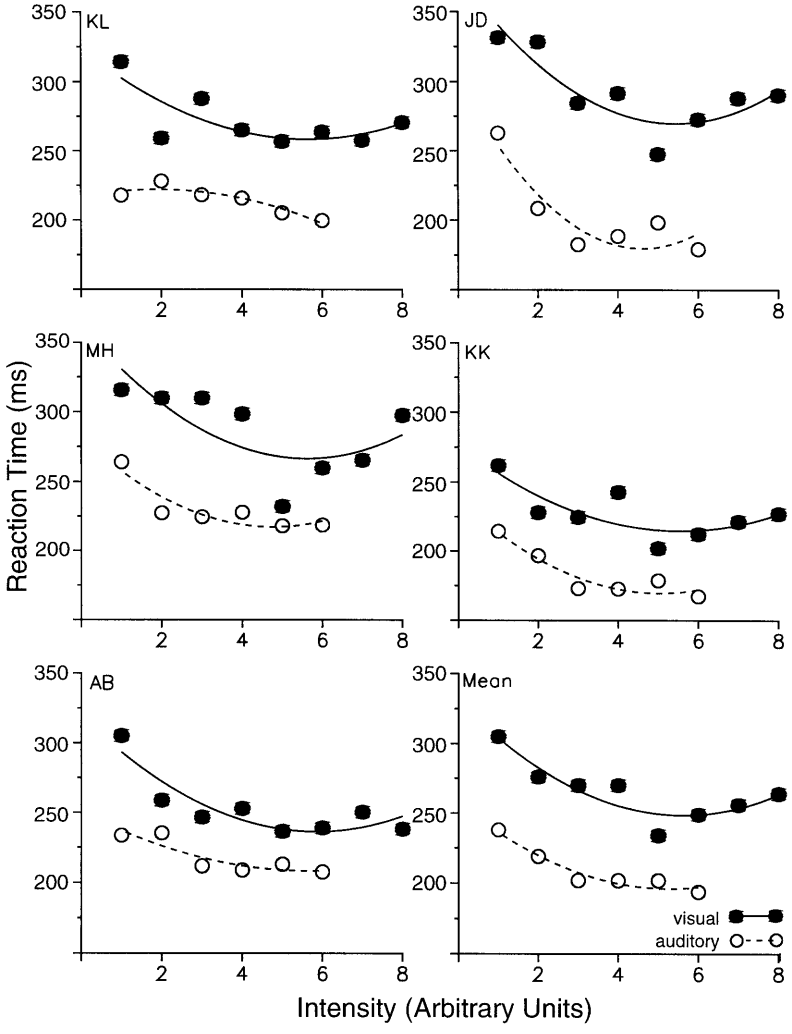

Fig. 1 The button-press mean reaction time (RT) plotted as a function of stimulus intensity for each of the eight visual and six auditory stimuli for each subject. The minimum values were the point where an increase in stimulus intensity no longer reduced RT. Because there is one common efferent pathway to control the muscles and produce a button press independent of stimulus modality, the difference between minimum values was considered to be a modality-processing difference or afferent delay. The curves were produced by a 2nd-order polynomial fit through the data points. Auditory intensities employed were 51, 59, 65, 72.5, 77, and 79 dB. Visual intensities employed were 0.11, 0.12, 0.25, 0.34, 0.80, 1.07, 2.00, and 2.15 cd/m^2

bined stop-signal types. Both correct and incorrect responses were analyzed separately for GO and stop trials. A correct GO trial was one in which the subject initiated a saccade to the eccentric target within 800 ms of its appearance. An incorrect GO trial occurred when the subject failed to initiate a saccade and instead maintained central fixation. A stop trial was correct when the subject maintained central fixation, whereas the initiation of a saccadic eye movement in the presence of a stop signal constituted an incorrect response. Saccadic RT was calculated for correct GO trials and incorrect stop trials.

Data were analyzed using analysis of variance on mean performance for the factors of direction (left vs right saccades) and signal type (auditory, visual and combined) and, for the stop trials, stop-signal delay (0, 50, 100, 150, 200, 250, 300, 350 ms) at an alpha of 0.05. Post hoc analyses were performed using linear contrasts with the conservative mean estimates (at 1 *df*).

Results

Modality intensity

Mean RT for a button press response was measured for each subject and plotted as a function of stimulus intensity for both auditory and visual stimuli (Fig. 1). As stimulus intensity increased, the mean RT decreased toward a minimum. The auditory stimulus yielded consistently shorter RT than the visual stimulus (see Table 1). During the subsequent countermanding task, we used the intensity values for both auditory and visual stimuli that produced the minimum button press RT values for each

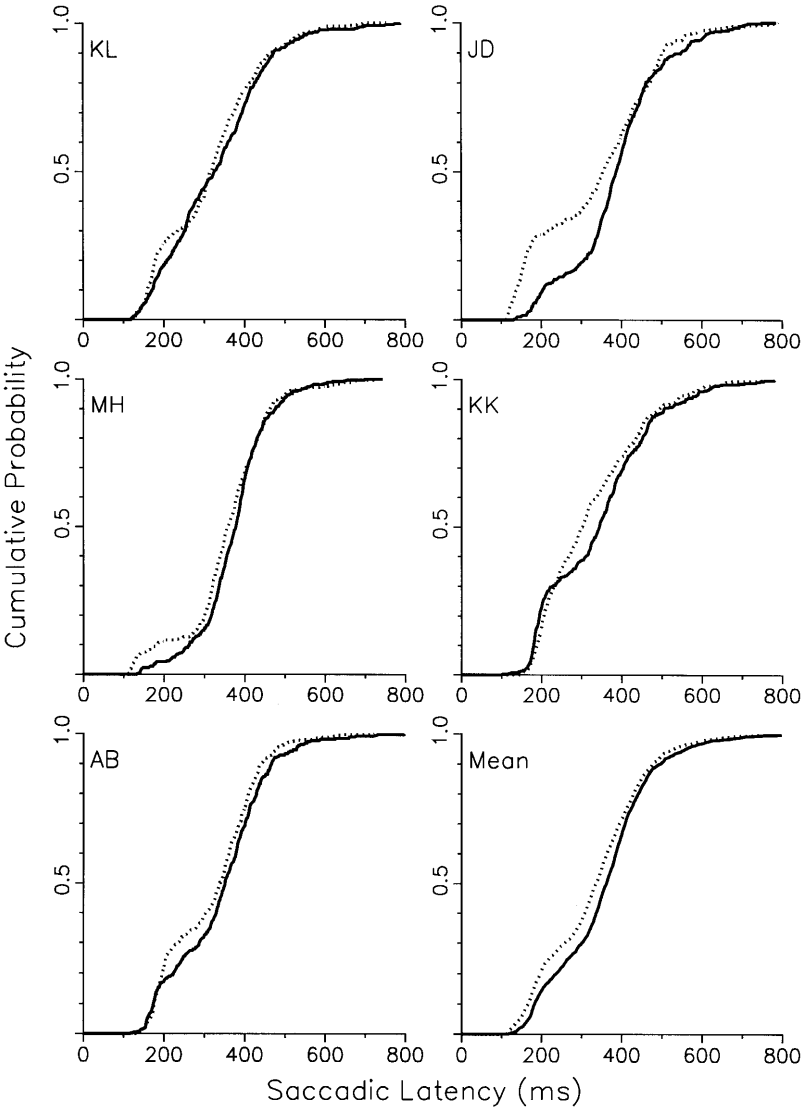

Fig. 2 GO trial saccadic RT cumulative probability distributions for rightward (*dotted traces*) and leftward (*solid traces*) saccades for each subject and mean RT

Table 2 Mean GO-trial saccadic reaction time (RT) ± SD (milliseconds) to the left and right targets and proportion of errors (combined directions)

Subject	RT ± SD		P(error)
	Left	Right	
AB	336±114	317±116	0
JD	392±139	330±150	0.001
KL	328±121	319±126	0
KK	324±153	309±144	0
MH	369±105	314±148	0.002
Mean	350	318	0.0006

subject. The mean difference in RT between auditory and visual stimuli among the five subjects was 51 ms (range 30–84 ms; Table 1). The intensities of the auditory and visual signals at the minimal RTs are also noted in Table 1 for each subject.

Countermanding

SSRT, the measure of inhibitory control, is estimated using the GO-trial RT distribution and the distribution of subject accuracy on stop trials as a function of stop-signal delay. We estimated SSRT for each stop-signal modality type. Before discussing SSRT performance, we describe performance on GO and stop trials separately.

GO trials

The cumulative distributions of leftward and rightward saccadic RT are shown in Fig. 2 for all correct GO trials. The saccadic RTs from all subjects tended to form a bimodal distribution, with an initial peak between 150 and 200 ms and a second later peak between 300 and 450 ms. Leftward saccades had slightly longer mean RT (Fig. 2, solid traces; mean ± SE 350±29 ms) than rightward saccades (Fig. 2, dotted traces; mean ± SE 318±8 ms). The

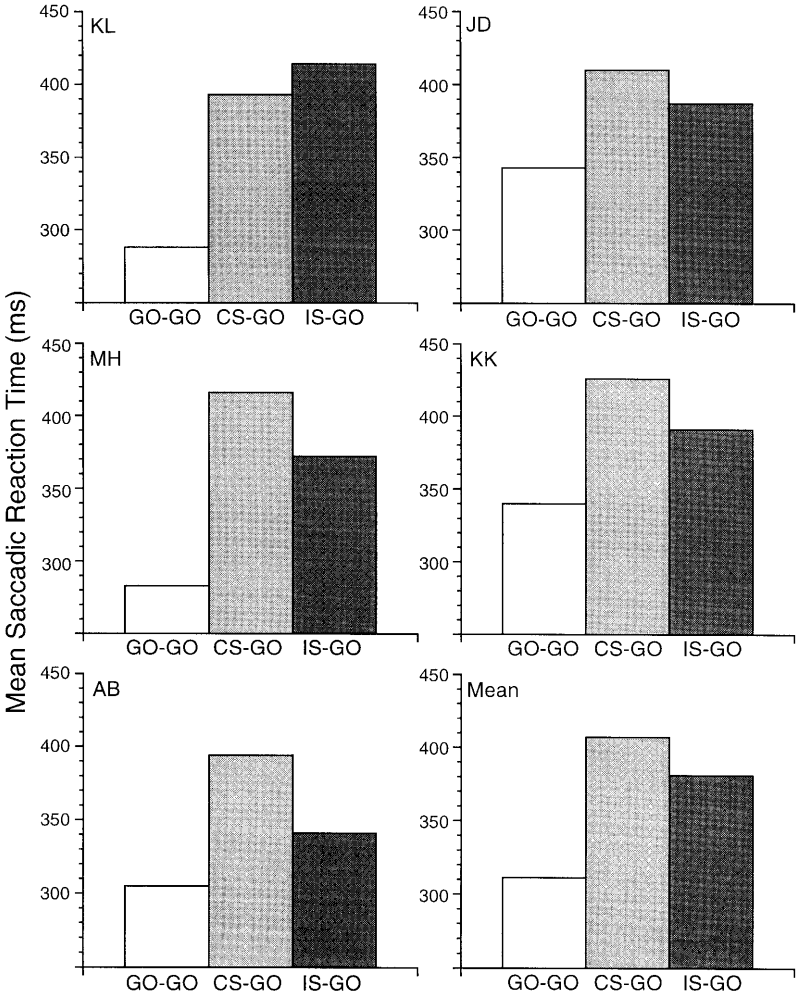

Fig. 3 Effect of the preceding trial on GO-trial saccadic RT. Data for each subject were separated by preceding trial type: a correct GO trial (*GO-GO*); a correct stop trial (*CS-GO*); and an incorrect stop trial (*IS-GO*)

difference in latencies between leftward and rightward saccades was significant ($F_{1,4}$=8.53, P<05; see Table 2).

Sequential effect on reaction time

Figure 3 shows the sequential effect on GO-trial RT when segregated by the previous trial type. GO-trial RT was segregated as a function of the preceding trial's accuracy for three possible trial types: a correct GO trial (GO-GO), a correct stop trial (CS-GO), and an incorrect stop trial (IS-GO). The fourth condition, incorrect GO preceding correct GO, was not analyzed because incorrect GO trials occurred so rarely (0.06% of trials). RTs were shorter (313 ms) in the GO-GO condition than if the preceding trial were a stop condition (395 ms; $F_{1,28}$=45.5, P<0.001). Performance on GO trials preceded by stop trials was dependent on the accuracy of the stop trials. If the preceding stop trial was incorrect, that is, the subject looked to target in error, the mean RT of the subsequent GO trial was shorter (379 ms) than when the preceding stop trial was performed correctly (412 ms; $F_{1,28}$=12.6, P<0.01). The modality of the stop signal had no effect on subsequent GO trial performance, F<1 for both CS-GO and IS-GO conditions.

Stop trials

Inhibition function

The inhibition function is a measure of subjects' ability to stop an eye movement at each stop-signal delay. Figure 4 shows the probability of a saccade, P(Response), as a function of stop-signal delay for auditory, visual and combined stop signals for each participant. As expected, after a brief stop-signal delay, subjects were more successful at suppressing saccades, but, over longer delays, subjects increasingly failed to withhold a saccade. In general, leftward saccades were stopped more effectively than rightward saccades (F1,192=24.33, P<0.0001). The leftward-saccade preference was consistent for all stop-signal delays and signal presentation modes. As shown in Fig. 4, the change in inhibition function depended on the mode of the stop signal except at the longest stop-signal delays. For the shorter delays, the auditory stop signal was less effective than either the visual or the combined signal types which produced similar functions ($F_{14,192}$=2.68, P<0.01).

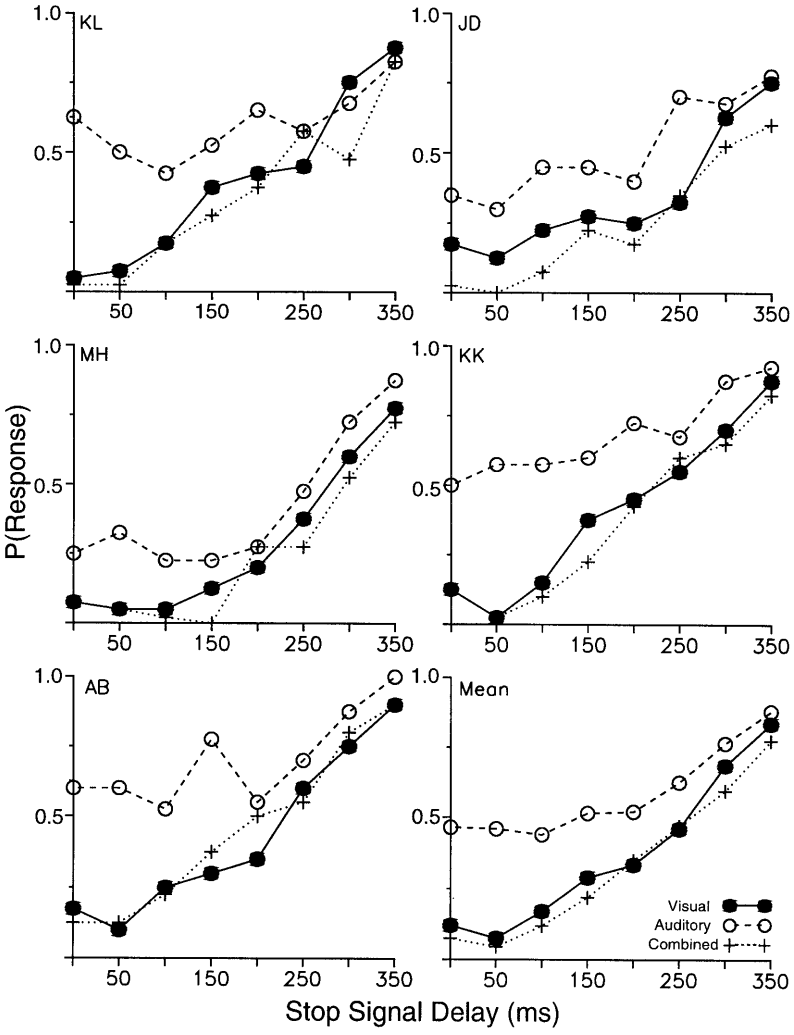

Fig. 4 The inhibition function for each subject and the mean inhibition function. The probability of response given the stop signal, *P(Response)*, is plotted as a function of stop-signal delay

Stop-signal reaction time

SSRT is an estimate of the time required to cancel a planned saccade and maintain central fixation. Logan and Cowan (1984) suggested that SSRT should be calculated using both the GO-trial saccadic RT cumulative probability (Fig. 2) and the inhibition function (Fig. 4). The cumulative GO-trial RT distribution is integrated from left to right until the integral is equal to the probability of a saccade given a stop signal. The stop-signal delay is subtracted from this value to yield the SSRT. We estimated each subject's SSRT in this fashion for each stop-signal type and delay.

Mean SSRTs were 201 ms, 113 ms, and 91 ms for auditory, visual and combined stop signals, respectively (see Table 3). The initial modality intensity task yielded shorter RT for auditory signals than for visual signals (see Fig. 1 and Table 1), probably reflecting differences in afferent delay inherent to the separate modalities. On average there was a 51-ms difference in afferent delay (see Table 1), favoring auditory stimuli. Adding this afferent delay difference to the auditory SSRT exaggerated the advantage for stop-signal SSRT with a visual component (see examples in Table 3). All subsequent analyses were performed on the data without the correction factor.

Table 3 Mean stop-signal reaction times for combined directions

Subject	Auditory (ms)	Corrected auditory (ms)	Visual (ms)	Combined (ms)
AB	261	291	126	135
JD	199	283	118	56
KL	177	222	116	76
KK	225	272	110	97
MH	142	189	94	93
Mean	201	251	113	91

SSRT was analyzed using a three-factor analysis of variance for the factors target direction (left vs right), stop-signal delay (0–350 ms), and signal type (auditory, visual, or combined). SSRT was relatively constant for all stop-signal delays when the visual and combined signals were presented, but the SSRT measure at brief delays was significantly longer for the auditory stop signals ($F_{14,56}$=3.35, P<0.005). At long delays, the auditory stop signal produced SSRTs that were smaller than at brief

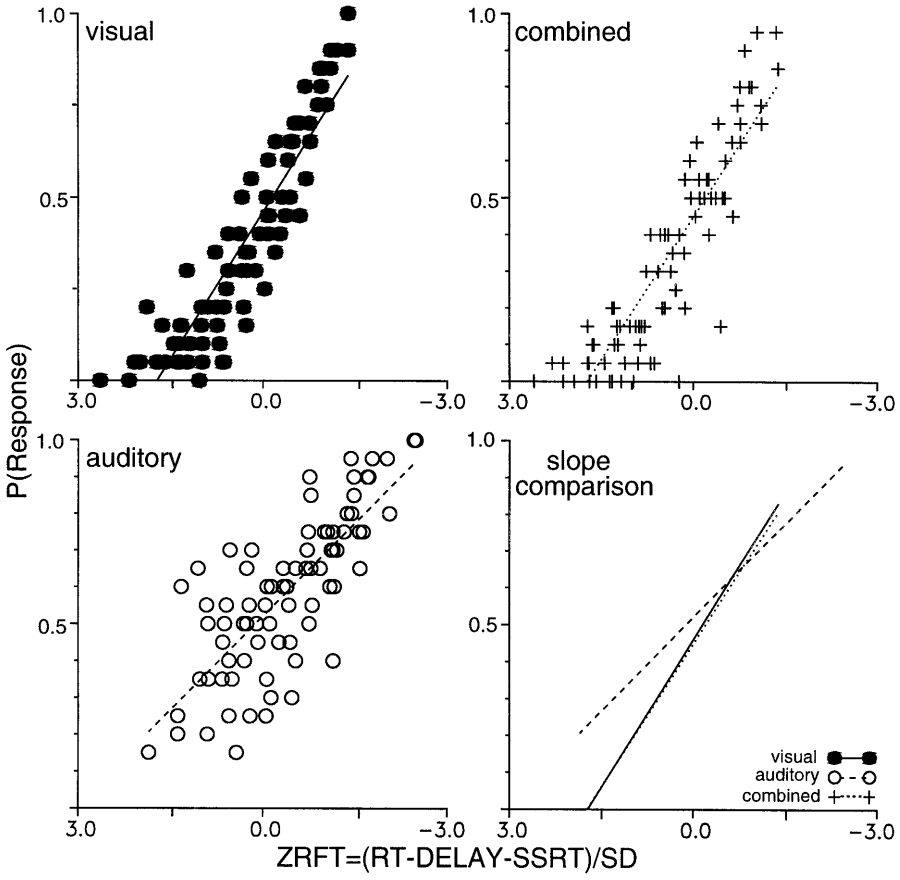

Fig. 5 The probability of response, *P(Response)*, as a function of the GO-trial RT distribution and the stopping process, expressed as a *Z*-score, combined across subjects for each stop signal. The slope lines were produced by a linear regression fit (auditory slope –0.16±0.05; visual slope –0.28±0.05; combined slope –0.27±0.05). (*SSRT* stop signal reaction time)

delays, but they continued to be much larger than the visual or combined stop signals.

Consistent with GO-trial RT, the visual and combined mean SSRT was shorter for leftward target presentations; in contrast, auditory mean SSRT was indifferent to the direction of the target ($F_{2,8}$=10.82, P<0.01). The direction of target presentation alone did not interact with stop-signal delay.

Control for RT and accuracy

Inhibitory control depends on the contingencies set up by the subject on GO trials as well as the subject's ability to inhibit a saccade. Logan and Cowan (1984) standardized the inhibition function by taking into account SSRT, stop-signal delay (SSD), the mean RT and standard deviation (SD) of the GO trials. The expression:

$$ZRFT = \frac{(RT - SSD - SSRT)}{SD} \quad (1)$$

normalizes reaction time and accuracy for each subject. Figure 5 shows the slopes of the normalized inhibition functions for each stop-signal type. An ANOVA performed on the slopes of the normalized inhibition functions for each direction of the saccade and for each stop-signal type revealed a difference between leftward and rightward stop-signal trials ($F_{1,4}$=7.21, P=0.05). More importantly, however, the slope of the normalized auditory inhibition function was less steep than the other two stop-signal types ($F_{1,4}$=10.83, P<0.05), and these slope differences were consistent for both target directions. Post hoc contrasts showed no difference in slopes between the visual and combined stop signals, $F_{1,4}$<1. Thus, the underlying inhibition control processes depended upon stop-signal modality.

Practice effects

Modality differences were not affected by practice. We examined performance across each day of the 5 recording days. As shown in Fig. 6, all but one subject showed improved stop-trial performance over the course of the 5 days of data collection ($F_{4,16}$=3.19, P<0.05). The practice effect was consistent across modalities and no interaction of practice with stop-signal modality was observed ($F_{8,32}$=1.33, P>0.25).

Discussion

Participants took longer to inhibit saccadic eye movements to auditory stop signals than to the visual or combined stop signals. The similarity of results for the visual-only and combined stop signals suggests that the visual component dominated behavioral responses in both. We

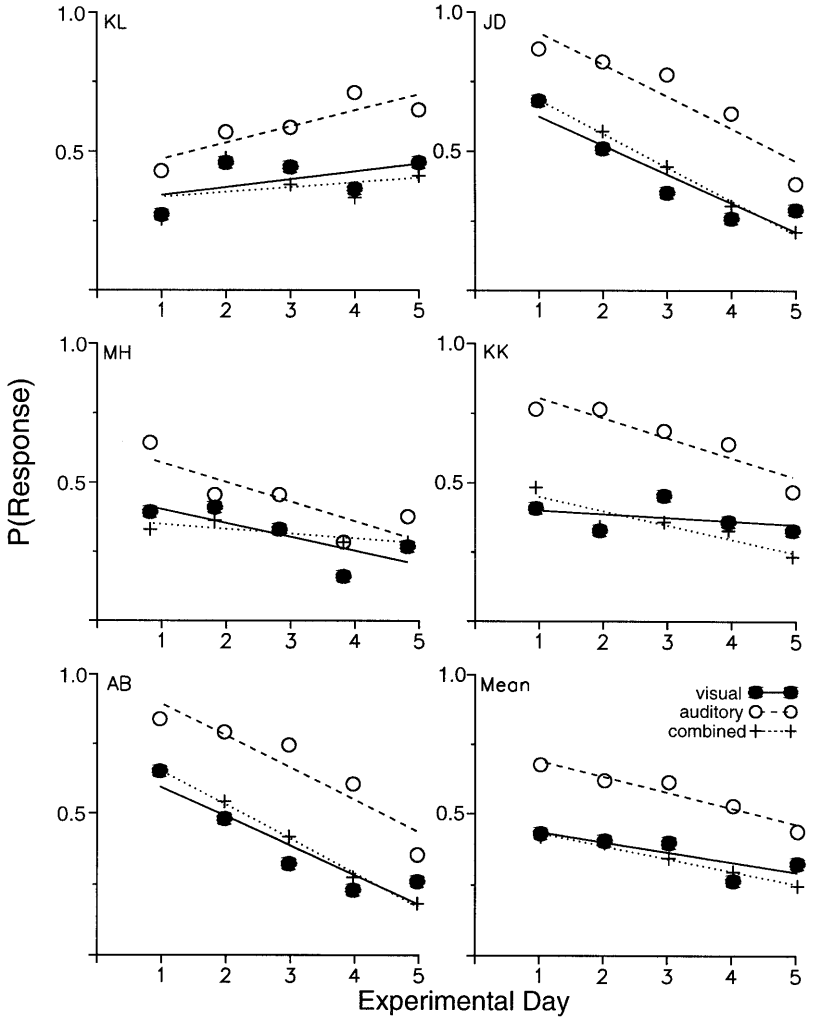

Fig. 6 Practice effects. Mean stop-trial accuracy (probability of responding on a stop trial) across days of practice. Lines were produced by a linear regression fit. Each data point represents the mean probability of responding across an entire session

conclude that the processes for inhibitory control of saccades following presentation of a nonlocalized auditory stimulus are distinct from the mechanisms controlling inhibition following presentation of foveal visual stimuli.

In the oculomotor countermanding task used in the current study, the foveal visual stimuli provided the stop instruction in two ways: (1) as an exogenous abrupt-onset stimulus, the sudden appearance of the visual stop signal initiated sensory processes that delayed or inhibited the planned saccadic movement (bottom-up); and (2) as an endogenous cue that elicited a more abstract instruction to inhibit the planned movement (top-down). These same mechanisms were not elicited by the auditory stimulus; hence, SSRT increased for the auditory stop signals. Thus, a unique property of visual pathways is that they can exert an advantageous influence of inhibitory control over saccade generation that is not available to auditory processing pathways. It is important to note that at this stage we cannot rule out the possibility that the difference in SSRT between visual and auditory stimuli was not due to modality per se, but rather due to the unique locations of the stimuli. Regardless, we can conclude that different processes were used to countermand saccades with the different stop signals we employed.

Modality equivalence

Varying the intensity of the stimuli allowed us to test equivalence of the stop signals across modalities. If we assume that the motor command to effect a button-press response is independent of the modality of the stimulus, then differences in RT across modality should reflect differences in afferent processing time between the auditory and visual stimuli. The time difference was on average 51 ms (see Fig. 1, Table 1), a value consistent with the range reported in the literature (Gouras 1967; Kraus et al. 1992). Correcting for the afferent processing time only exaggerated the difference in SSRT between visual and auditory stop signals (see Table 3).

GO trials

Subjects appeared to adopt very different strategies for orienting to the visual targets on GO trials. The RT data in Fig. 2 reveal a bimodal distribution with a first peak between 150 and 200 ms and a second peak between 300 and 450 ms. This bimodality, which was observed to varying degrees in all subjects, was independent of the

bimodality seen in many saccadic RT studies, in which the first peak has been termed express saccades (Fischer and Weber 1993). The range of latencies for express saccades are typically between 90 and 140 ms. Instead, the first peak in our distribution is analogous to the fast regular saccades described by Fischer and Weber (1993). The second, later peak we observe is manifested only in paradigms such as countermanding, in which subjects are often trying to delay saccade initiation until the last possible stop-signal delay has passed. This type of bimodality was observed to a lesser degree among na participants in another oculomotor countermanding study involving human subjects (Hanes and Carpenter 1999). It is of interest to note that, in the Hanes and Carpenter (1999) study, stop-signal delays ranged from 10 to 170 ms for high-contrast targets and from 50 to 210 ms for low-contrast targets, while the stop-signal delays we employed ranged from 0 to 350 ms. Perhaps the enhanced second peak of GO responses in our study reflects the longer stop-signal delays we employed. Despite this methodological difference, the calculated SSRT for foveal visual stop signals used in the two studies are comparable.

A novel finding in our study is that the previous history of trial type had a profound effect on GO trial RT (see Fig. 3). GO trial RTs were increased by about 80 ms when the previous trial was a stop trial. It is quite likely that this exaggeration in RT that followed a stop trial persisted beyond more than one trial. A recent neurophysiological study in nonhuman primates has revealed that the previous history of trial type can persist across multiple trials and influence the excitability of saccade-generating neurons in the brainstem (Dorris et al. 2000).

Countermanding

The SSRT values we obtained for auditory, visual, and combined stop signals were consistent with other countermanding studies that have used a variety of movement types and conditions in humans with SSRT values ranging between 100 and 400 ms (Lappin and Ericksen 1966; Logan 1982, 1983; Zbrodoff and Logan 1986; Hanes and Carpenter 1999). Further, a previous study implementing a saccadic version of the countermanding task in humans found mean SSRT around 135 ms when using a foveal visual stop signal (Hanes and Carpenter 1999). This value is comparable with the mean SSRT of around 113 ms in the visual-only condition in the current study.

We found SSRT in the auditory-only condition to be significantly longer than SSRT to visual or combined stop signals without adjusting for the differences in afferent processing. The SSRT difference between the modalities was unaffected by practice: the latency difference was found on the 1st day of data collection and remained present throughout all sessions. Furthermore, the slope of the standardized inhibition function was less steep for auditory stop signals than for visual or combined stop signals (Fig. 5). Because the slope of the normalized stop-signal inhibition function provides a measure of inhibitory control (Logan and Cowan 1984), the differences in slope between auditory and either visual or combined stop signals suggests that separate inhibitory processes are utilized by visual versus nonvisual signals. The visual and combined-signal responses are probably controlled by the same inhibitory processes: the foveal, visual stimulus onset, and nonfoveal instructional information provided by the appearance of the visual stop signal. The auditory response however is controlled differently, probably by the instructional information only.

Previous studies have modeled countermanding as a simple race between a go process, initiated by the appearance of the target, and a stop process, initiated by the appearance of the stop signal (Logan and Cowan 1984; Hanes and Schall 1995; Hanes and Carpenter 1999). The go and stop processes have been modeled as a linear rise to a fixed threshold, with the threshold for the go and stop processes being independent. Altering the timing between appearance of the target and the stop signal handicaps this race to threshold. Our results suggest that, following the presentation of a stop signal, multiple stop processes may be initiated that depend upon the modality (and possibly location) of the stop signal. Each of these different stop processes may have a unique threshold and rate of rise. Therefore, interpretation and modeling of countermanding data based solely upon a single stop and go process is too simplistic. New models will be required that reflect these additional processes.

Possible mechanism for visual and auditory differences

Models of saccadic RT rely on a threshold mechanism for initiation of movement (Carpenter and Williams 1995; Kopecz 1995). Variations in RT are attributed to either changes in the rate of accumulation toward a threshold and/or variations in baseline activity at the time of target appearance. Using an oculomotor countermanding task, Hanes and Schall (1996) found that neurons in the primate frontal eye field have a fixed threshold of discharge rate that is achieved 10–20 ms before saccade initiation. Variability in RT was related to a variable rate of rise in discharge rate toward this threshold. More recently, Hanes and Paré (1998) have made similar observations for saccade-related neurons in the intermediate layers of the primate superior colliculus. These saccade-related neurons are organized into a motor map coding the direction and eccentricity of contraversive saccades (Robinson 1972), and they begin to discharge a high-frequency burst of action potentials approximately 20 ms prior to the initiation of a saccade (Sparks 1978; Munoz and Wurtz 1995). Most of these neurons also respond to the appearance of a visual stimulus in their receptive fields, with a phasic burst of action potentials some 50–70 ms after stimulus appearance (Mohler and Wurtz 1976; Munoz and Wurtz 1995). These same neurons are inhibited at similar latencies following the ap-

pearance of visual stimuli elsewhere in the ipsilateral and contralateral visual field (Everling et al. 1998; Olivier et al. 1999). The intermediate collicular layers are known to mediate strong, short-latency lateral inhibitory interactions (Meredith and Ramoa 1998; Munoz and Istvan 1998). Therefore, the sudden appearance of a visual stimulus on the fovea will elicit a robust phasic visual response among neurons with foveal response fields (Munoz and Wurtz 1993; Krauzlis et al. 1997), and this response will interact locally with the developing saccade signal via the lateral inhibitory network to delay or prevent saccade initiation. Such a bottom-up mechanism probably accounted for the reduced SSRT we observed with visual stop signals.

Many of the saccade-related neurons in the intermediate layers of the superior colliculus receive convergent auditory and visual inputs (Meredith and Stein 1986; Jay and Sparks 1987a, 1987b; Stein et al. 1993; Frens and van Opstal 1996). However, in experiments employing awake monkeys, the magnitude of the visual responses recorded from these neurons tends to be much greater than that of the auditory responses (Frens and van Opstal 1996). The onset of an auditory stimulus will elicit only a very weak response, if any at all, within the intermediate collicular layers, and any developing motor program will not be affected (Corneil and Munoz 1996). Therefore, in the case of the auditory stop signal, the auditory information must be first processed in higher centers before being relayed to preoculomotor areas such as the superior colliculus to prevent saccade initiation. This extra processing requires additional time and contributes to the increase in auditory SSRT.

Conclusions

We have demonstrated that visual stop signals are more effective in inhibiting the initiation of saccadic eye movements than auditory stop signals. We hypothesize that this advantage is mediated by robust phasic visual inputs to presaccadic brain regions such as the intermediate layers of the superior colliculus.

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