



Saccadic countermanding: a comparison of central and peripheral stop signals

K.N. Asrress, R.H.S. Carpenter *

The Physiological Laboratory, University of Cambridge, Cambridge CB2 3EG, UK

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Abstract

We compared the effectiveness of central and peripheral targets in a saccadic countermanding task. Stop-signal reaction times (SSRTs) do not differ significantly for central and peripheral stop signals. Further, when central and peripheral stop signals are presented together, SSRTs behave as expected of independent processes in parallel. A linear rise-to-threshold race model (LATER) with independent go and stop processes describes the behavioural data successfully, predicting not only the latency distribution of saccades that escaped inhibition, but also the probability of successful countermanding. Central and peripheral stop signals appear to act independently and with equal effectiveness. © 2001 Elsevier Science Ltd. All rights reserved.

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1. Introduction

1.1. The Countermanding Paradigm

A technique much used to investigate decision processes is the countermanding paradigm (Vince, 1948; Logan & Cowan, 1984; DeLong, Coles, & Logan, 1995), a reaction time task in which the subject must withhold the response if a stop-signal is presented. In an oculomotor version (Hanes & Carpenter, 1999), in control trials, a central target disappears, with simultaneous appearance of a more peripheral one, to which the subject makes a saccade. In stop trials, the central target reappears after a stop-signal delay: the subject then withholds the saccade, sometimes successfully and sometimes not (Fig. 1).

A simple interpretation of latency processes is to imagine that the stimulus leads to a steady rise in a decision signal until it reaches a threshold level at which action is initiated. In the LATER model, the rise is linear, its rate varying randomly from trial to trial, and the threshold is constant (Fig. 1A; Carpenter, 1981; Carpenter & Williams, 1995; Reddi & Carpenter, 2000).

Behaviour in the countermanding task can then be interpreted in terms of independent go and stop signals racing towards their thresholds (Fig. 1B and C: Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984; Logan, 1994). The observed randomness of countermanding success is then a consequence of the variation of rate of rise from trial to trial, and one may then predict not only the proportion of successes for different stop-signal delays but also the statistical distribution of saccadic latency in unsuccessful trials (Hanes & Carpenter, 1999).

1.2. Neurophysiology of countermanding saccades

A fundamental parameter that can be estimated using the countermanding paradigm is the average time for the stop signal to reach its threshold, the *stop-signal reaction time* (SSRT). At around 137 ms in humans (Hanes & Carpenter, 1999), it is rather shorter than ordinary latencies, and one might well expect preparation of a movement to be more time-consuming than merely cancelling one already prepared. However, a confounding factor must first be taken into account: in these experiments, the stop signal was foveal, whereas the go signal was peripheral. Centre and periphery have very different functional roles in both the frontal eye field (FEF) and superior colliculus (SC) (Munoz & Wurtz, 1993; Schall & Thompson, 1999). Fixation cells

* Corresponding author. Tel.: +44-1223-333886; fax: +44-1223-333840.

E-mail address: rhsc1@cam.ac.uk (R.H.S. Carpenter).

in the rostral pole of the monkey SC increase their tonic discharge during active fixation, with inhibitory effects on saccade-related cells in the rest of the SC (Munoz & Wurtz, 1993; Munoz & Istvan, 1998). Similar fixation-related activity has been identified in the FEF (Hanes, Patterson, & Schall, 1998; Schall & Thompson, 1999) and is enhanced in countermanding trials by the central stop signal. The timing of this response suggests that such cells could be directly involved in the countermanding itself, and thus likely to make SSRTs shorter than when the stop signal is peripheral. Could the marked difference between SSRTs and ordinary reaction time be due merely the former being evoked centrally and the latter peripherally? Investigation of this point — as urged by Schall and Thompson (1999) — was the aim of these experiments.

2. Methods

With informed consent, six participants (RC, male, age 54; AM, female, 20; KA, male, 21; MK, male 26; JMW, male, 21; JSW, male, 20) performed the countermanding task while their eye movements were recorded; the procedures had received local ethical committee approval. The stimuli were rectangular yellow diffuse

LEDs subtending 14×23 min arc presented with 100% contrast against a colour-matched background of uniform luminance 5.6 cd m^{-2} .

2.1. Paradigm 1: central and peripheral stop signals

All trials began with a warning tone and presentation of a central fixation target. After an interval uniformly randomly distributed in the range 0.5–1.5 s, this target disappeared, another appearing simultaneously at 3° randomly to the left or right. In *control* trials, this is all that happened; in *stop* trials an additional stop signal was presented after a delay, the *stop-signal delay*, δ (Fig. 2). The stop signal was either central (reappearance of the fixation spot) or peripheral (appearance of the opposite peripheral target). For each participant, two values of δ differing by 20 ms were chosen, such that cancellation for one or other was successful in approximately 50% of the stop trials: this enhances the reliability of the SSRT estimation (Hanes & Schall, 1995). Experiments were performed in blocks of 200 or 300 trials with rests as required; control, central and peripheral stop signal trials occurred randomly in the proportions 6:1:1, and each value of δ was selected at random.

Participants initially practised with at least 500 control trials. Then, it was explained that on some trials, a

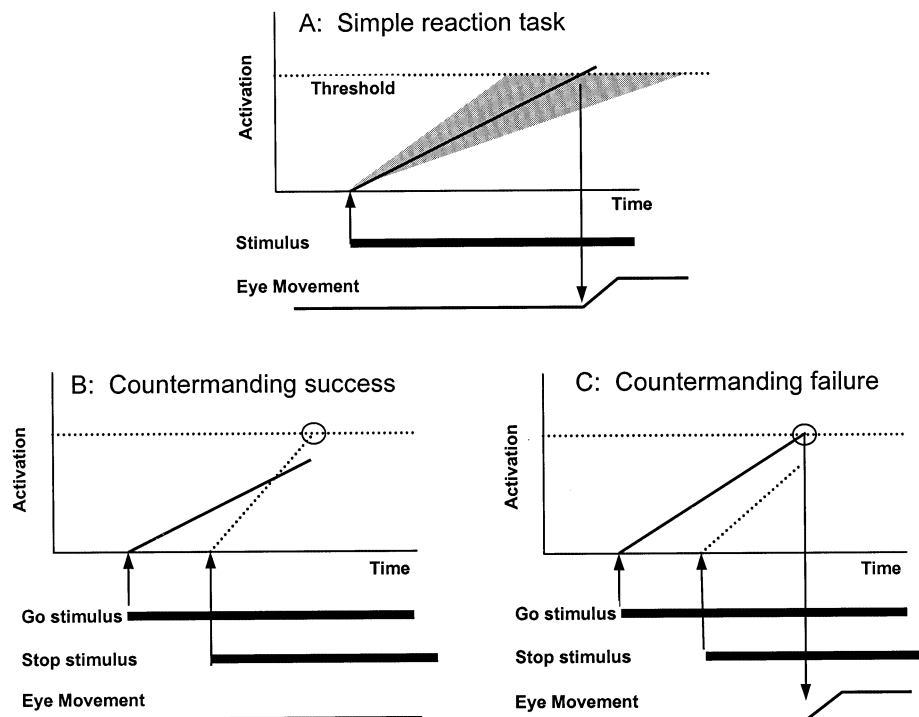


Fig. 1. Underlying model. (A) The LATER model proposes a decision signal that rises linearly in response to a single stimulus, initiating an eye movement when it reaches a given threshold level (dashed horizontal line); the rate of rise varies randomly from one trial to the next (shaded area). In a countermanding task (B and C), two such processes race against one another: a go process (solid line) and a stop process (dotted line). In countermanding success, the stop process rises faster than the go process and overtakes it and reaches threshold first, and the saccade is successfully inhibited (B). If the go process reaches threshold first (C), the saccade fails to be countermanded.

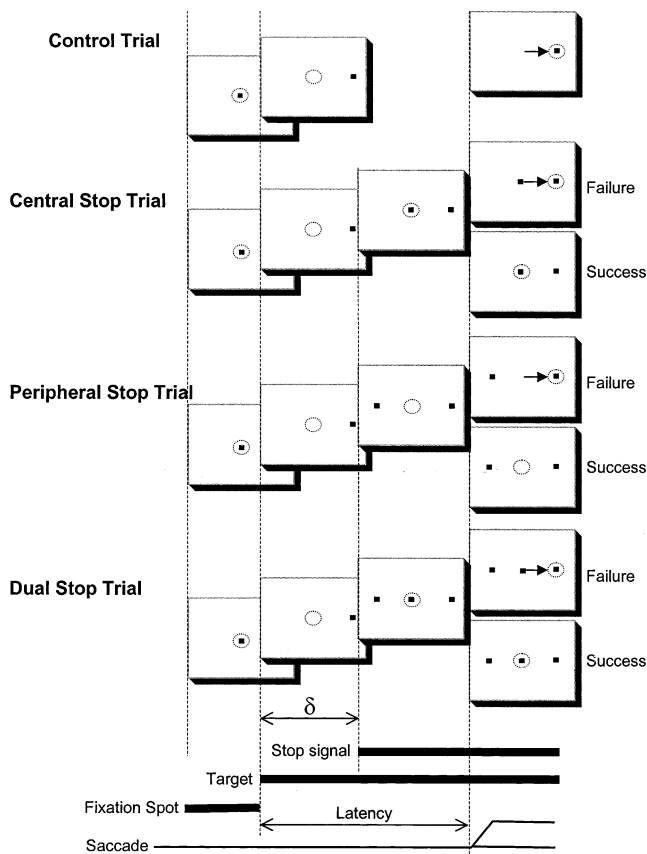


Fig. 2. Experimental paradigm for the countermanding tasks. The dotted circle indicates the focus of gaze at each interval, and the arrow indicates the saccade. For an explanation, see text.

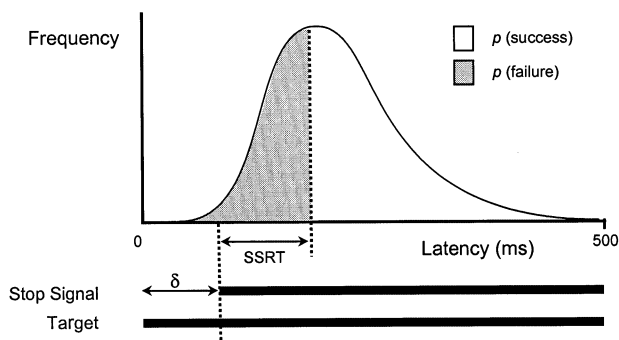


Fig. 3. Calculation of SSRT. The stop signal is delayed by δ and, after a further delay (the stop signal reaction time or SSRT), causes all saccades that would otherwise have occurred after that time to be cancelled. Consequently, of the total distribution of latencies seen in control trials (above), those lying in the shaded area will escape cancellation. Thus, the SSRT can be estimated by integrating the distribution until this proportion matches what is observed experimentally.

stop signal would appear, and the stop signals in the various paradigms were demonstrated. Participants were instructed to inhibit the saccade when the stop-signal occurred, but not to be concerned if they were unable to do so.

2.2. Paradigm 2: central, peripheral and dual stop signals

This was the same as Paradigm 1, except that a third, dual, stop signal was possible, in which the central and peripheral stop signals appeared at the same time (Fig. 2). The probabilities of control trials and each kind of stop trial were in the ratios 6:1:1:1. In this paradigm, a single value of δ was found to be sufficient, since a value generating roughly 50% countermanding success had already been estimated from the results of Paradigm 1.

2.3. Data collection and analysis

The output of the infrared oculometer (Carpenter, 1988) was sampled at 10 ms intervals by the PC-based saccadic analysis system SPIC (Carpenter, 1994), which controlled stimuli presentation, detected saccades in real time and displayed and stored the eye movement data. At the end of a series of blocks, the records were examined to eliminate those with blinks or other irregularities.

Using SPIC, Kolmogorov–Smirnov (K–S) one-sample tests (Kolmogorov, 1941) were performed to determine agreement between observed and theoretical distributions, and two-sample K–S tests to estimate the likelihood that two data sets come from the same distribution, and thus to confirm their compatibility before merging. Unless stated otherwise, significance levels for all statistical tests are taken as $P = 0.05$. Median latencies for eye movements in the two directions were routinely compared to check for significant left–right asymmetry, in which case the data for each eye were analysed separately.

SSRTs were estimated using the integral method (Hanes et al., 1998; Hanes & Carpenter, 1999). The control latency distribution is integrated from the time of target presentation, to determine the point where this integral, divided by the total area under the distribution, is equal to the proportion of non-cancelled trials at that stop signal delay. This point is then taken to represent the finishing line of the stop process, and thus equal to the stop signal delay (δ) plus the SSRT (Fig. 3). Although this method assumes that the SSRT is constant on each trial, violation of this assumption does not significantly alter the outcome of the analysis (Logan & Cowan, 1984; Hanes et al., 1998).

2.4. Simulations

Two different Monte Carlo simulations were performed using SPIC. The first consisted of the go process and a single stop process; the second had *two*

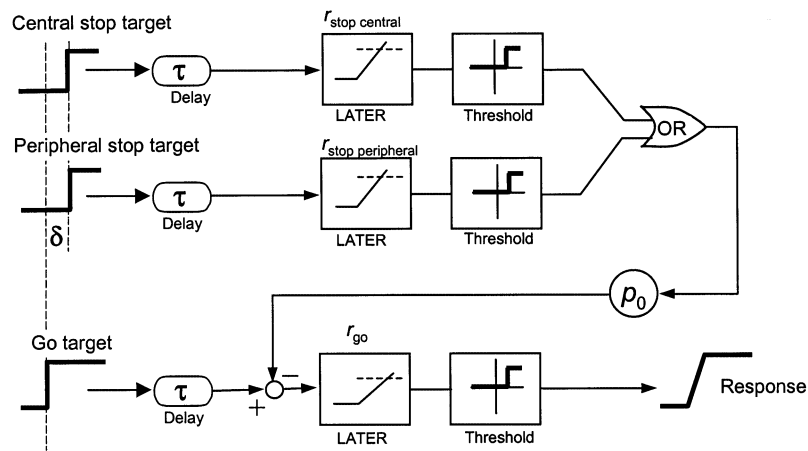


Fig. 4. Schematic representation of the simulation procedure for dual trials. The go signal (left) activates a LATER decision unit after a delay, τ , representing conduction and other necessary delays. When peripheral and central stop signals occur, after the stop-signal delay, δ , they similarly activate their corresponding LATER units. When either of the stop decision signals reaches its threshold, the input to the go decision unit is reduced by an amount p_0 . The first decision signal to reach threshold determines whether the movement is cancelled or not.

independent stop processes, modelling behaviour in dual trials (Fig. 4). In each simulated trial, stop and go processes race against one another until one reaches threshold. Values for the linear rise of each process, r_{go} and r_{stop} , are selected randomly from a pair of Gaussian populations of means μ_{go} and μ_{stop} and standard deviations σ_{go} and σ_{stop} (Hanes & Carpenter, 1999). The same procedure is used for the stop processes, but it begins after the stop signal delay, δ . A global transport delay, τ , is included for all processes, corresponding mostly to the latency of activation of central visual cells. Since such data are not available for humans, the value of 60 ms estimated for macaque was used (Goldberg & Wurtz, 1972; Hanes & Carpenter, 1999). As in Hanes and Carpenter (1999), the LATER model incorporated competition: arrival of the stop signal at its threshold resulted in inhibition, slowing the rate of rise of the go signal by an amount p_0 . In most cases, this cancels the saccade, but occasionally, it merely delays it. μ_{go} and σ_{go} were calculated from control distribu-

tions, and μ_{stop} from the SSRTs. σ_{stop} and p_0 were estimated to give the best fit to distributions.

3. Results

3.1. Paradigm 1: peripheral and central

A total of 7063 control, 1784 central stop and 1854 peripheral stop trials from all participants were analysed: the results are summarised in Table 1. For no participant was there any significant difference between SSRTs for the central and peripheral stop signals. Mean SSRT (\pm S.E.M.) over all participants was 129 ± 12 ms for centre and 128 ± 13 ms for periphery, again not a significant difference.

3.2. Paradigm 2

Table 2 summarises the results for each participant. For all but one, SSRTs in central trials were not significantly different from in peripheral. KA, JMW and JSW had SSRTs for dual trials that were not significantly faster than either for purely central or peripheral trials. For the other three participants, the SSRTs for central and dual trials were not significantly different. A possible reason could be that participants adopted a strategy of responding just to the central target, which appeared in the central as well as dual trials. To test this hypothesis, a subsidiary experiment was carried out on RC (for whom this phenomenon was most marked) where the proportions of the stop signals were changed so that this strategy would no longer be advantageous: the peripheral, central and dual stop signal appeared in the proportions 2:1:1. Mean SSRTs for the central and peripheral trials were then 139 ± 3 and 143 ± 3 ms, respectively, and not

Table 1
Number of trials and SSRTs for each subject in the two conditions of Paradigm 1

	Number of trials	SSRT (ms)	
		Central stop signal	Peripheral stop signal
RC	2239	134 ± 2	136 ± 3
AM	1771	134 ± 5	132 ± 5
MK	1780	149 ± 5	148 ± 5
KA	1703	124 ± 4	122 ± 3
JMW	1303	130 ± 5	127 ± 5
JSW	1905	101 ± 7	101 ± 7
Mean		129 ± 12	128 ± 13

Table 2
Number of trials, SSRTs, and significant differences (*t*-test; $P < 0.05$) for the different conditions in Paradigm 2: C (central), P (peripheral), D (dual)

	Number of trials	SSRT (ms)			Differences significant at 5%
		Central stop signal	Peripheral stop signal	Dual	
RC	1895	134 ± 3	144 ± 3	127 ± 5	P&D
AM	1656	132 ± 6	109 ± 5	118 ± 4	C&P
MK	1810	125 ± 5	133 ± 5	122 ± 5	
KA	1168	128 ± 4	124 ± 3	114 ± 3	C&D; P&D
JMW	1783	138 ± 3	132 ± 3	123 ± 3	C&D; P&D
JSW	1192	109 ± 6	115 ± 5	94 ± 5	C&D

Table 3
Values for the parameters used in the simulations (where not stated, units are in Hz for unit threshold)

Subject	μ_{go}	σ_{go}	μ_{stop}	σ_{stop}	Central or peripheral	δ (ms)	p_0	$P(\text{escape})_{\text{predicted}}$	$P(\text{escape})_{\text{observed}}$
RC	6.6	0.53	13.4	7.32	C	70	704 ± 3	0.44 ± 0.04	0.39 ± 0.04
						90	704 ± 34	0.60 ± 0.04	0.67 ± 0.04
					P	70	704 ± 7	0.44 ± 0.04	0.44 ± 0.04
						90	704 ± 4	0.60 ± 0.04	0.67 ± 0.04
KA	7.5	0.87	15.9	6.80	C	50	925 ± 20	0.26 ± 0.03	0.32 ± 0.04
						70	925 ± 5	0.46 ± 0.03	0.45 ± 0.04
					P	50	925 ± 40	0.26 ± 0.03	0.18 ± 0.03
						70	925 ± 35	0.46 ± 0.03	0.52 ± 0.04
JMW	6.8	0.60	14.6	4.56	C	70	746 ± 20	0.43 ± 0.03	0.52 ± 0.05
						90	746 ± 17	0.59 ± 0.03	0.57 ± 0.05
					P	70	746 ± 36	0.43 ± 0.03	0.48 ± 0.05
						90	746 ± 6	0.59 ± 0.03	0.52 ± 0.05
JSW	4.8	1.00	24.4	15.5	C	140	858 ± 4	0.33 ± 0.04	0.42 ± 0.04
						160	858 ± 2	0.47 ± 0.04	0.51 ± 0.04
					P	140	858 ± 2	0.33 ± 0.04	0.41 ± 0.04
						160	858 ^a	0.47 ± 0.04	0.51 ± 0.04
RC	6.45	0.49	12.4 (C) 12.4 (P)	7.32	D	80	704 ± 16	0.24 ± 0.03	0.14 ± 0.04
KA	8.07	0.82	15.2 (C) 15.2(P)	6.80	D	70	925 ± 30	0.43 ± 0.03	0.50 ± 0.04
JMW	6.80	0.61	13.5 (C) 13.5 (P)	4.56	D	70	746 ± 18	0.23 ± 0.03	0.23 ± 0.04
JSW	5.05	0.85	21.0 (C) 18.2 (P)	15.5	D	140	858 ± 39	0.31 ± 0.03	0.32 ± 0.04

^a Not compatible with observed distribution (K–S; $P < 0.05$).

significantly different. For dual trials, the mean SSRT was 120 ± 5 ms, significantly faster than each of the separate processes. That this alteration in SSRT was not simply a direct result of changing the proportions was demonstrated by carrying out the same experiment on another subject who did not show this aberrant bias. Two subjects generated significantly bimodal distributions; this was felt to preclude further quantitative analysis or simulations, since it suggests the possibility of more than one underlying generative mechanism.

3.3. Simulations: Paradigm 1

Table 3 gives the values of the parameters providing the best fit between simulations and observations for

the four participants whose distributions were not bimodal. For all but one, at every stop signal delay, the predicted and actual distributions were statistically indistinguishable. Furthermore, the predicted and observed probabilities of a saccade escaping from inhibition were not significantly different. The values of μ_{go} , σ_{go} and μ_{stop} are very similar to those estimated by Hanes and Carpenter (1999) who used only a central stop signal, and the values of σ_{stop} somewhat larger but not significantly so.

3.4. Simulations: Paradigm 2

The parameters used were all calculated as before (Table 3: any variation of parameters between the two

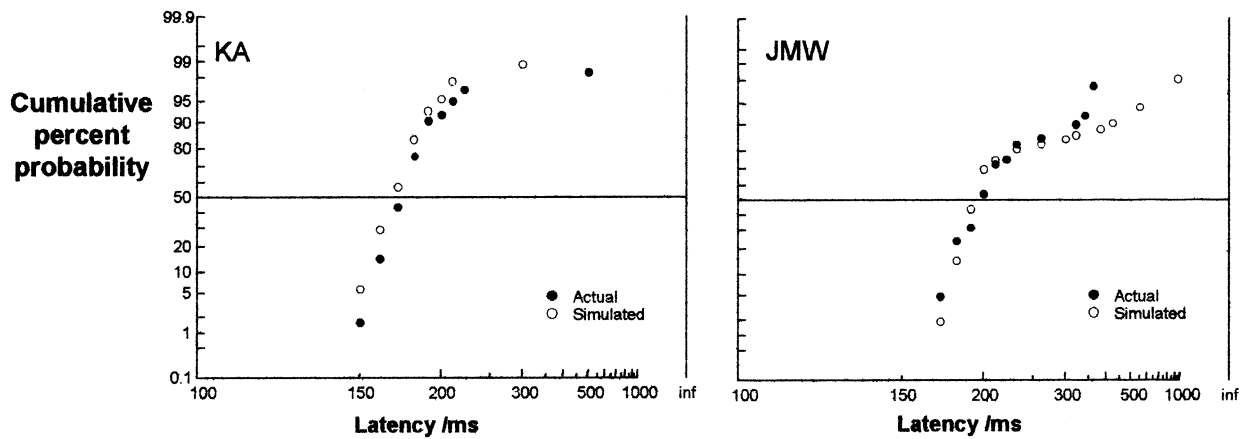


Fig. 5. Actual and simulated latency distributions of saccades that escaped inhibition in dual trials. Simulations used a modified LATER model of two parallel stop processes independently rising to threshold and partially inhibiting the go process.

halves is because the estimates are from different experiments, done on different days). All simulations were compatible with the actual distributions. The reciprobbit plots in Fig. 5 of predicted and actual distributions show how the incorporation of lateral inhibition, in addition to helping to explain more obvious aspects of the behaviour such as medians, can also help explain what is otherwise puzzling, the peculiarities that can often be seen at longer latencies in countermanding trials: similar effects were noted in Hanes and Carpenter (1999).

4. Discussion

The first experiment showed that for all six participants, SSRTs are not significantly different for centre and periphery, nor significantly different from the estimates by Hanes and Carpenter (1999) that used a central stop signal. In the second experiment, having both central and peripheral stop signals in the same trial appeared to shorten the SSRT. This suggests that the central and peripheral stop signals have independent inputs to the stopping process, and simulating probability summation of this kind provided a good description of the observed data. This suggests quite strongly, and contrary to the expectations expressed by Schall and Thompson (1999), that countermanding is not primarily a function of a specialised region of the FEF or colliculus. Rather, it seems that information from different parts of the visual field is equally effective in countermanding.

The fact that different countermanding tasks, involving for example, speech and key presses as well as eye movements, can all be stopped in about 200 ms (Logan & Cowan, 1984), similarly suggests that countermanding is an amodal, central process that can be activated with more or less equal facility in different ways. This

question could — as Schall & Thompson (1999) suggest — be investigated by experiments in which the modalities of the stop and go signals are different, to provide evidence for or against the ‘global mechanism for stopping’ hypothesis and to test the applicability of LATER to reaction-time tasks in general.

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