Biocybernetics and Biomedical Engineering
2012, Volume 32, Number 2, pp. 33–47

Saccadometry and Movement Inhibition

TYMOTHÉE POITOU, PIERRE POUGET*

Université Pierre et Marie Curie-CNRS-INSERM, ICM, Equipe Neurologie et Thérapeutique Expérimentale, Hôpital de la Salpêtrière, Paris, France

To reveal some functional constraints of the saccade inhibitory neuronal circuits, we investigated the influence of response monitoring in human. The subjects were instructed to perform a stop signal task in which the probability of stop trial occurrence was manipulated. The purpose of the work was to evaluate the time course necessary to adapt the behavior to changes in the occurrence of stop signal. Our results show that humans are capable to spatially monitor the relative probability event of stopping and to finely and quickly modulate their ability to inhibit a response. These results have important consequence to apprehend pathologies in which, an inaccurate control of inhibitory process results in a loss of fundamental capability of behavioral adaptation.

K e y w o r d s: saccade, countermanding, spatial inhibition

1. Introduction

"Work on saccadic reaction times in humans seems to suggest that the brain runs a kind of race between signals representing different possible targets, with more probable targets starting nearer the finishing post than less probable ones. There is also a huge random element, rather like a gratuitous random handicap, so that reaction times are very variable even when the stimuli and conditions are absolutely constant. This may well represent a deliberate mechanism for making sure our behaviour is not too predictable by our predators (and you may like to think of it as the neural mechanism behind our illusion of 'free will'."

Roger Carpenter. (http://babylon.acad.cai.cam.ac.uk/people/rhsc/oculo.html).

* Correspondence to: Pierre Pouget, Université Pierre et Marie Curie-CNRS-INSERM, ICM, Equipe Neurologie et Thérapeutique Expérimentale, Hôpital de la Salpêtrière, 47 boulevard de l'Hôpital, 75651 Paris Cedex 13, France, e-mail: pierre.pouget@upmc.fr
Received 02 August 2011; accepted 14 February 2012

This quote has been extracted from Roger Carpenter's website. For a lot of different reasons, these few sentences have been and still keep inspiring me. A fundamental aspect of this reasoning is reflected in a few words by its ability to organize, to link and to fall in the distinctions between notions such as behavior, computation and biology. As mentioned by Roger Carpenter, the race between horses has been used to illustrate other races that are supposed to take place in the brain among processes leading to decision and motor response. According to this hypothesis, scientists have suggested that the decision: the choice of action will depend on the result of a race. To study the processes supposedly referring to that race, the measurement of reaction time distributions and variability using saccadic eye movements possess important

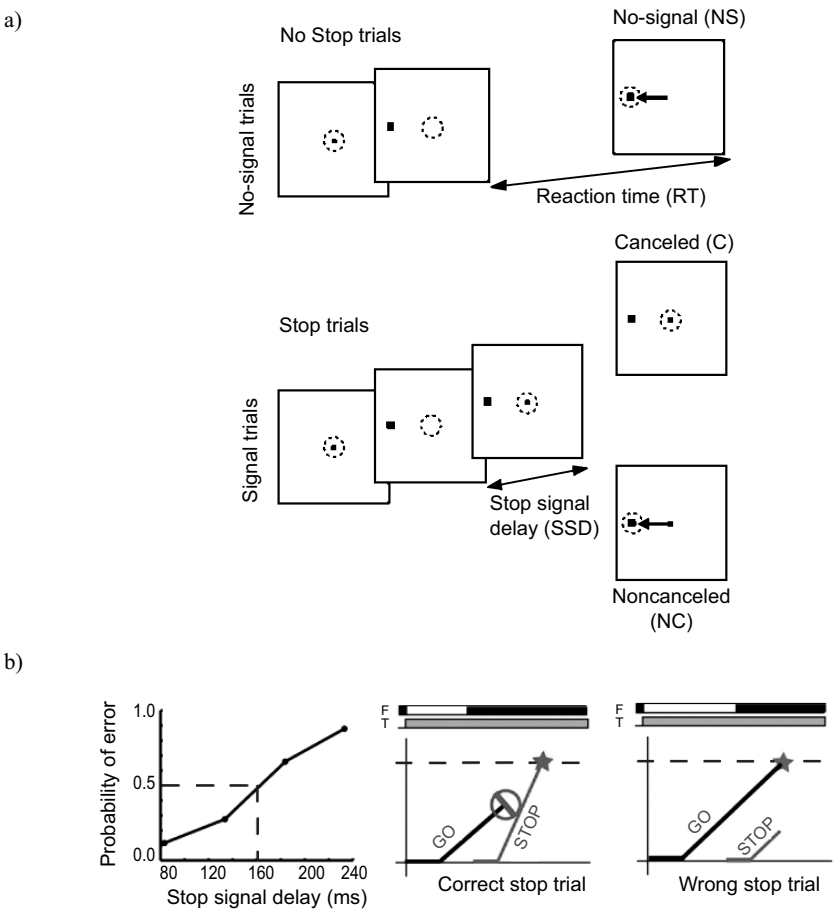

Fig. 1. Countermanding task. a) NoStop trial. Subjects are instructed to trigger as fast as possible a saccade towards a lateral target. b) Stop trial, in 30%, 50% or 70% of the trials (depending on the experimental conditions) a stop signal consisting in the re-illumination of the fixation target, occurred after a variable delay and instructed the subject to withhold the response they were about to trigger

advantages. First, in a few minutes hundreds of latencies can be acquired. Second, even in very young passive or old subjects the latencies of saccades can be recorded using non-invasive techniques. This is probably two of the reasons why for decade or so, researchers, engineers as well as medical doctors have used saccadometry for quantitative measures of cerebral performance. Saccadometry has been leading to improved measures of disease progression and diagnosis. The current areas of application include neurodegenerative disorders (Parkinson's and Huntington's diseases), population studies in relation to intelligence and personality, schizophrenia, and recently the effects of deep brain stimulation of the subthalamic nucleus. Aside these studies of movement execution, in everyday situations we are also often required to stop a prepared or ongoing action. As for the response time, the time required to stop a prepared movement can be precisely studied experimentally. The stop signal or the countermanding paradigm, which includes both a task (Fig. 1a) and a theoretical construct (Fig. 1b), was developed to investigate the control of action [1]. In an oculomotor version of countermanding task, subjects are instructed that if a stop signal occurs shortly after appearance a visual target, they have to maintain fixation rather than make a saccade to the target [2–12]. Prevailing theoretical models posit that performance in the countermanding task entails a race between two noisy processes (Fig. 1b), associated with a "go" and a "stop" responses [13].

The purpose of the work presented in that seminar was 1 – to evaluate the behavioral adjustments induced by a large range of occurrence of the stop signals in the context of a saccade countermanding paradigm and 2 – to determine the time course necessary to adapt the behavior to changes in the occurrence of stop signal.

2. Methods

Data were collected from 5 human subjects and 1 male rhesus monkey.

2.1. Human Data Collection

Human data were collected from 5 subjects (3 males (31years +/–7) and 2 females (23years +/–1)), using similar paradigms. Each subject participated in a minimum of 20 (maximum of 32) sessions (45 minutes each). All subjects reported having normal or corrected-to-normal vision. The volume of data from 1 subject was insufficient to provide sufficient statistical power for the comparisons examined below and could not contribute to all of the analyses. Eye position was monitored and sampled at 500 Hz using an SMI eye tracker (Sensomotoric Instruments, Germany). All countermanding trials began with the presentation of a central fixation target (fuchsia square of 0.5° visual angle). After a random delay (500–1500 msec) the fixation target went off and a lateral green target (0.5° square) appeared at a right or left 16° horizontal location. The subjects were instructed to trigger as fast as possible a saccade towards this

lateral target. In 30%, 50% or 70% of the trials (depending on the experimental conditions) the stop signal consisting in the re-illumination of the fixation target, occurred after a variable delay and instructed the subject to withhold the response they were about to trigger. The subjects were initially informed that they would be unable to inhibit approximately half of the stop signal trials. In order to obtain an inhibition function with probabilities ranging between 0.25 to 0.75, the stop signal delays ranged from 50 to 350 msec in 75 msec steps for 20% condition, from 50 to 450 msec in 100 msec steps for 40% condition, from 100 to 500 msec in 100 msec steps for 60% condition and from 150 to 650 msec in 125 msec steps for 80% condition. Each delay occurred with equal probability for each eccentric target. A session consisted in 300 trials.

2.2. Macaque Data Collection

Data were collected from 1 male rhesus monkey (*Macaca mulatta*; 16 kg). The maintenance of the monkey, all surgical procedures and the experimental protocols were carried out in strict accordance with the National Institutes of Health guidelines (1996) and the recommendations of the EEC (86/609) and the French National Committee (87/848). Prior to participating in the current study, the animal was periodically chaired, head-posted and trained to perform a series of tasks for a period of 6–12 months, until he became a regular and proficient performer.

The experiments were under computer control to present stimuli, record eye movements, and deliver reinforcement. Detailed descriptions of the behavioral training and tasks and the methods used to collect these data have been described in detail [14, 15]. Eye position was monitored using an ASL eye tracker (Applied science laboratory, USA) at a sampling rate of 240 Hz. The saccades were detected offline using conventional velocity and acceleration criteria. The animal was tested for approximately 3 hours a day, 5 days a week. During testing, water was given as the positive reinforcement. Access to water in the home cage was controlled and monitored. Fluids were supplemented as needed.

The countermanding task is illustrated in Fig. 1. All trials began when the monkey fixated a central fixation target for a variable interval (500–800 msec). The fixation stimulus was then extinguished and a peripheral target was presented at one of two possible right or left 12° locations, cuing the monkey to make a single saccade to the target. In the no stop signal trials, the monkey was reinforced for making the saccade within 100–700 msec to the target and fixating the target for 500–700 msec. In the stop signal trials, the central fixation target reappeared after a delay, referred to as the stop signal delay, instructing the monkey to inhibit the saccade initiation. This happened on 10–80% of the trials, depending on the block design condition. Two outcomes were possible on the stop signal trials; the monkey could either make the saccade (known as a noncancelled or nostop-signal-respond trial) or not (known as cancelled or signal-inhibit trials). The monkey was reinforced for maintaining fixa-

tion on the stop signal for 600–700 msec after the stop signal appeared. The saccade to the target on the stop signal trial was incorrect, not reinforced, and resulted in a 1500 msec timeout. In order to obtain an inhibition function with probabilities ranging from 0.25 to 0.75, the Stop Signal Delays (SSDs) ranged from 25 to 450 msec and remain constant within an individual session. Behavioral and neurophysiological data from these monkeys has appeared in previous publications [7, 11, 12, 16]. As for the human data collection, the behavioral data were obtained from monkey Y performing the saccade countermanding when the proportion of the stop signal trials was varied between 0.2 and 0.8 from session to session. A session consisted of approximately 1500 trials.

2.3. Primary Data Analysis

Behavioral data from the countermanding task consist of several parameters including distribution of the response times on trials with the no stop signal, distribution of the response times on the noncancelled trials, and probability of the responding as a function of the stop signal delay (SSD) [17]. The later provides the inhibition function. At the shortest SSD almost all saccades are cancelled, whereas at the longest SSD almost all saccades are not cancelled. In order to extract measures of the inhibition function, inhibition values at all SSDs were fit with a cumulative Weibull function of the form, $W(t) = \gamma - (\gamma - \delta) \cdot \exp(-(t/\alpha)\beta)$, where t is the time after target presentation, α is the time at which the inhibition function reaches 64% of its maximum value, β is the slope, and γ and δ are the maximum and minimum of the inhibition function, respectively.

The saccades were detected using an algorithm that detects the first significantly elevated velocity ($>30°$/s) using digital differentiation. The saccade initiation and termination were defined as the beginning and end of monotonic change in the eye position before and after the high velocity gaze shift. The trials during which the saccades were initiated after the target was presented while the monkey was fixating the central target and terminated on the target were classified as the valid trials. For each valid trial, the response time was the interval from the target presentation to the saccade initiation. The mean response time for each subject is the mean of session means and the standard error is the mean of the standard errors across sessions.

For each behavioral session, an estimate of the SSRT was determined from distribution of the response times on the no stop signal trials and the inhibition function. The SSRT can be estimated in at least two ways [18]. The first method assumes that it is a random variable. Logan and Cowan [13] showed that the mean SSRT is equal to the difference between the mean reaction time during the no stop signal trials and the mean value of the inhibition function. The second method of estimating the SSRT assumes that it is constant. By this method, the SSRT is estimated by integrating the no stop signal saccade response time distribution, beginning at the time of target presentation, until the integral equals the proportion of noncancelled trials at that

SSD. Detailed descriptions of these methods have appeared previously [8, 13, 19]. In practice, these two methods rarely give identical values of the SSRT because of noise and unavoidable measurement error. However, if enough trials are collected, then there is no reason to weight one method more than another [19]. Therefore, we identified a single estimate of the SSRT from the behavioral data collected during each session by averaging the SSRT estimates derived from both methods [7, 9].

3. Results

3.1. Adjustment to Manipulation of Stop Signal Probability

To determine if the global proportion of stop signal trials affects both the response time and the probability of responding, the behavioral data were obtained from one male macaque monkey and from 5 adult human subjects while systematically varying the fraction of stop signal trials between 0.3, 0.5 and 0.8 for the monkey and between 0.3, 0.5 and 0.7 across the sessions for each day of testing in the humans.

3.1.1. Effect on Ability to Inhibit

Monkey Y performed 28 sessions of the countermanding task over the course of 4 months. We observed a shift of the inhibition functions for 80% condition in comparison to 30 and 50 percent condition: the ability to inhibit the saccade increased with the percent of stop, resulting in a decreased percent of errors at the stop trials. To quantify the adjustment in the performance, the estimated delay at which the inhibition function reaches 50 percent of error was extracted for each experimental condition. As shown in Fig. 2a for an individual session, this delay increased with the manipulation of global stop signal probabilities reflecting adjustment of the behavior by increasing the reaction times and shortening the stop signal reaction times. As shown in Fig. 3a across the sessions, this delay reached 160 msec for the sessions with 30% of stop trials (4 sessions), 142 msec for 50% (2 sessions), and 211ms for 80% (4 sessions). As shown in Fig. 4b, a significant increase between 30 percent and 80 percent conditions was observed ($p = 0.0079$).

As illustrated in a single exemplary session (Fig. 2b), the similar adjustments of the performance were observed for the human subjects. On average five human subjects performed at least 20 sessions of the saccade countermanding over the course of 11 months (for 3 subjects, 6 extra sessions were needed to reach the standard quality needed for a proper estimation of the SSRT – see method section). A shift of the 3 inhibitions functions was observed (Fig. 3b), reflecting an increased inhibitory ability while increasing percentage of the stop signals. In order to quantify these behavioral adjustments, the estimated delay at which the inhibition function reaches 50 percent of error was extracted for each experimental condition. As shown in Fig. 4b, this delay increased with the manipulation of global stop signal probabilities.

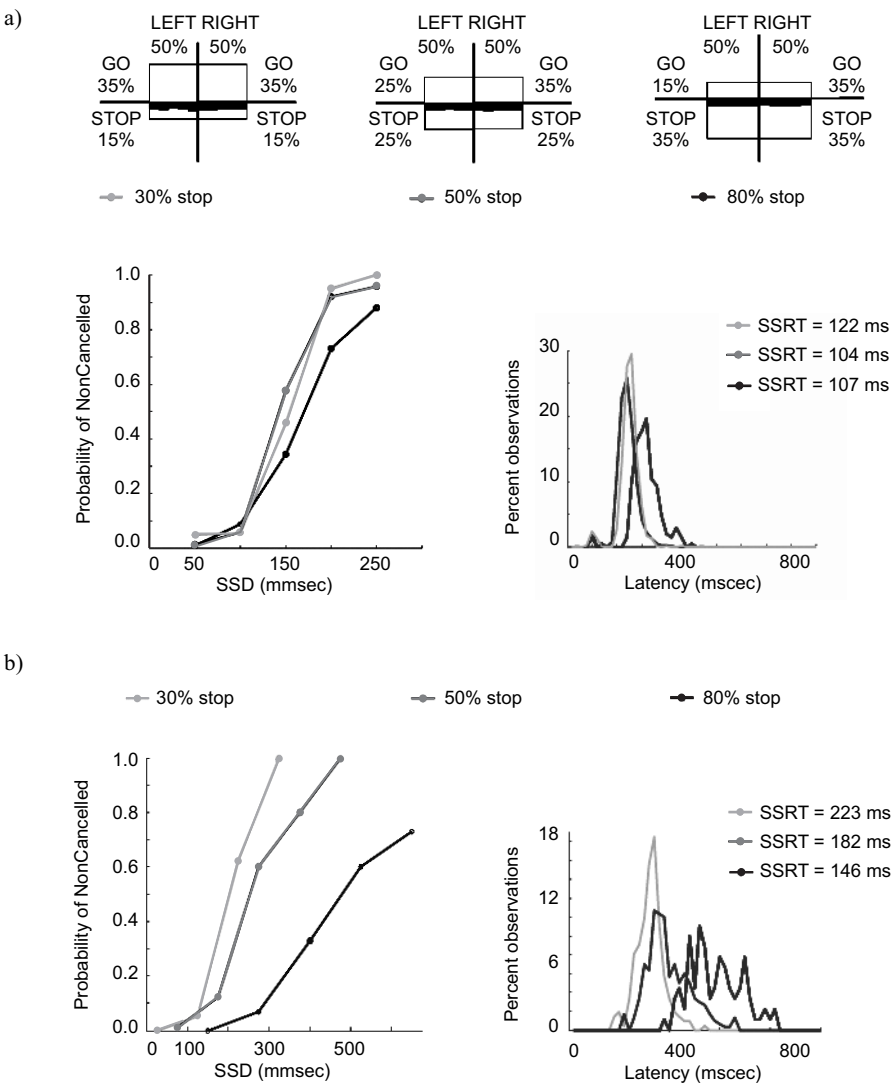

Fig. 2. a) Monkey single individual session. Left panel: inhibition functions as function of stop fraction manipulations, 30% (in light grey), 50% (grey) and 80% (black). Right panel: Latencies distributions as function of stop fraction manipulations, 30% (in light grey), 50% (grey) and 80% (black). b) Bottom panel: Human single individual session (same conventions as top panels)

For the humans, this delay reached 245 msec for 30% of stop, slightly increased (292 msec) for 50% of stop, and reached 389 msec for the 70% stop conditions. We found a significant increase between 30 percent and 70 percent conditions ($p = 0.0317$).

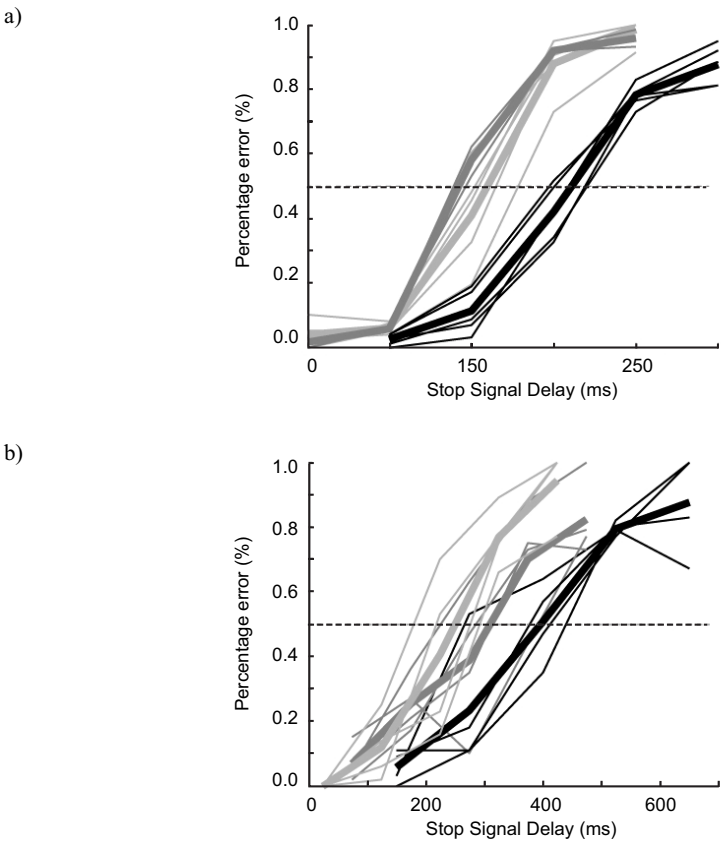

Fig. 3. a) Grand average monkey inhibition functions, 30% (in light grey), 50% (grey) and 80% (black). Thin lines represent each individual session (n = 4). Thick lines represent average across sessions for each stop condition. b) Same conventions as top panel: grand average inhibition functions for human subjects. For each condition, all the 4 subjects (thin line) and mean curve (thick line) are represented

3.1.2. Effect on Latencies

For the monkey, for the no stop trials significant shifts in the response time trials were found in the response to changes in the global stop fraction ($p < 0.05$). As expected, the latencies of no stop signal trials were modulated by probability of the stop trials, i.e. the latencies were longer with 80 percent of stop compared to the 30 percent condition (respectively 254.5 msec and 246.25 msec for the 30 and 50% stop trials conditions and 307.5 msec for the 80% ones). As shown in Figure 4a, a significant increase between 30 percent and 70 percent conditions was also observed ($p < 0.001$). As observed in the monkey, the human latencies of no stop signal trials were modulated by stops probabilities, i.e. the latencies were longer for 70% stop trials conditions compared to 30% ones with respectively 349 msec and 424 msec

average latencies for the 30 and 50% stop trials conditions and 501 msec average latencies for the 70% condition (Fig. 4c). A significant decrease was found between 50 percent and 30 percent conditions ($p < 0.001$) as well as a significant increase between 50 percent and 70 percent conditions ($p < 0.001$).

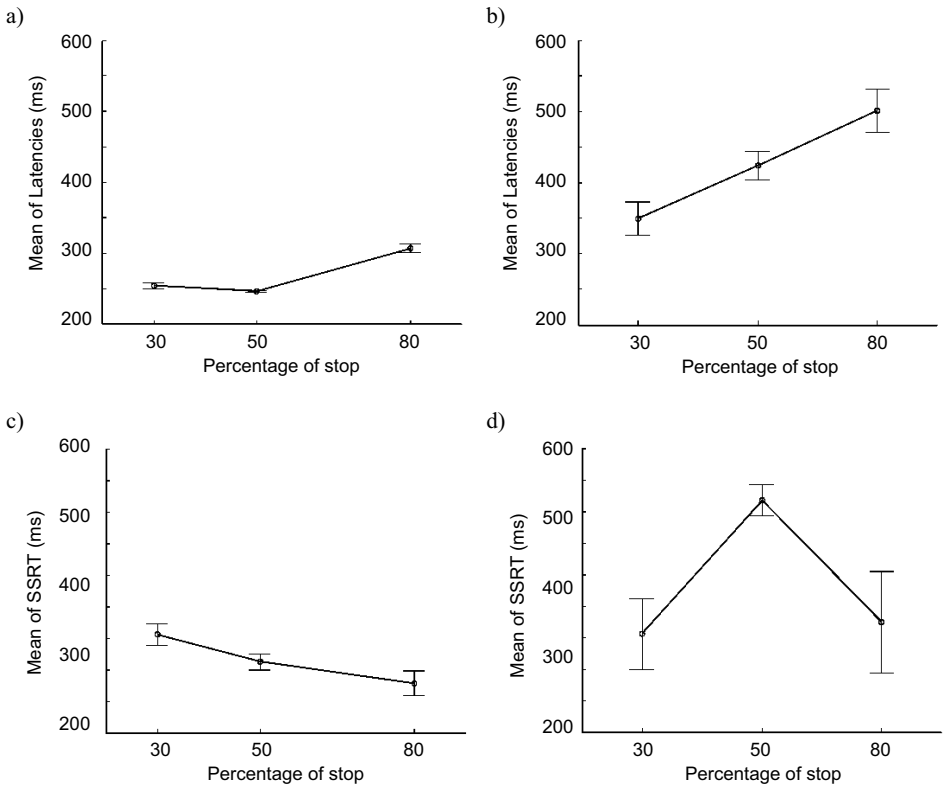

Fig. 4. a-c) Grand average monkey latencies (top panel) and Stop-Signal-Reaction-Times (SSRTs) as function of stop trials conditions (30, 50 and 80% percent of stop). b-d) Grand average for human subjects, same convention as for monkey data (30, 50 and 70% percent of stop)

3.1.3. Effect on SSRT

As shown in Figure 4b, in the monkey, the results suggest that the SSRT estimates were negatively correlated with the modulations of the stop trials probabilities. More specifically, the SSRT mean value was 107.5 msec and 103 msec respectively for 30 and 50% of the stop trials and 98.5 msec for the 80% stop trials conditions. A significant increase was found between 30 percent and 80 percent conditions ($p = 0.0018$). We did not observe any significant variations of the SSRT in the humans ($p > 0.4$) (Fig. 4d).

3.2. Time Course and Stop Signal Probability Tracking

As shown in Figure 5 for all 5 subjects, compared to 50% stop trials condition, rapid increase in the latencies is observed for conditions with larger probability of the stop trials (80%) while rapid decrease of response times is observed for the low stop probability conditions (30%). To assess the time course of latencies variations, a Bonferoni corrected t-test was used, in series of sliding windows covering 15 consecutive trials

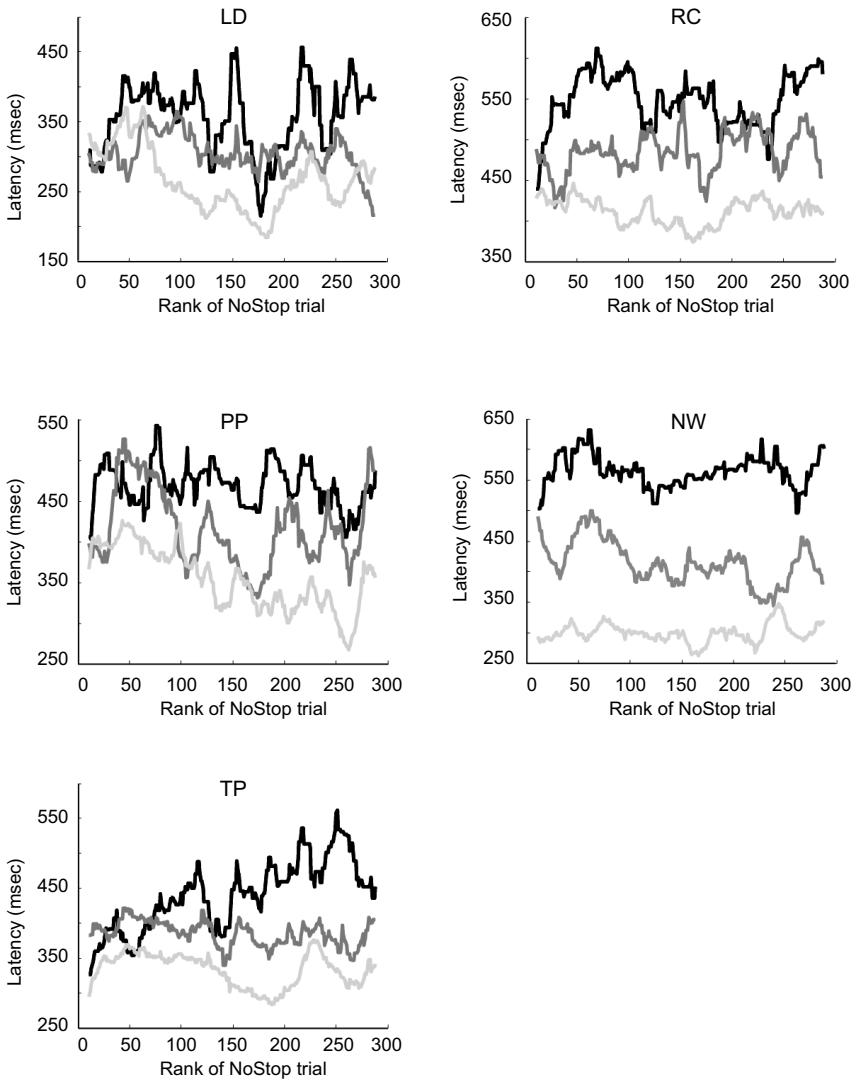

Fig. 5. Grand average time course of saccade latencies during the experimental session. Average No-stop trial latencies are represented using a sliding moving window (15 trials, step of 1 trial) as function of stop fraction manipulations, 30% (in light grey), 50% (grey) and 80% (black)

and moved at a step of 1 trial. On average across all subjects, in session associated with respective 80% versus 50 % and 30% versus 50% stop trials conditions, a significant difference between the saccade latencies appeared respectively after 22 and 75 trials for 80 vs 50 and 30 vs 50 stop trials conditions. Similarly to the monkey, a significant difference between the saccade latencies appeared respectively after 56 and 221 trials for 80 vs 50 and 30 vs 50 stop trials conditions.

4. Discussion

Based on the race between a "go" and a "stop" process, Logan & Cowan [13] demonstrated that the time needed to cancel a movement, the stop signal reaction time (SSRT), can be estimated from distribution of the response times when a no stop signal is presented and the probability of responding given that the stop signal occurred. This race model has been implemented in a linear rise to a threshold model framework [6] and in a network of interacting units with delayed potent inhibition [20, 21]. In addition to trial-to-trial variations in response time [9, 22–26] but see Nelson [27], human subjects increase response times with increases in the global fraction of stop signal trials, and these changes in response times are accompanied by changes in the probability of responding [28–30]. In the race model framework, accounting for any of these behavioral adjustments would require an adjustment of the "go" and/or "stop" processes. In the present study we investigated the time course selectivity of movement inhibition in human and non-human primates performing a saccade-countermanding task in which a large range of global probability of stop trial occurrence was manipulated. The results show that human and non-human primates are capable of monitoring the relative probability of stopping events. Furthermore, this work shows that a delay is required for both stop signal reaction times and ordinary response times to be modulated according to the stop probability.

Beside the few studies on expectancy and inhibition in the literature, Hanes and Carpenter [6] addressed the issue of a change in Go-latency distribution with a manipulation of the primary saccadic task in a countermanding paradigm: the saccade target was made more or less detectable by changing its contrast. This manipulation resulted in a decrease for high contrast targets or increase for low contrast targets of the Go trial reaction times. They also observed that the percentage of the NoGo escapes changes across the two conditions in exactly the same way that is predicted by the horse race model if the stop process is unchanged while the Go process is facilitated or inhibited. More precisely the subjects failed to inhibit saccadic movements after stop signal more often in the high contrast target condition compared to the low contrast target condition. Correspondingly the SSRT values computed in the two conditions were not statistically different. A discrepancy between our results and those of Hanes and Carpenter [6] who did not reveal any modulation of the stop process concomitant with the modulation of the Go process, is that their

manipulation was affecting the component of saccadic latency which is due to initial perceptual processing while our manipulation was likely more effective at the motor preparation and the decisional stages. An alternative interpretation of this finding is that the probability-bias effect is actually an attention effect. In other words we might hypothesize that the higher probability of the stop-signal occurrence on one condition facilitates the initial, attentional component of the sensorimotor process leading to the inhibition of saccade, the detection and discrimination of the stop-signal visual stimulus. In this event the probability bias could affect the inhibitory process in a similar way to the reduction of stop signal reaction time in the typical countermanding task, because the perceptual discrimination needed to distinguish a Stop (vs a NoStop) command would be facilitated thus shortened by the availability of attentional resources. Since the process of acquiring of perceptual information about target location affects the Go process in much the same way as the stop process, the attentional hypothesis could at once explain the decrease of latency in the Go trials and the decrease in the SSRT.

Possible neurophysiological bases:

The neuronal substrate of monitoring and adjustment of performance in the saccade-countermanding task is not fully understood. The saccade-related neural activity has been studied in detail by means of electrophysiological recordings and anatomical studies in several brain areas of non-human primates [14, 31]. In two of the most relevant areas implicated in the saccadic control – the superior colliculus and the frontal eye fields, researchers have also reported a neural activity correlated with the saccade inhibition. For instance, Paré and Hanes [11] have found that the relative activation of movement-related and fixation-related cells in the superior colliculus (SC) is predictive of the behavioral outcome (success or failure) in a saccadic countermanding task. Interestingly, by using a complex visuo-oculomotor task, Schall and colleagues [32] have reported that during the No-go trials in which a saccade was correctly inhibited some cells in the monkey FEF revealed a selective activation coherent with a motor preparation toward a location opposite to the visual stimulus instructing to maintain fixation. Although the experimental conditions between the present and Schall's study are not directly comparable, these findings are qualitatively in agreement with our behavioral observations concerning the specificity of inhibitory control. A different but also very interesting idea has recently been put forward by Goffart and colleagues [33]. According to these authors the SC fixation cells would actually represent position error vectors (in other terms they would also code for small saccades) and the stability of the gaze would be the result of a dynamic equilibrium between small saccades programmed towards all directions. In addition Munoz and Istvan [34] have studied in detail the mutual inhibitory interaction in the deep layers of the SC and they have pointed out that the activity of neurons in the so-called fixation zone seems to map a spatial continuum of the parafoveal area in the visual field.

In this sense the fixation-movement competition could also endorse a side-specific component. Therefore, one could speculate that the Go-Stop competition is actually translated into a directional push-pull mechanism in the superior colliculus.

So what mechanisms could be the basis for the time course and adjustment of inhibitory effects? Many studies have suggested that executive control over the perception, selection, and production systems is a central component of human cognition [35–41]. When the environment is ambiguous or presents competing demands, or the mapping of stimulus onto the response is complex or contrary to the habit – thereby making performance prone to errors – this executive control system is called into the action. Physiological evidence for a monitoring system in the medial frontal lobe has also been obtained. Event-related potential and neuroimaging studies have shown that activation in the medial frontal lobe, centered in the anterior cingulate cortex (ACC) is associated with registering the production of errors or conflicting processes, and need for the adjusted control of behavior [42–44]. Evidence consistent with this general hypothesis has been obtained in neurophysiological recordings from the supplementary eye field (SEF) and the ACC in monkeys performing the countermanding task [16, 45]. Our results let us speculate that the adjustments of performance due to modest stop probabilities occur through changes in the mechanisms that produce the response. To account for these results one might speculate that a selective mapping of inhibitory STOP networks within the fronto-medial cortex or the basal ganglia circuits may contribute to the encoding of the probability to inhibit an action.

5. Conclusion

The neural basis of the self-control of eye movements has been investigated with increasing precision in large part due to improved behavioral testing procedures and theoretical perspectives. Our study shows that signals to stop an impending saccade as well as those to facilitate it can be tuned according to the probability to execute or inhibit an action. We speculate that a selective inhibitory networks within the fronto-medial cortex or the basal ganglia circuits may contribute to the encoding of the probability to inhibit an action.

References

1. Logan G.D.: Spatial attention and the apprehension of spatial relations. Journal of Experimental Psychology: Human Perception and Performance 1994, 20, 1015–1036.
2. Armstrong I.T., Munoz D.P.: Inhibitory control of eye movements during oculomotor countermanding in adults with attention-deficit hyperactivity disorder. Experimental Brain Research 2003, 152, 444–452.
3. Asrress K.N., Carpenter R.H.: Saccadic countermanding: a comparison of central and peripheral stop signals. Vision Research 2001, 41, 2645–2651.

4. Cabel D.W., Armstrong I.T., Reingold E., Munoz D.P.: Control of saccade initiation in a countermanding task using visual and auditory stop signals. Experimental Brain Research 2000, 133, 431–441.
5. Curtis C.E., Cole M.W., Rao V.Y., D'Esposito M.: Canceling planned action: an FMRI study of countermanding saccades. Cerebral Cortex 2004, 15, 1281–1289.
6. Hanes D.P., Carpenter R.H.: Countermanding saccades in humans. Vision Research 1999, 39, 2777–2791.
7. Hanes D.P., Patterson W.F. 2nd, Schall J.D.: Role of frontal eye fields in countermanding saccades: visual, movement, and fixation activity. Journal of Neurophysiology 1998, 79, 817–834.
8. Hanes D.P., Schall J.D.: Countermanding saccades in macaque. Visual neuroscience 1995, 12, 817–834.
9. Kornylo K., Dill N., Saenz M., Krauzlis RJ.: Cancelling of pursuit and saccadic eye movements in humans and monkeys. Journal of Neurophysiology 2003, 89, 2984–2999.
10. Logan G.D., Irwin D.E.: Don't look! Don't touch! Inhibitory control of eye and hand movements. Psychonomic Bulletin and Review 2000, 7, 107–712.
11. Paré M., Hanes D.P.: Controlled movement processing: superior colliculus activity associated with countermanded saccades. The Journal of Neuroscience 2003, 23, 6480–6489.
12. Stuphorn V., Taylor T.L., Schall J.D.: Performance monitoring by the supplementary eye field. Nature 2000, 408, 857–860.
13. Logan G.D., Cowan W.B.: On the ability to inhibit thought and action: A theory of an act of control. Psychological Review 1984, 91, 295–327.
14. Pouget P., Stepniewska I., Crowder E.A., Leslie M.W., Emeric E.E., Nelson M.J., Schall J.D.: Visual and motor connectivity and the distribution of calcium-binding proteins in macaque frontal eye field: implications for saccade target selection. Frontiers in Neuroanatomy 2009, 3, 2.
15. Condy C., Wattiez N., Rivaud-Péchoux S., Gaymard B.: Ketamine-induced distractibility: An oculomotor study in monkeys. Biological Psychiatry 2005, 57, 366–372.
16. Ito S., Stuphorn V., Brown J.W., Schall J.D.: Performance monitoring by the anterior cingulate cortex during saccade countermanding. Science 2003, 302, 120–122.
17. Logan G.D., Cowan W.B., Davis K.A.: On the ability to inhibit responses in simple and choice reaction time tasks: A model and a method. Journal of Experimental Psychology: Human Perception and Performance 1984, 10, 276–291.
18. Logan G.D., Zbrodoff N.J., Williamson J.: Strategies in the color-word Stroop task. Bulletin of the Psychonomic Society 1984, 22, 135–138.
19. Band G.P.H., van der Molen M.W. Logan G.D.: Horse-race model simulations studies of the stop signal procedure. Acta Psychologica 2003, 112, 105–142.
20. Boucher L., Palmeri T.J., Logan G.D., Schall J.D.: Inhibitory control in mind and brain: an interactive race model of countermanding saccades. Psychological Review 2007, 114, 376–397.
21. Lo C.C., Boucher L., Paré M., Schall J.D., Wang X.J.: Proactive inhibitory control and attractor dynamics in countermanding action: a spiking neural circuit model. The Journal of Neuroscience 2009, 29, 9059–9071.
22. Emeric E.E., Brown J.W., Boucher L., Carpenter R.H., Hanes D.P., Harris R., Logan G.D., Mashru R.N., Paré M., Pouget P., Stuphorn V., Taylor T.L. Schall J.D.: Influence of history on saccade countermanding performance in humans and macaque monkeys. Vision Research 2007, 47, 35–49.
23. Carpenter R.H.S.: Express saccades: is bimodality a result of the order of stimulus presentation? Vision Research 2001, 41, 1145–1151.
24. Dorris M.C., Taylor T.L., Klein R.M., Munoz D.P.: Influence of previous visual stimulus or saccade on saccadic reaction times in monkey. Journal of Neurophysiology 1999, 81, 2429–2436.
25. Jüttner M., Wolf W.: Occurrence of human express saccades depends on stimulus uncertainty and stimulus sequence. Experimental Brain Research 1992, 82, 678–681.
26. Paré M., Munoz D.P.: Saccadic reaction time in the monkey: advanced preparation of oculomotor programs is primarily responsible for express saccade occurrence. Journal of Neurophysiology 1996, 76, 3666–3681.

27. Nelson M.J., Boucher L., Logan G.D., Palmeri T.J., Schall J.D.: Nonindependent and nonstationary response times in stopping and stepping saccade tasks. Attention, Perception, & Psychophysics 2010, 72, 1913–1929.
28. Logan G.D.: Attention, automaticity, and the ability to stop a speeded choice response. In: Long J.; Baddeley A.D. /Eds/, Attention and Performance IX. Hillsdale, NJ: Erlbaum; 1981.
29. Logan G.D., Burkell J.: Dependence and independence in responding to double stimulation: A comparison of stop, change, and dual-task paradigms. Journal of Experimental Psychology: Human Perception and Performance 1986, 12, 549–563.
30. Ramautar J.R., Kok A., Ridderinkhof K.R.: Effects of stop-signal probability in the stop-signal paradigm: the N2/P3 complex further validated. Brain & Cognition 2004, 56, 234–252.
31. Bruce C.J., Goldberg M.E.: (1985). Primate frontal eye fields. I. Single neurons discharging before saccades. Journal of Neurophysiology 1985, 53, 603–635.
32. Schall J.D., Sato T.R., Thompson K.G., Vaughn A.A., Juan C.H.: Effects of search efficiency on surround suppression during visual selection in frontal eye field. Journal of Neurophysiology 2004, 91, 2765–2769.
33. Goffart L., Quinet J., Chavane F., Masson G.S.: Influence of background illumination on fixation and visually guided saccades in the rhesus monkey. Vision Research 2006, 46, 149–162.
34. Munoz D.P., Istvan P.J.: Lateral inhibitory interactions in the intermediate layers of the monkey superior colliculus. Journal of Neurophysiology 1998, 79, 1193–1209.
35. Logan G.D.: Executive control of thought and action. Acta Psychologica 1985, 60, 60, 193–210.
36. Norman D.A., Sallice T.: Attention to action: Willed and automatic control of behavior. In: Davidson, R.J. Schwartz G.E.; Shapiro /Eds/, Conscioudness and Self- Regulation (Vol. 4): Advances in Research and Theory. New York: Plenum Press 1986, 1–18.
37. Allport D.A., Styles E., Hsieh S.: Shifting intentional set: Exploring the dynamics of control of tasks. In: Umilta C., Moscovitch M. /Eds/, Attention and Performance XV: Conscious and Nonconscious Information Processing. Cambridge, MA: MIT Press 1994, 421– 452.
38. Baddeley A., Della Sala S.: (1996). Working memory and executive control. Philosophical Transactions of the Royal Society B: Biological Sciences 1996, 351, 1397–403, discussion 1403–1404.
39. Logan G.D., Gordon R.D.: Executive control of visual attention in dual-task situations. Psychological Review 2001, 108, 393–434.
40. Repovs G., Baddeley A.: The multi-component model of working memory: explorations in experimental cognitive psychology. Neuroscience 2006, 139, 5–21.
41. Pouget P., Pradat-Diehl P., Rivaud-Péchoux S., Wattiez N., Gaymard B.: An oculomotor and computational study of a patient with diagonistic dyspraxia. Cortex 2011, 47, 476–483.
42. Van Veen V., Carter C.S.: The timing of action-monitoring processes in the anterior cingulate cortex. Journal of Cognitive Neuroscience 2002, 14, 593–602.
43. Nieuwenhuis S., Broerse A., Nielen M.M., de Jong R.: A goal activation approach to the study of executive function: an application to antisaccade tasks. Brain and Cognition 2004, 56, 198–214.
44. Ridderinkhof K.R., van den Wildenberg W.P., Segalowitz S.J., Carter C.S.: Neurocognitive mechanisms of cognitive control: the role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. Brain and Cognition 2004, 26, 129–140.
45. Stuphorn V., Schall J.D.: Neuronal control and monitoring of initiation of movements. Muscle Nerve 2002, 26, 326–339.