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Motor and non-motor inhibition in the Go/NoGo task: An ERP and fMRI study

Janette L. Smith [a,b,*], Sharna Jamadar [a,c], Alexander L. Provost [a], Patricia T. Michie [a]

[a] School of Psychology, Centre for Brain and Mental Health Research, University of Newcastle, Australia
[b] National Drug and Alcohol Research Centre, University of New South Wales, Australia
[c] Olin Neuropsychiatry Research Center, Institute of Living, Hartford, CT, USA

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ABSTRACT

The contribution of movement-related activity to Go/NoGo ERP differences has been debated for 25 years. In this study, we examined ERP and fMRI measures of activity in twenty adults performing non-motor (count) and motor (right-handed button press) trials of the Go/NoGo task. Task performance was highly accurate and similar in the ERP and fMRI environments. No significant task-related effects were observed for the N2 component; however, we observed a substantial increase in positivity for Press NoGo compared to Count NoGo trials. The fMRI results also revealed significant deactivations for Press NoGo relative to Count NoGo trials in several left-lateralised motor-related areas, including the inferior frontal gyrus, precentral gyrus and supplementary motor area. Together, the results indicate that the P3 NoGo > Go effect in motor tasks is caused not by movement-related negativity on Go trials but by inhibition-related positivity on NoGo trials, and that this is associated with deactivation of motor areas involved in the Go response.

1. Introduction

Response inhibition, or the cancellation of a planned response (Nigg, 2000) can be studied in the Go/NoGo task, in which participants must press a button to one type of stimulus (Go), and withhold that response to stimuli of another type (NoGo). Because inhibition is a covert process that, when successful, produces little or no overt behaviour for measurement, cognitive neuroscience techniques such as event-related potentials (ERPs) and functional magnetic resonance imaging (fMRI) are increasingly being used to gain understanding of these hidden processes.

Previous fMRI analyses of the Go/NoGo task and the related stop-signal task (in which participants perform a two-choice response task and a stop-signal is presented after the Go stimulus on a small proportion of trials, requiring participants to stop their ongoing response) have shown that successful inhibition trials are associated with increased activation (a positive blood oxygenation level-dependent (BOLD) signal) in a predominantly right hemispheric network, including the right dorsolateral and ventrolateral prefrontal cortices, precentral gyrus and pre-supplementary motor area (pre-SMA), anterior cingulate cortex, bilateral superior and inferior parietal lobules, precuneus, insula, and putamen (Aron and Poldrack, 2006; Braver et al., 2001; Durston et al., 2002; Fassbender et al., 2006; Garavan et al., 2006, 2003, 2002; Goghari and MacDonald, 2009; Horn et al., 2003; Hughes et al., 2012; Kaufman et al., 2003; Kelly et al., 2004; Konishi et al., 1998; Kühn and Brass, 2009; Menon et al., 2001; Mostofsky et al., 2003; Rubia et al., 2001, 2003; Simmonds et al., 2008; Tamm et al., 2004; Vallesi et al., 2009; Vink et al., 2005; Wager et al., 2005; Watanabe et al., 2002). The inferior frontal gyrus (IFG), particularly on the right, is robustly activated when inhibition is required (Aron et al., 2003, 2004; Garavan et al., 2002; Ridderinkhof et al., 2004; Rubia et al., 2003). Furthermore, inhibitory deficits have been observed in patients with lesions specific to that area (Aron et al., 2003; Swick et al., 2008). Chikazoe et al. (2007) have also shown that this activation is not dependent on the motor effector, with similar areas being activated in a saccade/anti-saccade task. Lastly, several studies have shown changes in stop-signal task performance with brain stimulation methods: Jacobson et al. (2011) have shown reductions in stop-signal reaction time (SSRT) with excitation of the right IFG using transcranial direct current stimulation, while Chambers et al. (2006) have shown increases in SSRT and reductions in the probability of inhibition after deactivation of the right IFG using repetitive transcranial magnetic stimulation (rTMS). In both cases, the alterations to performance were not generalised to responses to Go stimuli, nor were they apparent when other areas of the brain were stimulated. Thus, the right IFG has been pinpointed as a possible seat of inhibition processes by some authors.

However, more recent work has demonstrated that the IFG is not activated specifically by cues to response inhibition, but rather, by cues to update the plan of behaviour. For example, Hampshire et al. (2010) have shown that the IFG is activated not only when participants are presented with a stop-signal, but also when they are instructed to count the signals, or activate an unplanned response. Similarly, Sharp et al. (2010) demonstrated that IFG was activated to both stop-signals and ignore/continue-signals. Verbruggen et al. (2010) have also shown that

* Corresponding author at: National Drug and Alcohol Research Centre, University of New South Wales, Building R3, Randwick Campus, 22-32 King Street, Randwick, NSW 2031, Australia. Tel.: +61 2 9385 0274; fax: +61 2 9385 0222.
E-mail address: janette.smith@unsw.edu.au (J.L. Smith).

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rTMS disruption of rIFG produces not only longer stop-signal reaction times, but also increased reaction time to signals requiring a dual response. Those authors have suggested that IFG activation reflects attentional capture by task-relevant cues (Hampshire et al., 2010), or by unexpected events (Sharp et al., 2010), or the updating of action plans (Verbruggen et al., 2010). In contrast, Sharp et al. (2010) suggest the seat of inhibition may be the pre-SMA.

Analyses of electrical activity to Go and NoGo stimuli show robust differences in both the N2 and P3 components of the ERP. The N2 is frontocentrally maximal and is increased on NoGo compared to Go trials (e.g., Bokura et al., 2001; Jodo and Kayama, 1992; Kok, 1986), leading early researchers to interpret the N2 as a marker of inhibition (e.g., Kok, 1986). However, the N2 is larger for failed than for successful inhibition trials in the stop-signal task (e.g., Dimoska et al., 2006; Kok et al., 2004), and is larger to Go trials when these are rare or otherwise unexpected (Donkers and Van Boxtel, 2004; Randall and Smith, 2011; Smith et al., 2010). Such evidence cannot be explained by an inhibitory interpretation of N2. In contrast, the conflict theory states that conflict (and an increased N2) will arise whenever multiple, mutually incompatible representations are activated (Botvinick et al., 2001). Under this hypothesis, N2 is increased to NoGo trials not because they require inhibition per se, but because they require a different response to what is expected (by virtue of, for example, stimulus probability or task instructions). This interpretation has gained ground in recent years to become the dominant theory of N2 in most paradigms requiring behavioural control (Folstein and Van Petten, 2008), and the parallels with recent views on rIFG function (Hampshire et al., 2010; Sharp et al., 2010; Verbruggen et al., 2010) are clear.

In contrast, the frontocentral P3 component is becoming more accepted as a measure of the inhibitory process. The NoGo P3 is larger in participants who respond faster to Go stimuli (as fast responses are harder to inhibit; Smith et al., 2006), and is increased when a Go stimulus has been cued (Bruin et al., 2001; Randall and Smith, 2011; Smith et al., 2007) or is otherwise expected (Smith et al., 2010). Furthermore, in the stop-signal task, P3 is robustly increased for successful compared to failed inhibitions (Dimoska and Johnstone, 2008; Dimoska et al., 2006; Kok et al., 2004; Lansbergen et al., 2007; Ramautar et al., 2004, 2006). Thus, the frontocentral increase in P3 on trials requiring inhibition is hypothesised to reflect the inhibitory process. In Kok et al.'s study, it was suggested that the onset of the successful > failed effect may reflect the timing of the inhibitory process.

One issue that remains unresolved in the literature is whether the N2 and P3 effects (that is, the difference between Go and NoGo trials) represent cognitive or motor-related aspects of inhibition. Some authors, reviewed below, have argued that the P3 effect in particular is confounded by negative-going movement-related potentials on Go trials, which are absent for NoGo trials. The use of variations on the Go/NoGo task, including counting versions, has shed some light on this. A number of papers have used traditional tasks with equal or greater probability of Go compared to NoGo trials, ensuring that inhibition is difficult. Pfefferbaum et al. (1985) showed that the N2 and P3 NoGo effects were apparent for both Count and Press versions of a Go/NoGo task, although the N2 effect was larger in the Press task. van't Ent and Apkarian (1999) demonstrated that frontocentral P3 amplitude was enhanced for NoGo compared to Go trials, for both button press and saccade versions of the task, which did not differ, demonstrating that the P3 NoGo effect is not specific to finger movements. Bruin and Wijers (2002) varied Go/NoGo probability between conditions (25%, 50%, 75% Go trials), and reported similar N2 effects in Count and Press tasks, although these reduced with increasing stimulus probability. However, although the NoGo P3 was shifted anteriorly in the Count task, similar to the Press task, it was not larger in amplitude than the Go P3 at any site. Wang et al. (2002) reported N2 to be of equal amplitude for Press NoGo and Count NoGo, both larger than their respective Go trials, but did not report P3 amplitude. Nakata et al. (2004) reported an early frontal negativity to be larger for NoGo than for Go trials in both Count and Press versions; however, P3 was larger for NoGo than for Go only for the Press task. Lastly, Burle et al. (2004) used a task requiring an actual button press, or the imagination of a button press, or their inhibition on 25% of trials. N2 to NoGo targets showed similar amplitude for Press and Imagine. P3 was smaller in the Imagine than in the Press condition, but displayed an increase in positivity (relative to Go targets) for both conditions. Thus, the N2 effect can be elicited in Count versions of the task, and usually with similar amplitude to Press versions. Mixed results have been reported for the Count NoGo P3 effect: sometimes the effect is significant in both Count and Press versions, sometimes the effect is not significant for the Count task, and sometimes a more anterior topography on NoGo trials is displayed, but without being larger than Go trials.

A substantial body of papers has also examined response mode effects in oddball tasks, with target stimuli requiring a Count or Press response on 15–20% of trials, and no response on the remainder. Polich (1987) reported larger P3 to Count than to Press targets, while Barrett et al. (1987) replicated this result, with increased amplitudes particularly at Cz and C3, contralateral to the right-hand response required in the Press condition. Hatta et al. (1997) report a parietal maximum for targets in the motor task, and a centroparietal maximum in the Count task, while for non-targets, Press and Count tasks show similar P3. Starr et al. (1997) also reported no difference in the P3 to non-targets for Press and Count trials. Thus, in studies using oddball tasks, ERPs to non-targets generally do not differ, while targets generally show reduced P3 positivity for Press compared to Count, particularly at frontal and central sites, possibly due to negative movement-related potential overlap.

In a well-considered study, Salisbury et al. (2001) asked participants to complete three tasks in a counterbalanced order: in one block, participants pressed a button each time a tone was presented (Respond-all task); in a second block, they silently counted the same tones presented infrequently (15%) among other standard frequent tones (85%, Count task); in a third block, they made button press responses to the rare targets (Press task). ERPs in the Respond-all task were considered a good model of movement-related activity, in the absence of a P3 component since no discrimination/decision was required. Individual trials from the Respond-all task were matched for RT with trials from the Press task, and movement-related activity was subtracted from the Press ERPs. Raw (uncorrected) and Corrected P3 from the Press task was then compared with P3 from the Count task. Relative to the Count P3, the Uncorrected Press P3 was reduced in amplitude, particularly at the midline, showed a left < right effect frontocentrally, and showed a more parietal distribution, compared to a centroparietal maximum for the Count P3. After subtraction of movement-related potentials, the Corrected Press P3 showed increased amplitude, particularly in frontocentral regions, and removed the hemispheric asymmetry effect, to be more similar to the Count P3. The authors argued that P3 amplitude is reduced when a button press is required in oddball tasks; by extension, the P3 NoGo effect may be produced not by inhibitory potentials on NoGo trials, but rather by movement-related negativity on Go (Press) trials.

In a later study, Salisbury et al. (2004) compared ERPs to rare targets in three conditions: in one task, participants counted rare targets (15%) among frequent (85%) standards (silent count task). In a second task, participants pressed a button to rare targets (Go task), and in the third task, participants pressed a button to frequent standards, withholding that press to rare targets (NoGo task). At frontocentral sites, Go P3 was decreased relative to the NoGo P3, which was almost identical in amplitude and topography to the Count P3, despite no inhibition being required to Count stimuli. Therefore, they argued that the P3 difference typically observed for Go and NoGo trials was due to movement-related negativity on Go trials, rather than to inhibition-related positivity on NoGo trials. However, the experiment did not include a condition examining the inhibition of a count response.

Previous work from the current authors (Smith et al., 2008) included such a condition, and presented frequent Go (60%), rare Go