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CNV resolution does not cause NoGo anteriorisation of the P3: A failure to replicate Simson et al.

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ABSTRACT

For 35 years, some researchers have argued that CNV resolution may affect or even produce the increased P3 for NoGo compared to Go trials, and thus that no 'inhibitory' NoGo P3 exists. This is based on the work of Simson et al. (1977b), the scalp topography of potentials in auditory and visual Go/NoGo tasks. *Electroencephalography and Clinical Neurophysiology*, 43, 864–875, which compared Go and NoGo topography after CNV was subtracted from NoGo trials only. Specifically, the NoGo P3 topography showed the distinctive frontocentral maximum, which is often linked to motor inhibition, when referenced to a pre-target baseline. This NoGo topography changed to a more parietal maximum, similar to that on Go trials, when referenced to a pre-cue baseline. Many researchers have cited this study, while failing to use the delayed response design on which Simson et al. based their argument. We attempted to replicate Simson et al.'s experiment with delayed responses and also with immediate responses, as are more often used. As expected, the amplitudes of CNV and P3 to both Go and NoGo trials were increased when immediate compared to delayed responses were required, but we failed to replicate the topographic shift of NoGo P3 with different baselines for both delayed and immediate responses. That is, subtraction of the CNV from NoGo P3 did not change the distinctive frontocentral topography of this component. The results suggest that CNV may affect the amplitude and measurement of the NoGo P3, but that NoGo P3 anteriorisation is not caused by CNV resolution.

1. Introduction

Inhibitory processes can be examined in the Go/NoGo task, in which subjects must respond, usually with a button press, to one stimulus type (Go, usually at least 50% of trials, to ensure that the Go response is prepotent), but withhold that response to stimuli of another type (NoGo, usually 50% or less trials, to ensure that inhibition is rare and therefore more difficult). Event-related potentials (ERPs) show robust differences to these two stimulus types: on NoGo compared to Go trials, both N2 and P3 amplitudes are larger, and the P3 shows a more anterior topography (e.g., Bekker et al., 2004; Bruin and Wijers, 2002; Kok, 1986; Lavric et al., 2004; Nieuwenhuis et al., 2003; Smith et al., 2006). The NoGo N2 is thought to be generated in the anterior cingulate cortex (e.g., Bekker et al., 2005) and to represent conflict between the competing Go and NoGo responses (e.g., Nieuwenhuis et al., 2003), while the NoGo P3 is thought to represent the action of the inhibitory process itself, and is associated with deactivation of motor areas used to make the Go response (Smith et al., 2013).

The use of a cue to signal the identity and/or timing of an upcoming Go/NoGo stimulus allows simultaneous examination of preparatory processes, and increases the inhibitory requirements of the task, since a highly prepared response is harder to inhibit. The N2 and P3 effects are observed whether or not a cued task is used (e.g., uncued: Mathalon et al., 2003; Nieuwenhuis et al., 2003; Pfefferbaum and Ford, 1988; cued: Jodo and Kayama, 1992; Nativ et al., 1992; Roberts et al., 1994). Thus, the cue and the accompanying response preparation are not essential to understanding the N2 and P3 in the Go/NoGo task, at least where the cue is a predictor of target timing only, and not of target identity. However, in other inhibitory tasks with a cue-target trial structure, the processing of cue identity is integral to the interpretation of results in that task. For example, in the continuous performance task, as used by Roberts et al. (1994), subjects are required to respond to the target letter (X) only if it is preceded by a particular cue letter (A). The sequence A–X is considered a response preparation–execution (Go) sequence, while the sequence A -not-X is considered a response preparation -inhibition (NoGo) sequence. Thus, the letter A triggers preparatory processes and is important in successfully completing the task. In the Posner task (Posner et al., 1978), a cue stimulus informs the participant validly or invalidly of the location of the upcoming target. Thus the cue triggers a shift of spatial attention, which results in a reaction time (RT) benefit to validly cued targets, and a cost to invalidly cued targets, relative to when no cue or a neutral cue is presented. Some researchers have argued that response preparation

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on the cued side must be inhibited in the case of invalid cues, in order to give the correct response, thus, it is considered by some to be an inhibitory task (e.g., Gehring et al., 1992). The contingent negative variation (CNV; Walter et al., 1964) can be observed reliably in almost any task with a fixed cue–target interval. It is a slow negative shift arising between the cue and the target, peaking at or just before target onset, and usually frontocentrally maximal.[1] Thus, the CNV is considered to be the electrophysiological marker of stimulus anticipation and response preparation processes.

However, the cued paradigm causes problems with interpretation of post-target potentials, because the decay and termination of the negative shift of the CNV ("CNV resolution") are not necessarily instantaneous with the presentation of the target. Rather, CNV resolution may overlap with post-target potentials, thus possibly changing the observed amplitudes and topographies of ERP components occurring in that time range. Historical views of this problem have concentrated on the overlap of CNV with the P3 peak. It has been argued previously that the P3 was nothing more than the positive-going resolution of the CNV, not a component in itself (e.g., Donchin and Smith, 1970; Wilkinson and Ashby, 1974; Wilkinson and Spence, 1973). This may be due partly to the fact that the P3 and CNV were first described at around the same time (Sutton et al., 1965; Walter et al., 1964) in similar tasks (i.e., cued designs with some degree of target stimulus uncertainty and relevance to the subjects' task). Indeed, the original paper by Walter et al. (1964) shows a P3-like overshoot of baseline after the target stimulus. The similarity in tasks eliciting CNV and P3 led other researchers to suggest that CNV and P3 were separable waves, but activated by a common mechanism. That is, the mechanism that increases CNV before task-relevant, compared to irrelevant stimuli, is the same mechanism that increases post-target potentials to task-relevant stimuli (Näätänen, 1970). A flurry of work in the 1970s led to acceptance of the proposition that CNV and P3 are separate components, but that the positivity associated with CNV resolution may be added to the P3 positivity and hence influence the measurement of that component (see Donald and Goff, 1971, 1973; Hillyard et al., 1976; Karlin, 1970; Kok, 1988; Smith et al., 2007; Tueting and Sutton, 1973; but especially Donchin et al., 1975; Verleger et al., 2013). It should be noted here, as Oddy et al. (2005) have pointed out, that work has focused on the P3 despite the possibility that all post-target potentials are affected; for example, a possible relationship between the CNV and the N1 has been discussed by Jarvilehto and Fruhstorfer (1970), and Näätänen (1970) has argued that P2 amplitude is related to arousal in the prestimulus period, rather than any poststimulus processing.

After it was accepted that the CNV and P3 were separate processes, researchers often used a pre-cue baseline to study CNV and other post-cue processes, and a pre-target baseline to study post-target potentials, as if the electrical potentials occurring after the target were completely independent of those occurring before the target. This approach allows comparison with non-cued versions of tasks, which routinely have a pre-target baseline period, but ignores the contribution of CNV resolution, partly due to the lack of a method to remove it.

In the Go/NoGo task in particular, it has been an important problem, and Simson et al. (1977b) were the first to attempt a solution. Participants performed a cued task with equiprobable Go/NoGo stimuli, and were instructed to delay their response to the Go stimuli by about 1 s. The CNV developed over the cue–target interval as participants prepared a response, and showed the typical central maximum. Simson et al. argued that, on NoGo trials, CNV resolved almost immediately, since the subjects were required to do nothing further for the trial, but on Go trials, subjects maintained their state of readiness until it was appropriate to execute the response, thus delaying CNV resolution. After the target, the usual frontocentral increase in P3 for NoGo stimuli was observed (with a pre-target baseline; compare solid red (NoGo) and green (Go) lines in the left half of Fig. 1). That is, a parietocentral maximum was observed for Go P3, while a central maximum was observed for NoGo P3. Simson et al. noted the similarity in central topographies for the CNV (blue lines in Fig. 1) and NoGo P3, and argued that the immediate resolution of CNV on NoGo trials was the cause of the observed NoGo P3 central topography. When the NoGo P3 was referred to a pre-cue baseline (so as to minimise the influence of CNV development and resolution; dashed red lines in Fig. 1), the Go/NoGo differences were no longer apparent. That is, when the CNV topography was removed in this fashion, the NoGo P3 amplitude was reduced and its topography was changed to a parietal maximum, similar to the Go P3 (see Fig. 1). Therefore, Simson et al. suggested that the NoGo P3 effect was solely due to CNV resolution differences, not to any inhibitory process occurring on NoGo trials.

The method of using different baselines has been popular. Indeed, before Simson et al. (1977b) published their results, Donchin et al. (1975) measured both N1 and P3 relative to a pre-cue and a pre-target baseline. Other researchers continue to refer to Simson et al.'s method of using different baselines for Go and NoGo P3 (e.g., Roberts et al., 1994; Verleger et al., 2006), with results both similar to, and different from, those of Simson et al. (see Fig. 1). For example, Verleger et al. reported effects similar to Simson et al., yet Roberts et al. reported that, after subtraction of the CNV from NoGo P3 amplitude, the topography was not similar to the centroparietally maximal Go P3, instead displaying a frontal maximum and retaining the frontal NoGo > Go effect. Still other studies do not perform the subtraction method, yet refer to differential CNV resolution as a possible explanation of their results, in contrast to an inhibitory explanation (e.g., Eimer, 1993; Jodo and Inoue, 1990; Kok, 1986, 1988). However, none of those studies used a delayed response paradigm, which was the reason for the Go/NoGo differences in CNV resolution in Simson et al., and thus the crux of the argument for the use of differing baselines. Hence, due to the importance attributed to Simson et al.'s results despite no attempts to replicate them, in the current study we seek to discover whether we can reproduce the results of Simson et al. using delayed responses, and whether these effects are altered when an immediate response is used, as in more recent research. We compare our results to previously published work from Simson et al., Roberts et al., and Verleger et al., and report some notable similarities and differences between studies.

2. Methods

2.1. Participants

Participants were twenty adults (1 male, 1 left-handed) with a mean age of 22.9 (SD = 8.4) years who participated in the study for

[1] At least three separate waves contribute to the CNV: an early orienting response to the cue stimulus, often frontally negative and parietally positive (e.g., Connor and Lang, 1969; Weerts and Lang, 1973); the stimulus preceding negativity (SPN), a negative-going parietally maximal wave occurring when the participant expects a salient stimulus and most purely observed in the absence of a motor response to the stimulus (e.g., Damen and Brunia, 1985; Van Boxtel and Böcker, 2004); and the readiness potential (RP), a negative-going centrally and contralaterally maximal wave time-locked to the subject's response (Kornhuber and Deecke, 1964, 1965). The early and late waves can be separated by the use of a long cue–target interval or by the use of principal components analysis (PCA). However, reliable separation and/or pure observation of the SPN and RP involves the use of vastly different experimental paradigms. In cue–target–response paradigms, stimulus anticipation and response preparation processes can never be observed alone, since the notion of preparing a response to some stimulus necessarily entails some expectancy for the stimulus. Similarly, in studies of the SPN, the expected stimulus often provides feedback about prior task performance and/or instructions about future tasks, and therefore it can be argued that SPN does have some aspect of preparation for future responses. Thus, the processes are not easily separable, and rather should be considered as "extremes on a perception–action continuum" (Van Boxtel and Böcker, 2004, p. 61). The relative contribution of the SPN and RP to the 'late CNV' depends on the task requirements: in tasks which are primarily motoric, it is argued that the late CNV consists principally of response preparation processes, while in sensory tasks or in those which use meaningful or interesting stimuli requiring no response, the late CNV consists mostly of stimulus anticipation processes. We interpret the late CNV in this study as principally motoric; however, we do not believe that this interpretation, or the possibility of separating SPN and RP, is important for the research question in this study.