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RESEARCH REPORT

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A possibility of error-related processing contamination in the No-go N2: The effect of partial-error trials on response inhibition processing

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Abstract

This study investigated whether error-related negativity (ERN) elicited by partial errors and No-go N2 represent distinct or similar components. We also investigated whether the error positivity (Pe) and No-go P3 represent distinct or similar components. Partial-error trials are behaviourally classified as correct trials but preceded by covert muscular activities. Recent studies have reported that analysing partial-error trials is useful for investigating the functional roles of ERN and No-go N2. In this study, 23 participants performed a Go/No-go flanker task. They performed nine blocks of 60 trials each. Stimulus-locked event-related potentials (ERPs) were averaged separately for Go-congruent pure-correct trials, Go-incongruent pure-correct trials and No-go pure-correct trials. In addition, we compared the stimulus-locked ERPs among No-go pure-correct trials, No-go partial-error trials, Go-incongruent pure-correct trials and Go-incongruent partial-error trials. Electromyogram (EMG)-locked ERPs were averaged separately for correct trials, overt errors in No-go trials, partial errors in No-go trials, overt errors in incongruent trials and partial errors in incongruent trials. N2 was remarkably larger in No-go partial-error trials than in No-go pure-correct trials. Consistent with previous findings, the No-go partial-error N2 might reflect error-related processing. P3 amplitudes were larger in the No-go trials than in both the Go-congruent and Go-incongruent trials. These results suggest that the No-go P3, but not the No-go N2, might reflect inhibition of overt movement. The present findings provide further evidence that the previously reported increase in No-go N2 may be due to an overlap of the ERN elicited by partial errors.

KEYWORDS
error-related negativity, No-go N2, No-go P3, partial errors

Abbreviations: ANOVA, analysis of variance; Cz, midline central; DC, direct current; EEG, electroencephalogram; EMG, electromyogram; EOG, electrooculogram; ERN, error-related negativity; ERP, event-related potential; FCz, midline frontocentral; Pe, error positivity; Pz, midline parietal; RT, reaction time.

1 | INTRODUCTION

Inhibitory control is a critical executive function (Miyake et al., 2000). Consequently, numerous studies have determined the neural correlates of response inhibition. In the Go/No-go paradigm, where frequent Go stimuli signalling responses and rare No-go stimuli inhibiting responses are randomly presented, an enhanced N2 is observed approximately 200 ms after the No-go stimulus onset (e.g., Fallgatter & Strik, 1999; Iannaccone et al., 2015; Jodo & Kayama, 1992; Pfefferbaum et al., 1985). The enhanced N2 for the No-go stimulus is referred to as No-go N2 (Kok, 1986), which has been believed to reflect response inhibition (Abdul Rahman et al., 2017; Falkenstein et al., 1999; Fallgatter & Strik, 1999; Jodo & Kayama, 1992; Kok, 1986).

However, some studies have proposed a different perspective: The functional significance of N2 components, including both Go N2 and No-go N2, is a response conflict (Donkers & van Boxtel, 2004; Iannaccone et al., 2015; Nieuwenhuis et al., 2003; Randall & Smith, 2011; Smith et al., 2007; Smith et al., 2013; Yeung et al., 2004). This is because the No-go N2 amplitude was larger in the high-conflict trials than in the low-conflict trials (Iannaccone et al., 2015; Randall & Smith, 2011), larger for the unexpected No-go stimulus than for the expected No-go stimulus (Randall & Smith, 2011) and larger for the low-frequency Go stimulus than for the high-frequency No-go stimulus (Nieuwenhuis et al., 2003).

Furthermore, from the perspective of error processing, previous studies have confirmed that covert erroneous muscular activities, referred to as *partial errors*, can be observed from the withhold hand for the No-go stimulus (Maruo et al., 2017; Masaki et al., 2012; Masaki & Segalowitz, 2004; Nguyen et al., 2020; Vidal et al., 2000). This indicates the existence of covert responses on successfully withholding trials, raising questions about the definition of 'success' in response inhibition. As described below, it is pivotal to consider the functional significance of No-go N2 because covert muscular activities elicited by the No-go stimulus are unsuccessful response inhibitions at the peripheral level.

Although error-related negativity (ERN, Falkenstein et al., 1990; Gehring et al., 1990) is elicited by overt errors in conflict tasks, it can also be elicited by erroneous muscular activities on the wrong response limb preceding corrective response (i.e., partial errors) in correct trials. This is referred to as partial-error ERN, which shows a scalp distribution similar to that of No-go N2 over the frontocentral regions (Burle et al., 2008; Masaki et al., 2012; Masaki & Segalowitz, 2004). Given that ERN is elicited by partial errors in No-go trials, the partial-error ERN likely overlaps with No-go N2 in time, thereby enhancing negativity.

Nguyen et al. (2016) found a larger No-go N2 in partially inhibited trials (i.e., partial-error trials) than in the inhibition-success trials, suggesting that the enhanced No-go N2 might reflect error processing. However, Nguyen et al. (2016) did not compare stimulus-locked No-go N2s with response-locked ERNs, considering partially inhibited trials for the No-go stimulus. These findings raise the question that the widely accepted No-go N2 might reflect a mixture of error detection and response inhibition processes.

Therefore, it is reasonable to presume that No-go N2s were contaminated by partial error processing in previous studies that did not record muscular activities from response hands. It is noteworthy that the functional significance of No-go N2 cannot be determined unless partial errors are eliminated. In this study, we investigated the involvement of partial errors in No-go N2 and whether the same findings for No-go N2s are obtained for trials free of partial errors, using a suitable task to robustly observe the No-go N2. We used a Go/No-go flanker task (Iannaccone et al., 2015) where Go-congruent and Go-incongruent stimuli were randomly presented, and some of them were No-go stimuli (20%). Iannaccone et al. (2015) found that N2 was larger in the high-conflict trials (Go-incongruent) than in the no-conflict trials (Go-congruent). However, they did not compare the high-conflict trials (Go-incongruent) and no-conflict trials (Go-congruent) with No-go trials. If the response conflict influences the N2 amplitudes, N2 should be larger in No-go pure-correct trials (i.e., partial-error free trials) than in the Go-congruent trials.

We recorded electromyograms (EMGs) from both forearms to identify partial errors and rigorously analysed both Go N2 and No-go N2, classifying trials according to the presence or absence of erroneous EMG activities. The steps for analysing event-related potentials (ERPs) were as follows. First, we compared the stimulus-locked ERPs among three pure-correct trials (i.e., excluding partial errors): (1) Go-congruent pure-correct trials, (2) Go-incongruent pure-correct trials and (3) No-go pure-correct trials. If the previously reported No-go N2s included partial-error ERNs, the stimulus-locked N2s on pure-correct trials in our study should not differ between Go-congruent pure-correct trials and No-go pure-correct trials. In addition, if N2 reflects response conflict (Nieuwenhuis et al., 2003), the stimulus-locked N2 should be larger in Go-incongruent pure-correct trials than in Go-congruent pure-correct trials. Second, we compared the stimulus-locked ERPs among four different trials: (1) No-go pure-correct trials, (2) No-go partial-error trials, (3) Go-incongruent pure-correct trials and

(4) Go-incongruent partial-error trials. Given that the N2 in partial-error trials reflects error detection (Masaki et al., 2012; Nguyen et al., 2016), the No-go N2 should be larger in partial-error trials than in pure-correct trials. For the same reason, we also expected that the N2 elicited by incongruent stimuli would be larger in partial-error trials than in pure-correct trials.

No-go N2 is followed by No-go P3, which is distributed over the frontocentral regions in No-go trials (De Jong et al., 1990; Dimoska et al., 2006;Gajewski & Falkenstein, 2013; Groom & Cragg, 2015; Nguyen et al., 2020, 2016; Smith et al., 2006, 2008). Compared with No-go N2, No-go P3 is, seemingly, more involved in response inhibition (Donkers & van Boxtel, 2004; Nguyen et al., 2020; Randall & Smith, 2011; Smith et al., 2013). Previous studies found larger P3s for No-go stimuli than for Go stimuli (Gajewski & Falkenstein, 2013; Groom & Cragg, 2015; Nguyen et al., 2020, 2016; Smith et al., 2006, 2008). In addition, Nguyen et al. (2016) found larger No-go P3s in successfully inhibited trials than in partially inhibited trials. Smith et al. (2013) found that No-go P3 was larger in a button press condition where a motor response was inhibited for the No-go stimulus than in a count condition where a cognitive response was inhibited, whereas the No-go N2 did not differ between the two conditions. These findings suggest that No-go P3 may reflect inhibition control of an overt movement (Nguyen et al., 2020; Smith et al., 2013) and completion of adequate inhibition of the planned response (Nguyen et al., 2016).

Based on previous findings, we presumed that No-go P3, unlike No-go N2, represents response inhibition. In this study, we were also motivated to examine the functional significance of the No-go P3. If No-go P3 exclusively reflects response inhibition, No-go P3 should be larger in No-go trials than in Go trials, independent of stimulus congruency and the occurrence of partial errors. Furthermore, we examined if the error positivity (Pe; Falkenstein et al., 1991) is superimposed on the No-go P3 in our Go/No-go task. Few studies have reported the involvement of partial errors in the Pe. Previous studies have identified early and late Pe in overt-error trials (Endrass et al., 2007; Maruo et al., 2017; O'Connell et al., 2007; Thurm et al., 2020). These studies reported that the early Pe distribution over the frontocentral regions might reflect error detection (Endrass et al., 2007; O'Connell et al., 2007; Thurm et al., 2020; Wang et al., 2020), and late Pe, which is distributed over the centroparietal regions, might reflect error awareness (Endrass et al., 2012, 2007; O'Connell et al., 2007; Thurm et al., 2020). However, the roles of early Pe and late Pe remain unclear. If the error positivity is superimposed on the No-go P3, the early positivity might reflect response inhibition rather than error detection.

In terms of response conflict, this study tests the assumption that the ERN elicited by overt errors should be larger in incongruent trials than in No-go error trials because response conflict is stronger in incongruent trials than in No-go trials (Braver et al., 2001). However, the ERN may not differ between these two trials, supporting the assertion that the ERN reflects the online monitoring of responses (Carbonnell & Falkenstein, 2006), not the response conflict (Yeung et al., 2004).

2 | METHOD

2.1 | Participants

Twenty-three participants (17 men, mean age ± SEM = 22.1 ± .6 years) were recruited from Waseda University's Faculty of Sports Sciences. Participants had normal or corrected-to-normal vision and were paid 2400 yen (about 28 US dollars) for their participation. Written informed consent was obtained from all participants. This study was approved by the Ethics Committee of Waseda University.

2.2 | Procedure

Participants performed a Go/No-go flanker task (Figure 1) (Iannaccone et al., 2015). In each trial, two white-coloured arrowheads (i.e., either ▲ or ▼, 1.1° × 1.1° each) were presented vertically 100 ms preceding the target stimulus' onset on a computer monitor placed in front of the participant (1-m distance). Subsequently, the target stimulus (i.e., either ▲, ▼ or ◆) appeared between the flanker stimuli for 200 ms, functioning as the imperative stimulus. Trials in which the target triangle was identical to the flanked triangles were defined as Go-congruent trials, and those in which the target triangle was flanked by upturned triangles were defined as Go-incongruent trials. The rhombus stimulus indicated No-go trials. The Go/No-go flanker task consisted of 40% Go-congruent trials, 40% Go-incongruent trials and 20% No-go trials. These stimuli were followed by a blank screen lasting either 750, 850 or 950 ms. The stimuli were presented with a tachistoscope system (Iwatsu Isel, IS-702). Participants were instructed to respond to the direction of the triangle quickly and accurately with a brisk finger extension. They were also instructed not to respond to the rhombus (No-go stimulus) and ignore the flanking triangles.

Participants rested both forearms and palms comfortably on a table to minimize any movements unrelated to their responses. Responses were recorded using two

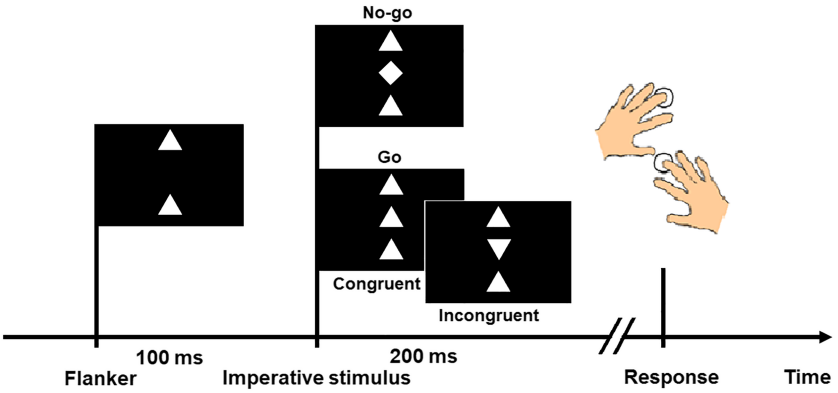

FIGURE 1 Procedure of a Go/No-go flanker task used in the present study. Participants were asked to respond to the pointing direction of the white triangle stimulus (i.e., up or down), but not to respond to the rhombus

microswitches mounted 150 mm apart in the midsagittal line. The microswitches included a small cantilever that required a small upward displacement for switch closure. A plastic plate (30 × 20 × 1 mm) was attached to the end of the cantilever key to provide leverage. Participants placed their middle fingers at the end of the plastic plate. The weight of the finger at rest was sufficient to depress the key. Half of the participants were asked to briskly lift the right middle finger in response to ▲ and the left middle finger in response to ▼, and the other half, vice versa. If participants did not respond within 450 ms in either the Go-congruent or Go-incongruent trials, a feedback letter appeared on the screen ('Too Late!') for 500 ms, indicating no response in that trial (time-out trial). Before the experiment, participants performed 30 practice trials and subsequently performed nine consecutive blocks (60 trials per block). After every three blocks, if the participants' error rate was less than 10%, they were instructed that the time interval allowed for a response in both Go-congruent and Go-incongruent trials would be shortened by 50 ms. Reaction time (RT) was measured as the interval between the imperative stimulus onset and the response onset.

2.3 | EEG recording

The EEGs from 128 scalp sites and both horizontal and vertical electrooculograms (EOGs) from the left and right outer canthi and from above and below the left eye, respectively, were recorded using direct current (DC) and a 100-Hz low-pass filter using the Biosemi Active-Two system (Biosemi Inc.) that adopted Ag/AgCl active electrodes. We also recorded bipolarly EMGs from the extensor digitorum muscles in the response forearms with Ag/AgCl active electrodes using the Biosemi Active-Two system. EMGs were high-pass filtered at 5.31 Hz and full-wave rectified off-line with the Vision Analyser (Brain Products) software. All physiological signals were recorded at a sampling rate of 2048 Hz.

2.4 | Data analysis

RT was measured as the interval between imperative stimulus onset and microswitch closure. The percentage of errors was calculated separately for the Go-congruent, Go-incongruent and No-go trials. The percentage of time-outs was calculated for the Go-congruent and Go-incongruent trials. For stimulus-locked ERP averaging, EEG epochs ranging from −300 to 700 ms relative to the imperative stimulus onset were segmented for each response type, as we investigated N2 and P3 after imperative stimulus onset (e.g., Fallgatter & Strik, 1999; Iannaccone et al., 2015; Jodo & Kayama, 1992; Pfefferbaum et al., 1985). For EMG-locked ERP averaging, EEG epochs ranging from −300 to 500 ms relative to the EMG onset were segmented in each response type because we investigated the ERN and Pe after the EMG onset (e.g., Falkenstein et al., 1990; Gehring et al., 1990). The trials were sorted based on the response type (pure-correct, partial-errors and overt-errors) as well as the stimulus condition (congruent vs. incongruent). Thus, stimulus-locked ERPs were separately averaged for pure-correct and partial-error trials, whereas EMG-locked ERPs were separately averaged for overt-error and partial-error trials. Partial errors in incongruent trials were characterized by rectified EMG activity of the incorrect response hand that did not lead to a switch closure, which was followed by corrective EMG activity within 250 ms. Partial errors in No-go trials were detected according to the rectified EMG activity of the No-go correct-trial limb. The EEG was re-referenced using mean activities across all scalp electrodes (i.e., average reference) and band-pass filtered .1–30 Hz (roll off 12 dB). Ocular artefacts were corrected using an algorithm developed by Gratton et al. (1983), which implements a Brain Vision Analyser for users. This algorithm corrects ocular artefacts using regression analysis (Hoffmann & Falkenstein, 2008). We excluded trials from ERP averaging in which the response time was less than 100 ms, or EEG

voltages exceeding a threshold of 75 μV during the recording epoch.

To determine EMG onset, we identified a deflection of EMG activities that first exceeded 4 SD of the rectified EMG derived from a baseline window (i.e., −700 to −550 ms before the EMG onset). We then backward searched the nearest negative peak from the exceeding point using a semi-automatic macro implemented in the Brain Vision Analyser. When we found any invalid EMG onsets with visual inspection, we corrected them manually, which allowed us to detect incorrect EMG activities (i.e., partial errors) (Maruo et al., 2017; Masaki & Segalowitz, 2004; Vidal et al., 2000). Adopting the extension of the middle fingers as a response, we appropriately detected small EMG activities, such as partial errors, because the extensor digitorum muscles involved in finger extensions existed more surface and were more suitable for surface-EMG recordings than the flexor digitorum superficialis muscles involved in finger flections. Thus, our procedure excluded partial errors from the ERPs in purely correct trials.

The mean voltage amplitudes were determined using a collapsed localizer approach (Luck & Gaspelin, 2017). The difference waveforms collapsed across all individuals. The time windows were 230–330 ms for N2 and 330–480 ms for P3, respectively, according to the time range of the ERP activities of interest. The mean voltage during the 100 ms before imperative stimulus onset served as the baseline voltage. For the EMG-locked ERPs (i.e., ERN and Pe), we calculated the mean amplitude of ERN at the midline frontocentral (FCz) in a time window ranging from 100 to 200 ms following EMG onset. The baseline was defined as the mean voltage from −300 to −200 ms before EMG onset. For the early Pe, we calculated mean amplitudes at midline central (Cz) in a time window ranging from 150 to 250 ms. For the late Pe, we calculated mean amplitudes at the midline parietal (Pz) in a time window ranging from 250 to 350 ms.

First, N2 and P3 were compared among the pure-correct trials. Both N2 and P3 amplitudes were subjected to one-way repeated-measures analysis of variance (ANOVA) with factor trial type (Go-congruent correct/Go-incongruent pure-correct/No-go pure-correct) using JASP (Vearsion 0.16.1; JASP Team, 2022). In addition, both N2 and P3 were compared between partial-error and pure-correct trials. Both N2 and P3 amplitudes were subjected to one-way repeated-measures ANOVA with factor trial type (No-go pure-correct/No-go partial-error/Go-incongruent pure-correct/Go-incongruent partial-error). However, both ERN and Pe amplitudes were subjected to two-way repeated-measures ANOVA with factor trial type (incongruent/No-go) and error type (full/partial). Degrees of freedom for all F-ratios were adjusted using the Greenhouse–Geisser procedure; however, the original degrees of freedom were reported with the epsilon value where required. Bonferroni correction was applied for post-hoc comparisons. A post-hoc power analysis was conducted using G*Power 3.1 to determine if the study had sufficient power to identify a significant main effect (Faul et al., 2007). We obtained power values of .17, .80 and .99 for small ($f = .10$), medium ($f = .25$) and large effect sizes ($f = .40$), respectively.

3 | RESULTS

3.1 | Performance measures

A paired t test revealed that RT was significantly longer in incongruent trials than in congruent trials (congruent: 302 ms, SEM = 3.6; incongruent: 360 ms, SEM = 2.8; $t(23) = 16.29$, $p = .001$, $d = 3.40$). One-way ANOVA revealed a main effect of error type ($F(2, 44) = 47.10$, $p = .001$, $p\eta^2 = .68$). The error rate in the incongruent trials was significantly higher than that in the congruent trials (congruent: 2.6%; SEM = .5; incongruent: 16.0%; SEM = 1.7; $t(22) = 7.82$, $p = .001$, $d = 1.63$). The error rate in the No-go trials (18.8%, SEM = 1.8) was significantly higher than that in the congruent trials ($t(22) = 9.39$, $p = .001$, $d = 1.96$). The error rate did not differ between the incongruent and No-go trials ($t(22) = 1.58$, $p = .37$, $d = .12$). A paired t test revealed that the time-out rate was significantly higher in incongruent trials than in the congruent trials (congruent: 2.3%, SEM = .4; incongruent: 5.3%, SEM = .7; $t(22) = 3.81$, $p = .001$, $d = .79$).

3.2 | Stimulus-locked ERP

3.2.1 | Comparison among pure-correct trials

Figure 2 depicts the stimulus-locked ERPs at FCz for the pure-correct trials. N2 did not emerge over the frontocentral regions in the pure-correct trials. The average number of trials for the pure-correct N2 was 203.3 (SEM = 1.9) in the Go-congruent, 97.4 (SEM = 5.5) for Go-incongruent and 30.9 (SEM = 3.3) for No-go trials, respectively. The minimum number of averages for the pure-correct N2 was 182 in the Go-congruent, 67 in the Go-incongruent and 7 in the No-go trials. The mean N2 amplitudes on Go-congruent trials, No-go trials and incongruent trials were −.16 μV (SEM = .55), .95 μV (SEM = .68), and −.88 μV (SEM = .63), respectively. A one-way ANOVA showed a main effect of trial type

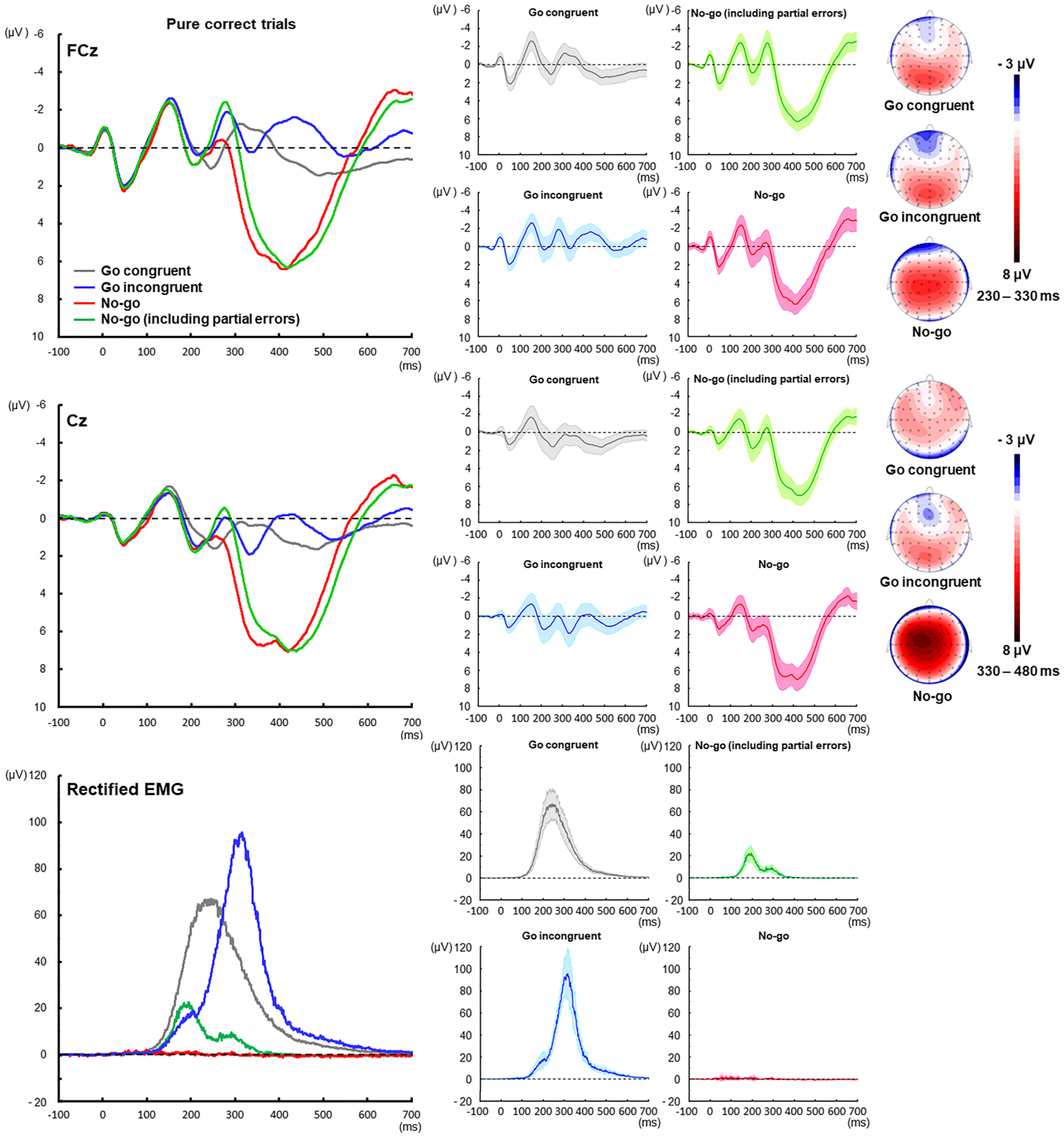

FIGURE 2 Stimulus-synchronized grand averaged waveforms ($n = 23$) at FCz and Cz on pure-correct trials (time window 230 to 330 ms for the N2 and 330 to 480 ms for the P3). We compared the stimulus-synchronized event-related potentials (ERPs) among three pure-correct trials (i.e., excluding partial errors). The waveform for No-go (including partial errors) is only for illustrative purposes. Rectified EMG waveforms for both overt responses and partial errors (i.e., an incorrect hand response) are also shown. Note that the initial incorrect EMG waveform is followed by a corrective response for No-go partial errors (green lines). Waveforms in the middle panels show individual ERPs with 95% confidence intervals. Topographies represent activities across the time window from 230 to 330 ms for the N2 and 330 to 480 ms for the P3. Cz, midline central; EMG, electromyogram; FCz, midline frontocentral

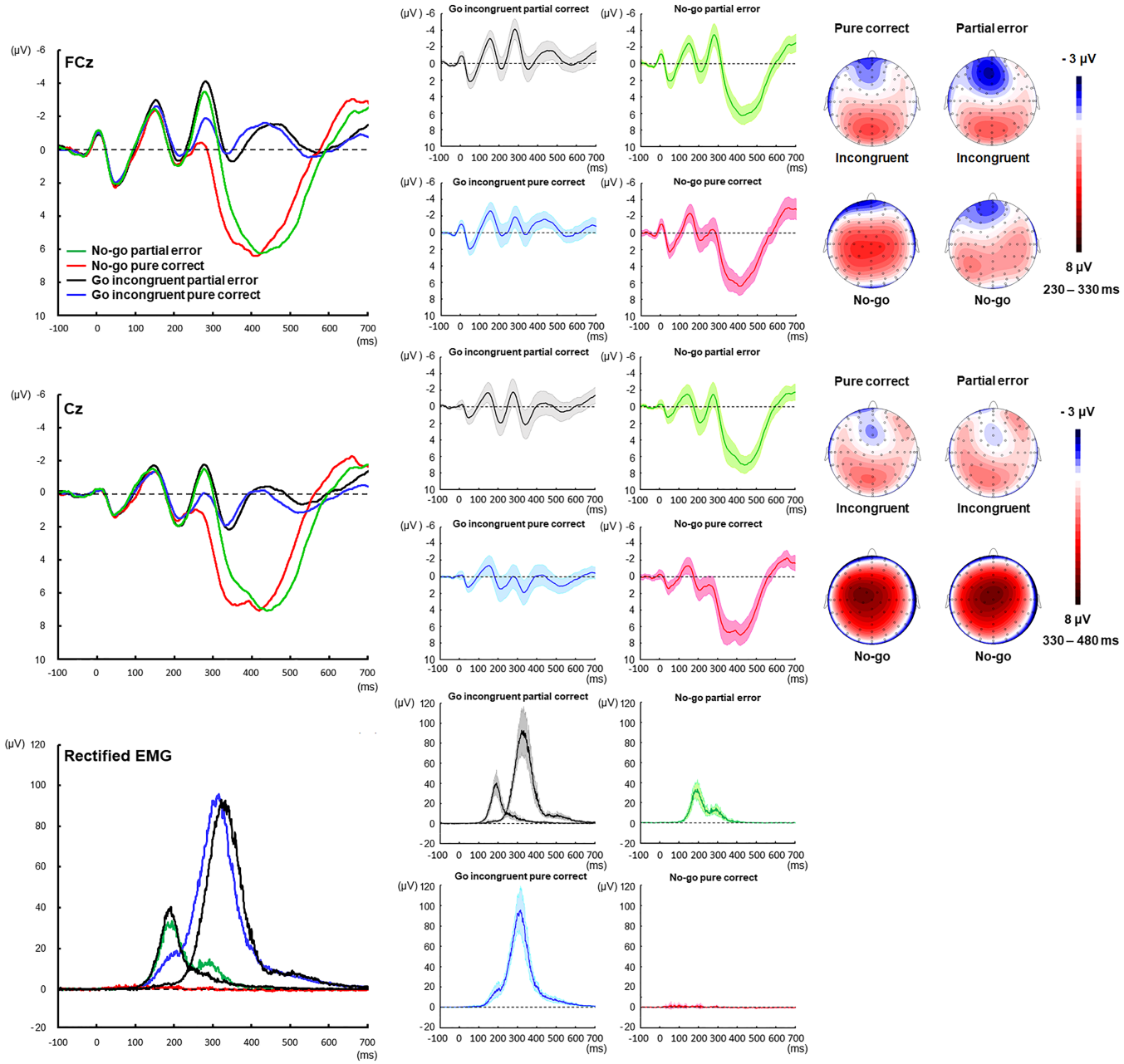

FIGURE 3 Stimulus-synchronized grand averaged waveforms ($n = 23$) at FCz and Cz on pure-correct trials and partial-error trials (time window 230 to 330 ms for the N2 and 330 to 480 ms for the P3). Rectified EMG waveforms for both overt responses and partial errors (i.e., an incorrect hand response) are also shown. Note that the initial incorrect EMG waveform is followed by a corrective response for Go-incongruent partial errors (black lines). We compared the stimulus-synchronized event-related potentials (ERPs) between partial-error trials and pure-correct trials. Waveforms in middle panels show individual ERPs with 95% confidence intervals. Topographies represent activities across the time window from 230 to 330 ms for the N2 and 330 to 480 ms for the P3. Cz, midline central; EMG, electromyogram; FCz, midline frontocentral

($F(2, 44) = 8.89$, $p = .002$, $p\eta^2 = .29$). Post-hoc tests revealed that N2 was larger in the Go-congruent trials than in the No-go trials ($t(22) = 2.53$, $p = .04$, $d = .53$). N2 was larger in the Go-incongruent trials than in the No-go trials ($t(22) = 4.19$, $p = .001$, $d = .87$).

Figure 2 also depicts P3 at Cz for the pure-correct trials. Mean P3 amplitude at Cz on Go-congruent, Go-incongruent and No-go trials were .96 μV (SEM = .49), 6.50 μV (SEM = .60) and .45 μV (SEM = .60), respectively. A one-way ANOVA showed a main effect of trial type ($F(2, 44) = 118.51$, $p = .001$, $p\eta^2 = .84$). Post-hoc tests revealed that P3 was larger in the No-go trials than in both the congruent ($t(22) = 12.72$, $p = .001$, $d = 2.65$) and Go-incongruent trials

($t(22) = 12.87$, $p = .001$, $d = 2.89$). P3 did not differ between the congruent and incongruent trials ($t(22) = 1.16$, $p = .76$, $d = .24$).

3.2.2 | Comparison between partial-error trials and pure-correct trials

Figure 3 depicts the N2 at FCz for the No-go pure-correct, No-go partial-error, Go-incongruent pure-correct and Go-incongruent partial-error trials. The average number of trials for the N2 in the No-go pure-correct, No-go partial-error, Go-incongruent pure-correct and Go-incongruent partial-error trials were, in order, 30.9 (SEM = 3.3), 53.5 (SEM = 2.6), 97.4 (SEM = 5.5) and 78.0 (SEM = 4.9), respectively. The minimum number of averages for N2 in the No-go pure-correct, No-go partial-error, Go-incongruent pure-correct and Go-incongruent partial-error trials were 7, 42, 67 and 50, respectively. A one-way ANOVA revealed a main effect of trial type ($F(3, 66) = 25.73$, $p = .001$, $p\eta^2 = .54$). Post-hoc tests showed that N2 in Go-incongruent partial-error trials ($M = -2.32$ μV, SEM = .58) was larger than in both No-go pure-correct ($t(22) = 8.42$, $p = .001$, $d = 1.76$) and Go-incongruent pure-correct trials ($t(22) = 3.72$, $p = .003$, $d = .78$). In addition, N2 was larger in No-go partial-error trials ($M = -1.53$ μV, SEM = .61) than in No-go pure-correct trials ($t(22) = 6.38$, $p = .001$, $d = 1.33$). N2 was larger in Go-incongruent pure-correct trials than in No-go pure-correct trials ($t(22) = 4.70$, $p = .001$, $d = .98$). The N2 amplitudes did not differ between the Go-incongruent pure-correct trials and No-go partial-error trials ($t(22) = 1.68$, $p = .59$, $d = .35$) and between No-go partial-error trials and Go-incongruent partial-error trials ($t(22) = 2.04$, $p = .27$, $d = .43$).

Figure 3 also depicts P3 at Cz for each trial. A one-way ANOVA revealed a main effect of trial type ($F(3, 66) = 110.21$, $p = .001$, $p\eta^2 = .83$). Post-hoc tests showed that P3 was larger in the No-go pure-correct trials than in the Go-incongruent pure-correct trials ($t(22) = 13.25$, $p = .001$, $d = 2.76$), larger in No-go partial-error trials ($M = 6.09$ μV, SEM = .59) than in the Go-incongruent partial-error trials ($M = .43$ μV, SEM = .71, $t(22) = 12.43$, $p = .001$, $d = 2.59$), larger in No-go partial-error trials than in Go-incongruent pure-correct trials ($t(22) = 12.37$, $p = .001$, $d = 2.58$), and larger in No-go pure-correct trials than in Go-incongruent partial-error trials ($t(22) = 13.32$, $p = .001$, $d = 2.78$). The P3 amplitudes did not differ between the No-go pure-correct and No-go partial-error trials ($t(22) = .89$, $p = 1.00$, $d = .18$) or between Go-incongruent pure-correct trials and Go-incongruent partial-error trials ($t(22) = .06$, $p = 1.00$, $d = .01$).

3.3 | EMG-locked ERP

Figure 4 depicts the ERN at FCz for incongruent overt-error, incongruent partial-error, No-go overt-error and No-go partial-error trials. The average number of trials for the ERN in the incongruent overt-error, incongruent partial-error, No-go overt-error and No-go partial-error trials were 38.3 (SEM = 3.9), 78.3 (SEM = 4.9), 22.6 (SEM = 2.1) and 54.0 (SEM = 2.9), respectively. The minimum number of averages for the ERN in the incongruent overt-error, incongruent partial-error, No-go overt-error and No-go partial-error trials were 15, 32, 8 and 26, respectively. There was a significant interaction between error type and trial type ($F(1, 22) = 18.39$, $p = .001$, $p\eta^2 = .46$). Post-hoc tests revealed that ERN in the incongruent overt-error trials was larger than in both incongruent partial-error trials ($t(22) = 6.62$, $p = .001$) and No-go partial-error trials ($t(22) = 9.99$, $p = .001$). ERN in No-go overt-error trials was larger than in both incongruent partial-error trials ($t(22) = 5.57$, $p = .001$) and No-go partial-error trials ($t(22) = 10.48$, $p = .001$). The ERN in incongruent partial-error trials was larger than that in No-go partial-error trials ($t(22) = 5.57$, $p = .001$). ERN amplitudes did not differ between incongruent and No-go overt-error trials ($t(22) = .63$, $p = 1.00$).

Figure 4 also depicts the early Pe waveforms at Cz in each trial. There was a significant interaction between error type and trial type ($F(1, 22) = 65.43$, $p = .001$, $p\eta^2 = .75$). Post-hoc tests revealed that early Pe in No-go partial-error trials was larger in No-go overt-error trials ($t(22) = 10.10$, $p = .001$), incongruent overt-error trials ($t(22) = 9.76$, $p = .001$) and incongruent partial-error trials ($t(22) = 10.34$, $p = .001$). Early Pe in incongruent partial-error trials was larger in both incongruent overt-error trials ($t(22) = 3.79$, $p = .004$) and No-go overt-error trials ($t(22) = 3.71$, $p = .005$). The early Pe did not differ between the No-go overt-error trials and incongruent overt-error trials ($t(22) = .06$, $p = .96$).

Figure 4 also depicts the late Pe waveforms at Pz in each trial. A two-way ANOVA revealed a main effect of error type ($F(1, 22) = 4.72$, $p = .04$, $p\eta^2 = .18$). Post-hoc tests revealed that late Pe was larger in the overt-error trials than in the partial-error trials ($t(22) = 2.17$, $p = .04$, $d = .45$). A two-way ANOVA revealed a main effect of trial type ($F(1, 22) = 7.94$, $p = .01$, $p\eta^2 = .27$). Post-hoc tests revealed that late Pe was larger in the No-go trials than in the incongruent trials ($t(22) = 2.82$, $p = .01$, $d = .59$). The interaction between trial type and error type was not significant ($F(1, 22) = 3.32$, $p = .08$, $p\eta^2 = .13$).

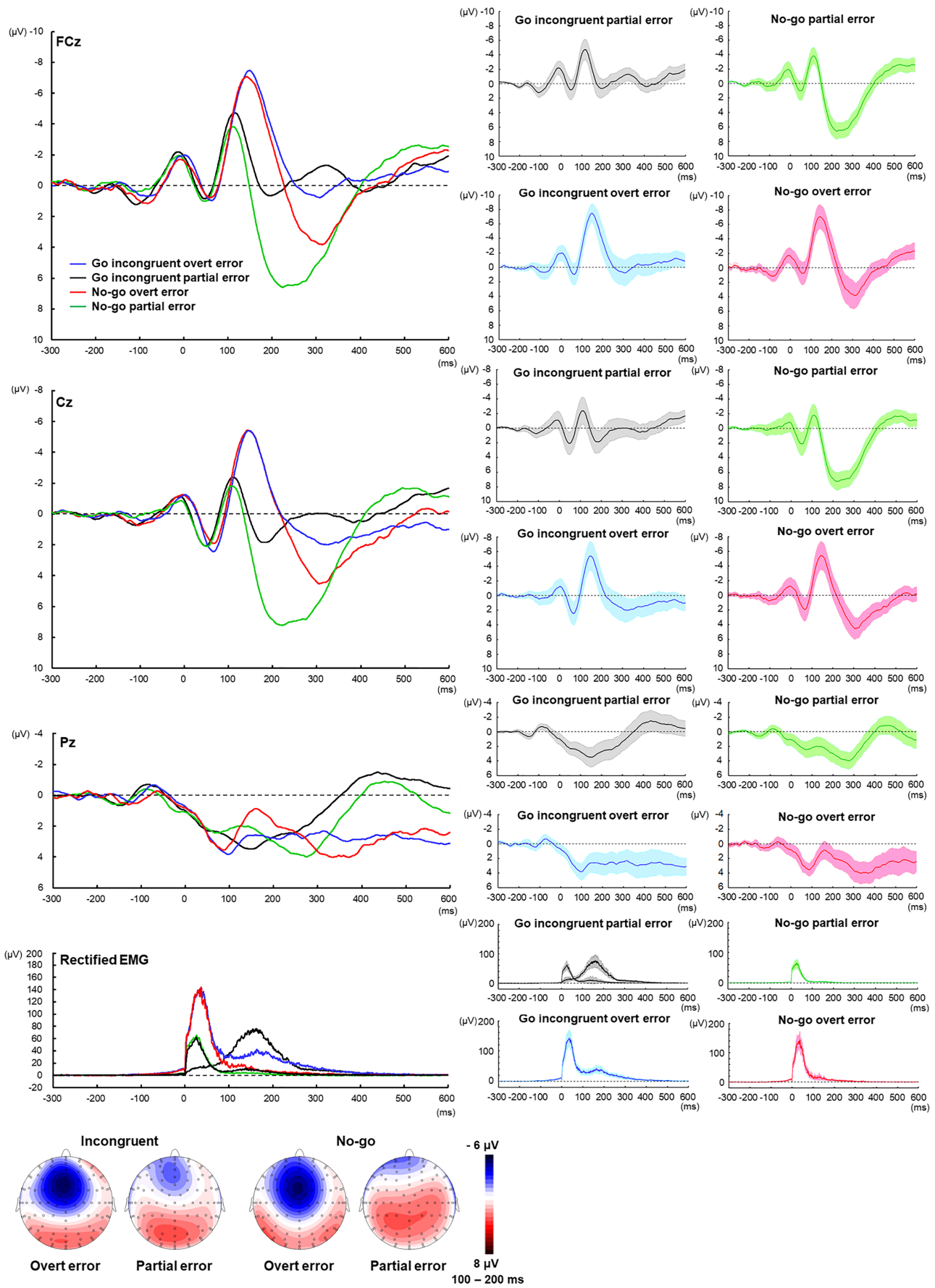

FIGURE 4 Legend on next page.

FIGURE 4 EMG-synchronized grand averaged waveforms ($n = 23$) at FCz, Cz and Pz and rectified EMG waveforms on overt-error trials and partial-error trials (time window 100 to 200 ms for the error-related negativity [ERN]). Rectified EMG waveforms are also shown. Note that the initial incorrect EMG waveform is followed by a corrective response for Go-incongruent partial errors (black lines). Waveforms in the right panels show individual event-related potentials (ERPs) with 95% confidence intervals. Topographies represent activities across the time window from 100 to 200 ms for the ERN. Cz, midline central; EMG, electromyogram; FCz, midline frontocentral

4 | DISCUSSION

We investigated whether partial errors could yield No-go N2 and No-go P3 using a Go/No-go flanker task. We found longer RTs and higher error rates in incongruent trials than in congruent trials. Although N2s did not differ among pure-correct trials, P3s were larger in both No-go pure-correct and No-go partial-error trials than in Go correct trials and incongruent pure-correct trials. In addition, No-go N2 was larger in partial-error trials than in pure-correct trials. Furthermore, ERN was larger in the overt-error trials than in the partial-error trials.

We identified partial errors embedded in muscular activities for both the No-go and incongruent trials. The fact that the stimulus-locked ERPs did not differ among pure-correct trials suggests that the increased No-go N2s reported in previous studies might be due to the contamination of ERN elicited by partial errors. The current result is consistent with our previous study (Maruo et al., 2017), in which N2 did not differ between Go correct trials and No-go pure-correct trials. Most studies of No-go N2 have asserted that No-go N2 reflects response inhibition (Falkenstein et al., 1999; Kok, 1986) and/or response conflict (Nieuwenhuis et al., 2003; Yeung et al., 2004). If so, the No-go N2 in No-go pure-correct trials should have been larger than in Go correct trials, even after partial errors were excluded from ERP averaging. This was not the case in this study. Thus, it is plausible that the increased No-go N2s in the No-go trials in previous studies might have reflected a composite waveform. Our results cast doubt on the existence of the so-called No-go N2, which is believed to represent response inhibition processes.

It is plausible that the increased No-go N2 in our study was due to a superimposition of the ERN elicited by partial errors (Maruo et al., 2017; Masaki et al., 2012; Vidal et al., 2000). This suggests that the error-monitoring system detects covert EMG activities in the absence of overt responses. Nguyen et al. (2016) found a larger No-go N2 in partially inhibited trials (i.e., partial-error trials) than in successfully inhibited trials. Maruo et al. (2017) also found a larger No-go N2 in partial-error trials than in pure-correct trials. Although Nguyen et al. (2016) did not record EMG activities, the No-go N2 on partial-error trials may have reflected the error processing.

Some studies relying on the framework of the conflict-monitoring theory have reported larger N2 amplitudes in high-conflict trials than in low-conflict trials (Iannaccone et al., 2015; Nieuwenhuis et al., 2003). The conflict-monitoring theory asserts that the pre-response conflict in correct trials results in a larger N2. However, we did not find any difference in N2 amplitudes between the incongruent and No-go trials, contrary to the conflict-monitoring theory. Previous studies of the No-go N2 that advocated conflict-monitoring theory did not remove the contamination of partial errors from ERP averaging. Even so, it should be noted that we cannot completely rule out the conflict-monitoring account in No-go N2 because we did not apply the same computer simulation as in a previous study to our dataset (Yeung et al., 2004).

P3 amplitudes were larger in the No-go trials than in the Go-congruent and incongruent trials. These results are consistent with the notion that the No-go P3 reflects inhibition of overt movement (Nguyen et al., 2020; Smith et al., 2013) and the complete inhibition of the planned response (Nguyen et al., 2016). Although we found larger P3s in the No-go trials, there was no difference in P3 amplitudes between the No-go pure-correct and No-go partial-error trials. Expanding the findings of Smith et al. (2013) that No-go P3 was enhanced by the inhibition of overt movements, our results further suggest that No-go P3 can be enhanced by both the complete inhibition of response (i.e., no peripheral manifestation of erroneous activities) and withdrawal of implicit erroneous activities (i.e., partial errors).

Given that the ERN reflects response conflict (Yeung et al., 2004), the ERN should have been larger in incongruent trials than in No-go trials, in accordance with the findings of Braver et al. (2001). However, this was not the case in the present study. Our results may support the online monitoring account (Carbonnell & Falkenstein, 2006; Yordanova et al., 2004). Carbonnell and Falkenstein (2006) suggested that ERN amplitudes might reflect neither the degree of conflict nor error, whereas ERN latency might reflect error processing.

For the EMG-locked ERPs, the early Pe was larger for No-go partial errors than for both overt errors and incongruent partial errors. Previous studies have identified two distinct positive components associated with

error processing: early and late Pe (Endrass et al., 2007, 2012; Maruo et al., 2017; O'Connell et al., 2007). They suggested that the early Pe showing frontocentral distributions may reflect error monitoring and error awareness (Endrass et al., 2007, 2012; O'Connell et al., 2007; Thurm et al., 2020). However, these notions cannot unambiguously explain why the early Pe was the largest in No-go partial-error trials in our study. Furthermore, Pe cannot be elicited by partial errors because the participants do not recognize their own partial errors in most cases, and thus, evaluate those trials as correct (Burle et al., 2008). Although a previous study suggested that the participants might consciously detect a few partial errors (Rochet et al., 2014), the largest Pe in the No-go partial error trials in our study is difficult to be explained by the transiently induced consciousness account. Considering the possibility of overlap between stimulus- and response-related activities in our Go/No-go task, it is plausible that the enhanced early Pe in No-go partial error trials might reflect response inhibition processes provoked by the No-go stimulus rather than error monitoring and error awareness. This interpretation is also supported by the result that the Pe was more obvious for No-go trials than for incongruent trials regardless of error types (i.e., overt and partial errors). Further research is needed to elucidate the functional significance of the early Pe.

In conclusion, partial errors may affect standard No-go N2. The present findings provide further evidence that the previously reported No-go N2 may be due to an overlap of the ERN elicited by partial errors. In contrast, No-go P3s in both the pure-correct and partial error trials appeared to reflect the response inhibition processes. Our results show that analysing partial errors is useful in determining the functional significance of No-go N2 and No-go P3. The present findings provide further evidence that the widely accepted No-go N2 might merely represent the partial-error ERN or at least reflect contamination of the ERN. Furthermore, No-go P3 may represent purely inhibition processes.

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CONFLICT OF INTEREST

The authors declare no competing financial interests.

AUTHOR CONTRIBUTIONS

Yuya Maruo performed the conceptualization, data curation, formal analysis, and writing of the original draft.

Hiroaki Masaki carried out the conceptualization, data curation, formal analysis, funding acquisition, project administration, and writing of the original draft.

PEER REVIEW

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DATA AVAILABILITY STATEMENT

The datasets generated for this study are available on request to the corresponding author.

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