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Impaired Conflict Detection Differentiates Suicide Attempters From Ideating Nonattempters: Evidence From Event-Related Potentials

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ABSTRACT

BACKGROUND: Existing research suggests that inhibitory control deficits may differentiate individuals who think about suicide from those who make a suicide attempt. However, no available research, to our knowledge, has examined whether suicidal behaviors are associated with disruptions in the ability to determine when inhibitory control is needed or the ability to engage inhibition of an inappropriate or maladaptive behavior. The current study utilized event-related potentials to investigate specific facets of inhibitory control and their associations with suicide attempt history among a heterogeneous clinical sample who reported current suicidal ideation.

METHODS: Ideators with no past suicide attempts (n = 46) and those with a history of suicide attempts (n = 22) completed a complex go/no-go task. Raw waveforms and temporospatial principal components analysis were used to index conflict detection (i.e., ΔN2) and motor inhibition (i.e., ΔP3a). Behavioral performance indices were also examined.

RESULTS: Suicide attempters exhibited deficits in detecting the need for inhibitory control, as indexed by a more positive ΔN2 factor, than did ideating nonattempters, even when accounting for psychiatric comorbidity and age. However, these results only emerged in the principal components analysis–derived latent factor. No differences in behavioral performance or ΔP3a amplitude emerged.

CONCLUSIONS: A relative inability to detect when to inhibit a maladaptive behavior, but not the ability to engage motor inhibition to stop that behavior, may distinguish suicide ideators who make a suicide attempt from those who do not. However, future research with prospective designs are needed to determine how conflict detection deficits may contribute to the emergence or escalation of a suicidal crisis.

Keywords: Conflict detection, Event-related potentials, N2, Response inhibition, Suicide, Suicide attempts

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Suicide rates have increased markedly despite decades of research (1), with one half of all U.S. states experiencing >30% increases in suicide deaths (2). Although most individuals with suicidal ideation (SI) never make a suicide attempt (SA) (3,4), there are limited scientific data differentiating suicide attempters from those who think about suicide but do not make an SA (3,5). This gap has renewed urgency for suicide research within an ideation-to-action framework wherein the emphasis is placed on identifying factors that facilitate the transition from thinking about suicide to making an SA (3,5–9).

Inhibitory control is a facet of prefrontal cortex–driven cognitive control (10) that may have utility for understanding the progression from SI to SA (11,12). Inhibitory control is typically measured using go/no-go tasks in which individuals frequently press a button (i.e., go trials) and are infrequently required to withhold this response (i.e., no-go trials). At least 2 rapid, successive neural processes compose inhibitory control: 1) detecting the need for inhibition (i.e., conflict detection) (13–15) and 2) suppressing the inappropriate behavior (i.e., motor inhibition) (16,17). Thus, inhibitory control relies on both a warning signal for the need for inhibition (conflict detection) and the ability to engage inhibition to stop the behavioral response (motor inhibition).

Multiple theoretical perspectives suggest that impaired inhibitory control may enhance the likelihood that SI is translated into suicidal behaviors through difficulty stopping an undesirable behavior (i.e., enacting a suicide plan) in the context of an acute suicidal crisis (6,7,11,18). Indeed, accumulating evidence demonstrates poor inhibitory control among those with SA histories relative to psychiatric control subjects (11,12,19–21). A meta-analysis (21) and 2 recent studies (19–21) found that depressed attempters performed

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worse on inhibitory control tasks than depressed nonattempters did. A cross-national sample found that ideators with psychiatric disorders characterized by poor inhibitory control (e.g., substance use, conduct disorders) were at the greatest risk for making an SA (4). Another study found that suicide attempters, compared with ideating nonattempters, performed worse on measures of response inhibition across 14 empirical studies (12). Only one study, to our knowledge, has examined neural activity and SA history during an inhibitory control task; however, functional magnetic resonance imaging revealed null group differences in neural regions active during inhibition (20), perhaps because functional magnetic resonance imaging lacks the temporal resolution necessary to study neural processes that occur in rapid succession.

In contrast, event-related potential (ERP) methodology possesses high temporal resolution (22) and is ideally suited to examine rapid neural processes underpinning inhibitory control. ERPs are derived from electrocortical data and reflect the extent of neural activity during specific temporal stages of processing (i.e., conflict detection, motor inhibition). For instance, during go/no-go inhibition tasks, individuals must engage conflict detection to determine when a no-go stimulus is presented. This conflict detection signal is reflected by a frontal negative deflection peaking around 200 ms following no-go stimuli (i.e., the N2) such that a less negative N2 on no-go trials reflects a weak signal for the need for inhibitory control (13,15). In contrast, the P3a component derived from a go/no-go task is a frontocentrally maximal component peaking around 300 ms (23,24) that reflects greater cognitive resources devoted to inhibiting motor responses (16,17).

To our knowledge, no research to date has investigated whether suicide attempters exhibit specific disruptions of conflict detection and/or motor inhibition. However, there is evidence for those with suicidal behavior exhibiting worse conflict-related functional connectivity (25,26) in the same neural region associated with the conflict-related N2 component (dorsal anterior cingulate cortex) during similar tasks that require conflict detection but do not necessarily engage inhibitory control (27). To the extent that self-directed violence and other-directed violence represent nonorthogonal constructs (28), we might expect sucidal behavior to be linked with a blunted N2 but no difference in the P3a, as has been observed in offenders with impulsive aggressive tendencies (29,30). Taken together, these results suggest that conflict detection, and not motor inhibition, may underlie inhibitory control deficits among those who engage in violence, but it is important to note that there are no data to suggest that this specificity also applies to self-directed violence (i.e., SAs).

The current study addressed this gap by utilizing ERP methodology to examine differences in N2 (conflict detection) and P3a (motor inhibition) amplitudes during a go/no-go response inhibition task among current suicide ideators with and without a history of SAs. Based on theoretical (6,7,11,18) and empirical (12,19–21) evidence, we hypothesized that among individuals with current SI, SA history would predict worse accuracy indicating impaired inhibitory control. Given extant literature associating a blunted N2 with other-directed aggression (29,30), we hypothesized that SA history would be associated with a blunted (i.e., less negative) N2 specifically on no-go trials indicating poor conflict detection (13–15). Lastly, in line with literature showing impaired inhibitory control among attempters (12,19–21) and linking the P3a with motor inhibition (16,17), we hypothesized that those with SA histories would exhibit a reduced (i.e., less positive) P3a on no-go trials, representing weak engagement of cognitive resources supporting motor inhibition.

METHODS AND MATERIALS

The present sample comprised a subset (n = 75) of individuals with active SI (i.e., Beck Scale for Suicide Ideation [BSS] $>$ 0) (31,32) selected from a larger study including 280 individuals presenting for a randomized clinical trial assessing the efficacy of 2 computerized interventions for suicide risk (Depression and Anxiety Reduction Treatment for Suicide [DARTS]; NCT01941862). Of those with active ideation (n = 75), several were removed for not completing the task (n = 1), performance $\leq$50% (n = 1), or technical problems (n = 5), resulting in a final sample of 68 participants. Eligibility criteria for the parent study included being at least 18 years of age and elevating at least 1 of several risk factors for SI (i.e., thwarted belongingness, perceived burdensomeness, or current SI). Exclusion criteria were history of neurological conditions, uncorrected visual impairments, uncontrolled bipolar or other psychotic spectrum disorders, and imminent suicide risk requiring hospitalization.

Ages ranged from 18 to 74 years old (mean = 36.63, SD = 15.51). The sample was majority Caucasian (60.3%) followed by African American (20.6%), Asian (2.9%), Pacific Islander (1.5%), American Indian/Native American (1.5%), and Other (e.g., biracial; 13.2%). Veterans composed 35.3% of the sample. Nearly all participants (98.5%) met DSM-5 criteria for at least 1 diagnosis, with 67.6% meeting criteria for 2 or more psychiatric diagnoses (mean = 2.49, SD = 1.63).

Measurement

Suicidal Ideation. The BSS (33,34) is a 19-item self-report index of SI that was used to select ideators (non-0 scores) from the larger sample. The BSS has demonstrated strong psychometric properties in the past (33) and adequate internal consistency in the current study (α = .88).

SA History. The Suicide History Form (35) is a 5-item self-report measure that was used to identify individuals with at least 1 prior SA (n = 22; 32.4%) or no prior SAs (n = 46; 67.6%). The current study also validated SA histories with a clinician-administered structured interview (31,36). Positive and negative SA histories converged across assessment modality for nearly all participants (n = 65; 95.6% agreement).[1] Among attempters, the number of past SAs ranged from 1 to 6 (mean = 1.91, SD = 1.44).

[1]Three participants reported past SAs on the Suicide History Form but not during the clinical interview. All analyses revealed the same pattern of results when excluding these participants. Therefore, these participants were retained.

Psychiatric Diagnoses. The Structured Clinical Interview for DSM-5, Research Version (37) was administered by highly trained doctoral-level therapists and reviewed by a licensed clinical psychologist, with excellent interrater reliability (κ = .86). Frequencies of depressive disorder (major depressive disorder, persistent depressive disorder, not otherwise specified depressive disorder), posttraumatic stress disorder (PTSD), and substance use disorders (SUD) among ideators and attempters are presented in Table 1.

Experimental Stimuli

Go/No-Go Complex Paradigm. Go/no-go tasks are commonly used to index inhibitory control by requiring participants to frequently respond to stimuli (i.e., go trials) and infrequently withhold these prepotent responses (i.e., no-go trials). We utilized a complex go/no-go task that required working memory to detect the no-go trials (38). No-go trials were signaled by the repeated presentation of the same letter (e.g., the fifth letter in the sequence X-Y-X-Y-Y) (39–42), thus indexing inhibitory control in the context of working memory demands. Past research has found that complex go/no-go tasks produce reliable ERPs (38).

Our task included 126 go (75%) and 42 no-go (25%) trials over 7 blocks with distinct sets of letters. Instructions were presented at the beginning of each block. Go/no-go stimuli were presented for 296 ms followed by an 1150-ms response window then feedback for 1000 ms. Intertrial intervals lasted 900 ms. ERPs were derived from correct responses to ensure that only trials in which participants were engaged were used.

Stimulus Delivery and Physiological Response Measurement. Data were collected using Neuroscan Acquire software (Compumedics, Charlotte, NC), two 64-channel Neuroscan SynAmps RT amplifiers (Compumedics), and a Brain Vision actiCap 96-channel cap (Morrisville, NC) (1000-Hz sampling rate, with an online analog bandpass filter of 0.05–100 Hz). The online ground and reference were AFz and FCz, respectively. Electro-oculogram activity was collected using electrodes placed laterally to each eye and above/below the left eye. Impedances were below 10 kΩ throughout recording.

Data Preprocessing

Data were first downsampled to 250 Hz, and then high-pass (0.1 Hz; ripple = 0.05 dB, attenuation = 80 dB) and low-pass (40 Hz; ripple = 0.01 dB, attenuation = 40 dB) finite impulse response (FIR) filters were applied. Offline, data were rereferenced to the averaged mastoids (TP9 and TP10) and FCz was regenerated. Continuous data were separated into epochs using the full trial length and 100-ms prestimulus baseline correction (i.e., −100-ms to 2440-ms epochs). The Fully Automated Statistical Thresholding for EEG Artifact Rejection algorithm (43) was then used for artifact detection and rejection (see the Supplement).

Area Around the Peak Measurement. To mitigate the influence of latency variability on component measurement,[2] we utilized an area around the peak approach in which we identified the local peak and averaged all data points in the 20 ms preceding and following that peak (22). This strategy improves on the use of static time windows by allowing for individual differences in component latency by allowing for different time windows for each participant. To compute the N2 component, the local negative-going peak between 200 and 348 ms was identified, and then all data points ±20 ms at Fz were averaged. Finally, the P3a component was defined as the average of ±20 ms at FCz of the local positive-going peak between 296 and 500 ms (24). Split-half reliabilities were calculated by randomly splitting each subject's available trials into halves, computing the Pearson correlation between the mean amplitude of each half, and transforming the resulting coefficient into a Z score (44–46). This process was repeated 1000 times, then Z scores were recomputed into correlations and then corrected using the Spearman-Brown prophecy formula to obtain an internal consistency estimate (see Table 2). Reliability estimates were excellent for raw waveforms and poor to adequate for difference scores.

Temporospatial Principal Components Analysis ERP Measurement. Temporospatial principal components analysis (PCA) was also used to extract latent N2 and P3a components (47). In contrast to area around the peak measurements, PCA minimizes the influence of overlapping components on the N2 and P3a as well as component latency variability by extracting linear combinations of data points to distinguish consistent neurophysiological activity across time and sensors (47). PCA identifies latent N2 and P3a components while minimizing noise.

Temporospatial PCA was conducted using the ERP PCA Toolkit version 2.63 (48) using the full sample to maximize power for detecting the latent PCA factors. Per recommendations (48–50), a temporal PCA with promax rotations, covariance matrix, and Kaiser normalization (48,49,51) was conducted first. Variables were time points (−100 ms to 1150 ms; i.e., prestimulus through the response window) and observations were participants, trial types (i.e., go vs. no-go), and sensors. Twelve temporal factors were extracted based on Scree plots (52). Next, a spatial PCA was conducted with infomax rotation (48,49); recording sites were entered as variables; and participants, trial types (i.e., go, no-go), and temporal factors were entered as observations. Five spatial factors emerged, yielding a total of 60 unique temporospatial factors.

[2]Predefined time windows for the N2 and P3a were inadequate for several reasons: 1) Most prior studies utilized simple (as opposed to complex) go/no-go tasks and/or samples not readily comparable to the present study, such as healthy or diagnostically homogenous groups (38,53), making a prior time-window predictions tenuous; 2) Psychiatric disruptions can be associated with latency variability for both the N2 and P3a, which would render predefined time windows across participants insufficient to measure individual differences in amplitude; and 3) Measurement of the N2 is obfuscated by neighboring positive-going deflections (i.e., the P2 and P3a).

Table 1. Descriptive Statistics for Attempters and Nonattempters and Bivariate Correlations

	SA Group (n = 22), Mean (SD) or n Endorsed	No-SA Group (n = 46), Mean (SD) or n Endorsed	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. SA History	–	–	–																		
2. Depressive Disorders	13	28	−.02	–																	
3. PTSD	5	16	−.12	.22[a]	–																
4. SUD	5	8	.06	−.14	−.08	–															
5. Age, Years	42.91 (16.09)	33.63 (14.45)	.28[b]	.24[b]	.07	−.05	–														
6. Accuracy, % Correct	91.72 (5.23)	93.74 (4.82)	−.19	.01	.01	−.05	−.36[c]	–													
7. Reaction Time, ms	521.10 (88.08)	503.49 (84.34)	.10	.11	−.17	.07	.37[c]	−.47[d]	–												
8. Raw ΔN2	0.38 (1.90)	−0.18 (2.61)	.11	.20	.36[c]	−.06	.19	−.04	.01	–											
9. Raw No-Go N2	0.68 (3.69)	−0.07 (3.63)	.10	.30[b]	.33[c]	−.13	.35[c]	−.03	.11	.66[d]	–										
10. Raw Go N2	0.71 (3.70)	0.47 (3.51)	.03	.23[a]	.12	−.12	.30[b]	.00	.13	<.001	.75[d]	–									
11. Raw ΔP3	0.56 (4.41)	−0.27 (3.40)	.11	.11	.13	−.18	−.20	.20	−.34[c]	.11	.16	.12	–								
12. Raw No-Go P3	9.35 (5.85)	9.52 (6.02)	−.01	.00	.00	−.17	−.12	.32[c]	−.29[b]	−.01	.42[d]	.56[d]	.61[d]	–							
13. Raw Go P3	3.51 (3.77)	4.46 (4.64)	−.10	−.08	−.10	−.08	.00	.25[b]	−.11	−.09	.40[d]	.62[d]	<.001	.79[d]	–						
14. Latent ΔN2	0.71 (1.30)	−0.34 (1.73)	.30[b]	.14	.21[a]	−.04	.21[a]	.00	.02	.47[d]	.21[a]	−.14	−.12	−.20[a]	−.16	–					
15. Latent No-Go N2	1.87 (2.39)	0.12 (2.71)	.30[b]	.22[a]	.18	−.11	.27[b]	−.13	.20	.31[b]	.42[d]	.30[b]	−.17	−.03	.09	.62[d]	–				
16. Latent Go N2	1.98 (2.17)	1.15 (2.71)	.15	.17	.06	−.11	.17	−.17	.24[a]	.02	.38[c]	.48[d]	−.12	.12	.25[b]	<.001	.79[d]	–			
17. Latent ΔP3a	0.43 (3.18)	−0.20 (3.25)	.09	.14	.25[b]	−.17	−.22[a]	.32[c]	−.30[b]	.26[b]	.20[a]	.05	.73[d]	.51[d]	.08	−.01	−.01	.00	–		
18. Latent No-Go P3a	5.61 (5.44)	6.25 (5.67)	−.05	.03	−.01	−.20	−.18	.34[c]	−.22[a]	.01	.42[d]	.55[d]	.49[d]	.93[d]	.80[d]	−.19	−.01	.13	.58[d]	–	
19. Latent Go P3a	1.04 (3.87)	2.32 (4.83)	−.13	−.06	−.19	−.12	−.07	.19	−.06	−.17	.37[c]	.64[d]	.08	.78[d]	.92[d]	−.23[a]	−.01	.16	<.001	.82[d]	–

The SA group includes current suicide ideators with a past suicide attempt. The No-SA group includes current suicide ideators with no past suicide attempts. Depressive disorders include presence/absence of major depressive disorder, persistent depressive disorder, or mood disorder not otherwise specified. Raw N2 and P3a components were scored using the area around the peak. Latent N2 and P3a components were extracted using temporospatial principal components analysis.

ΔN2, Residualized difference between no-go N2 and go N2; ΔP3a, residualized difference between no-go P3a and go P3; PTSD, posttraumatic stress disorder; SA, suicide attempt; SUD, substance use disorder.

[a] $p < .10$.
[b] $p < .05$.
[c] $p < .01$.
[d] $p < .001$.

Table 2. Spearman-Brown Corrected Split-Half Reliabilities for Area Around the Peak Raw Amplitudes

Trial Type	Spearman-Brown Corrected *r* (95% CI)
Raw ΔN2	.45 (.10, .66)
Raw N2 No-Go	.90 (.83, .94)
Raw N2 Go	.97 (.96, .98)
Raw ΔP3	.67 (.46, .80)
Raw P3a No-Go	.94 (.90, .96)
Raw P3a Go	.97 (.94, .98)

Split-half reliabilities were calculated by randomly splitting each subject's available trials into halves, computing the Pearson correlation between the mean amplitude of each half, and transforming the resulting coefficient into a *Z* score (44–46). This process was repeated 1000 times, then *Z* scores were recomputed into correlations and then corrected using the Spearman-Brown prophecy formula to obtain an internal consistency estimate.

ΔN2, residualized difference score of no-go N2 regressed on go N2; ΔP3a, residualized difference score of no-go P3a regressed on go P3a; CI, confidence interval.

As recommended (51), each factor accounting for >1% variance (n = 17) was converted into microvolt-scaled waveforms (50) and assessed for resemblance to the N2 and P3a components. Despite TF6SF1 (1.72% variance) being a frontal positivity (as opposed to negativity), this factor most resembled all other characteristics expected in the N2 (13,15), including peaking at 228 ms at Fz and the expected trial-type differentiation ($F_{1,67}$ = 12.28, p = .001) such that no-go trials (mean = 0.69, SD = 2.72) were significantly less positive (i.e., more negative) than go trials (mean = 1.42, SD = 2.56). Notably, the relative positivity of the N2 elicited by the complex go/no-go task has been replicated in a distinct sample (53). The factor most resembling the P3a (TF1SF1; 14.60% variance) peaked at Cz at 336 ms and, as expected (16,17), was significantly more positive on no-go (mean = 6.04, SD = 5.56) relative to go (mean = 1.90, SD = 4.55) trials ($F_{1,67}$ = 112.34, p < .001).

Data Analytic Plan

First, simple group differences in demographic variables and SI severity were examined. Variables differing across groups (attempters, nonattempters) were included as covariates to probe significant simple group differences in later analyses. Because of associations with inhibitory control and suicide, depressive disorder (54), PTSD (55,56), and SUD (57,58) were included as covariates regardless of group differences. Independent samples *t* tests (i.e., Student's *t* tests) were used for continuous variables and logistic regression for dichotomous outcomes. Group differences in overall task accuracy (go and no-go trials) and go trial reaction time were also examined using independent samples *t* tests.

Trial-type differential neural responding (i.e., N2 and P3a on go/no-go trials) was examined using identical progressions of analyses for all raw and latent N2 and P3a amplitudes. First, the simple group differences were examined using a repeated measures analysis of variance (RM-ANOVA) in which trial type (go, no-go) served as a within-subjects factor and group (attempters, nonattempters) served as a between-subjects factor. Independent samples *t* tests were used to evaluate simple group differences in go and no-go amplitudes. No covariates were included during this step.

Significant trial type × group simple interactions were then probed to evaluate whether the observed simple effects held when including relevant covariates. For these analyses, repeated measures analysis of covariance (RM-ANCOVA) was utilized with relevant demographics, depressive disorder, PTSD, and SUD diagnostic status entered as covariates. Significant trial type × group interactions in the RM-ANCOVA were further probed using a series of hierarchical linear regressions to assess the directionality of observed effects. For all analyses, step 1 included SA history and psychiatric comorbidity variables. Relevant demographic variables were entered in step 2. Dependent variables were, in order, unstandardized residualized difference between no-go, relative to go, trials (e.g., no-go regressed on go; ΔN2) per recommendations (59), no-go component of interest (e.g., no-go N2), and go component of interest (e.g., go N2).

RESULTS

Group Differences in Demographics, Psychiatric Comorbidity, and SI

Demographics. Groups did not differ based on yearly family income (t_{65} = 1.04, p = .301), biological sex (odds ratio [OR] = 0.64, p = .394), or veteran status (OR = 1.43, p = .504). Those with prior SAs (mean = 42.91, SD = 14.45) were significantly older than those with no SAs (mean = 33.63, SD = 16.09) (t_{66} = −2.39, p = .020). Therefore, age was entered as a covariate for the primary analyses.

SI Severity. Those with prior SAs (mean = 7.55, SD = 6.29) and no SAs (mean = 6.57, SD = 6.18) did not differ in SI severity (t_{66} = −0.61, p = .545).

Psychiatric Comorbidity. Those with prior SAs were not more likely to meet DSM-5 diagnostic criteria for depressive disorders (OR = 0.93, p = .888), PTSD (OR = 0.55, p = .318), or a SUD (OR = 1.40, p = .602).

Equality of Variances. No significant findings emerged (p > .095) indicating that homoscedasticity/homogeneity of variance assumptions were not violated (see Supplemental Table S1).

Behavioral Performance

No significant differences in overall task performance (t_{66} = 1.57, p = .121) or reaction time (t_{66} = −0.79, p = .430) emerged. Notably, both the SA (mean = 91.7%, SD = 0.05%) and no SA (mean = 93.7%, SD = 0.05%) groups demonstrated excellent accuracy.

Raw Average Waveforms

Raw N2 Simple Group Differences. An RM-ANOVA with trial type entered as a within-subjects factor and SA history entered as a between-subjects factor revealed a nonsignificant SA history × trial type interaction ($F_{1,66}$ = 0.60, p = .441, η_p^2 = .009) and a nonsignificant between-subjects effect ($F_{1,66}$ = 0.32, p = .574, η_p^2 = .005). Independent samples *t* tests revealed null group differences in no-go (t_{66} = −0.79, p = .430) or go (t_{66} = −0.26, p = .798) N2 amplitudes (see Figure 1).

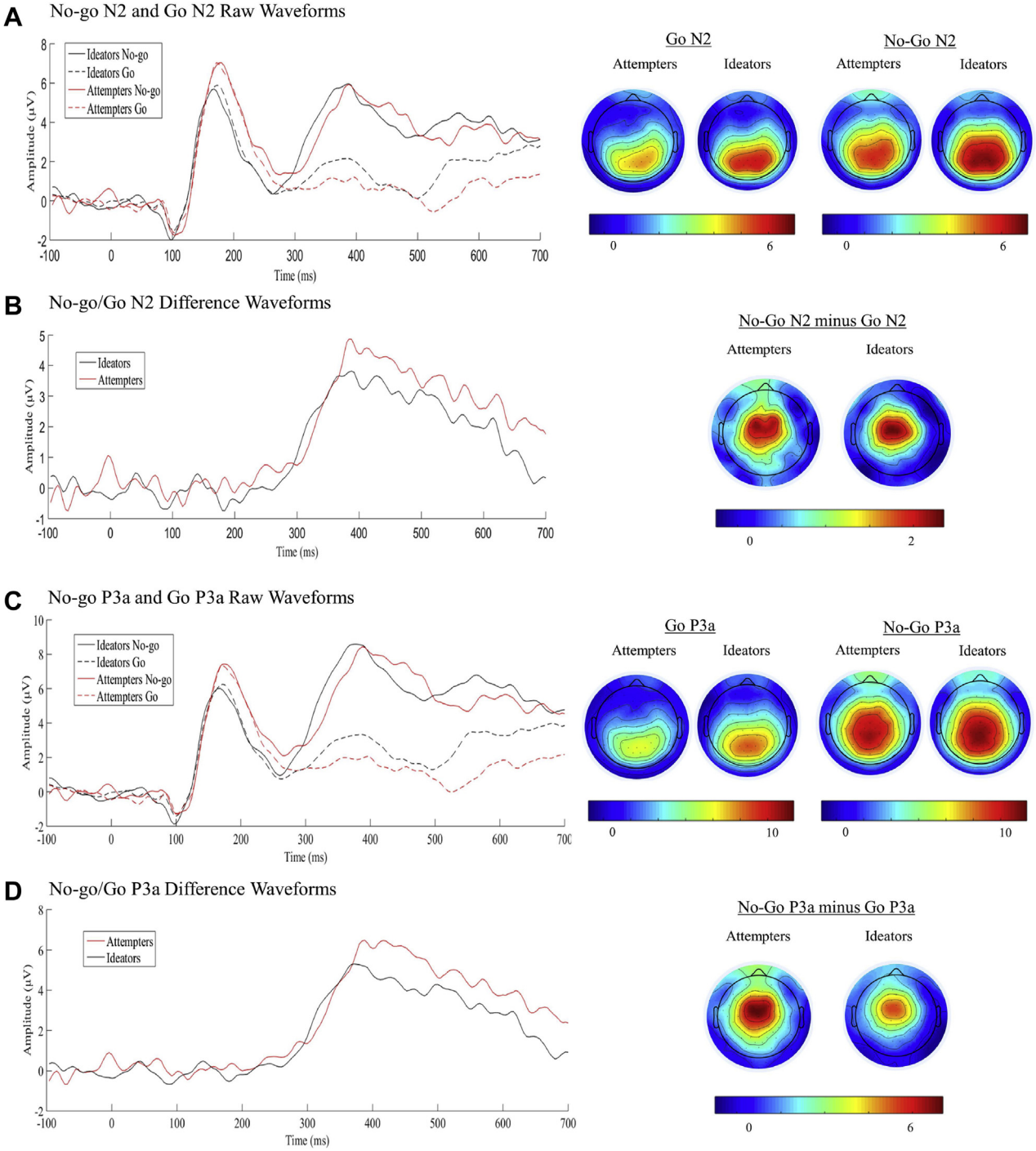

Figure 1. Waveforms and topographical maps depicting the raw grand average N2 among ideators with a history of suicide attempts (i.e., attempters) and no history of suicide attempts (i.e., ideators). **(A)** Attempters appeared to have a more positive no-go N2, though differences were not significant. For illustrative purposes, topographical maps index average activity from 200 to 348 ms. **(B)** No significant group differences in raw N2 amplitude on no-go trials, relative to go trials, emerged. **(C)** No significant group differences in raw P3a amplitude emerged. For illustrative purposes, topographical maps index average activity from 296 to 500 ms. **(D)** No significant group differences in raw P3a amplitude on no-go trials, relative to go trials, emerged. N2 waveforms were extracted from Fz. P3a waveforms were extracted from FCz.

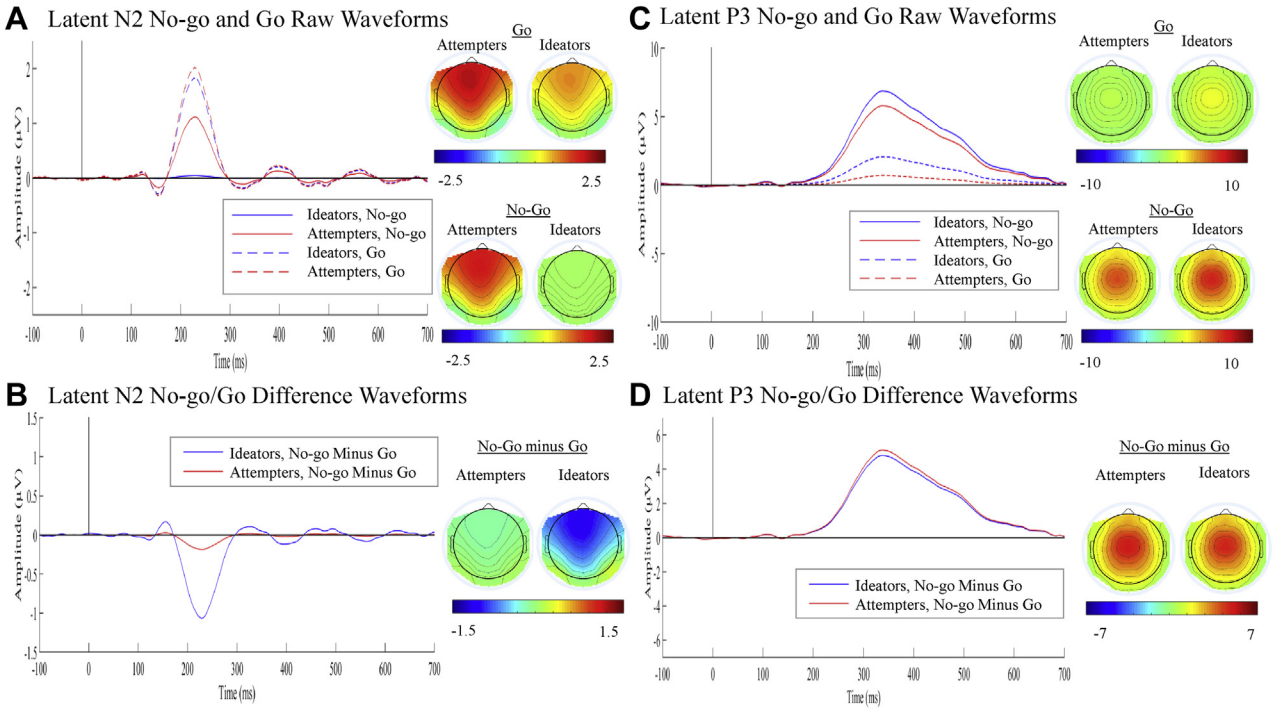

Figure 2. Temporospatial principal components analysis–derived latent N2 and P3a components across ideators with a history of suicide attempts (i.e., attempters) and no history of suicide attempts (i.e., ideators). **(A)** Attempters demonstrated a significantly more positive latent no-go N2 factor compared with ideating nonattempters, and no group differences in go N2 factor amplitude. **(B)** Attempters demonstrated a significantly blunted latent N2 on no-go trials relative to go trials. **(C)** No significant group differences in latent P3a amplitude emerged. **(D)** No significant group differences in latent P3a amplitude on no-go trials, relative to go trials, emerged.

Raw P3a Simple Group Differences. An RM-ANOVA with trial type entered as a within-subjects factor and SA history entered as a between-subjects factor revealed a nonsignificant SA history × trial type interaction ($F_{1,66}$ = 0.65, p = .422, η_p^2 = .010) and a nonsignificant between-subjects effect ($F_{1,66}$ = 0.20, p = .659, η_p^2 = .003). Independent samples t tests revealed null group differences in no-go (t_{66} = 0.12, p = .908) or go (t_{66} = 0.83, p = .407) P3a amplitudes (see Figure 1).

Temporospatial PCA-Derived Latent Waveforms

Latent N2 RM-ANOVA Simple Group Differences. An RM-ANOVA with trial type entered as a within-subjects factor and SA history entered as a between-subjects factor revealed a significant SA history × trial type interaction ($F_{1,66}$ = 4.47, p = .038, η_p^2 = .063) and a significant between-subjects effect ($F_{1,66}$ = 4.17, p = .045, η_p^2 = .059). Independent samples t tests further revealed that individuals with an SA history (mean = 1.87, SD = 2.39), relative to those with no SA history (mean = 0.12, SD = 2.71), exhibited a significantly more positive no-go N2 factor (t_{66} = −2.59, p = .012). Group differences in go N2 amplitude were not significant (t_{66} = −1.26, p = .212; see Figure 2).

Latent N2 RM-ANCOVA Group Differences When Accounting for Psychiatric Comorbidity and Age. An RM-ANCOVA with trial type entered as a within-subjects factor and SA history, depressive disorder, PTSD, SUD, and age entered as between-subjects factors revealed that the within-subject interaction of SA history × trial type ($F_{1,62}$ = 4.23, p = .044, η_p^2 = .064) remained when covariates were included. The between-subjects effect ($F_{1,62}$ = 3.44, p = .068, η_p^2 = .053) was not significant (see Figure 2 and Table 3).

Latent N2 Hierarchical Linear Regressions Examining Group Differences When Accounting for Psychiatric Comorbidity and Age. Next, 3 hierarchical linear regressions were conducted to probe the directionality of these effects. SA history predicted a more positive ΔN2 in both the first step, accounting for psychiatric comorbidity (β = .33, t = 2.82, p = .007), and second step, accounting for age (β = .30, t = 2.46, p = .017), indicating that SA history predicted a more positive N2 on no-go, relative to go, trials even when accounting for relevant covariates. Follow-up hierarchical regressions indicated that SA history predicted a more positive no-go N2 accounting for psychiatric comorbidity in the first step (β = .33, t = 2.90, p = .005) and when adding age in the second step (β = .29, t = 2.44, p = .017). In contrast to the effects observed on no-go (i.e., inhibition) trials, SA history was not significantly related to the N2 amplitude on go trials (i.e., those requiring a motor response) in either step of the hierarchical regression (p > .178) (see Table 4).[3]

[3]Analyses utilizing dimensional measures of depression severity and PTSD symptom severity as covariates are provided in Supplemental Table S2.

Table 3. Two Separate RM-ANCOVAs Assessing Latent N2 Differential Responding on Go/No-Go Trials

	SS	*df*	MS	*F*	*p*	η_p^2
Latent N2: Go/No-Go Within-Subjects Effects						
Trial type	9.83	1	9.83	6.98	.01	.101
Trial type × SA history	5.95	1	5.95	4.23	.04	.064
Trial type × depressive disorder	0.11	1	0.11	0.08	.79	.001
Trial type × PTSD	3.92	1	3.92	2.78	.1	.043
Trial type × SUD	0	1	0	0	.97	<.001
Trial type × age	0.36	1	0.36	0.25	.62	.004
Error	87.33	62	1.41			
Latent N2: Go/No-Go Between-Subjects Effects						
SA history	40.04	1	40	3.44	.07	.053
Depressive disorder	14.78	1	14.8	1.27	.26	.020
PTSD	9.66	1	9.66	0.83	.37	.013
SUD	7.64	1	7.64	0.66	.42	.010
Age	10.69	1	10.7	0.92	.34	.015
Error	720.83	62	11.6			

Depressive disorders include presence/absence of major depressive disorder, persistent depressive disorder, or mood disorder not otherwise specified.

MS, mean squares; PTSD, posttraumatic stress disorder; RM-ANCOVA, repeated measures analysis of covariance; SA, suicide attempt; SS, sum of squares; SUD, substance use disorder.

Latent P3a RM-ANOVA Simple Group Differences. An RM-ANOVA with trial type entered as a within-subjects factor and SA history entered as a between-subjects factor revealed a nonsignificant SA history × trial type interaction ($F_{1,66}$ = 0.58, p = .450, η_p = .009) and a nonsignificant between-subject effect ($F_{1,66}$ = 0.58, p = .449, η_p^2 = .009) of SA history on P3a amplitude. Independent samples t tests showed no group differences in no-go (t_{66} = 0.44, p = .662) or go (t_{66} = 1.08, p = .284) P3a amplitudes (see Figure 2).

DISCUSSION

In partial support of hypotheses, the present study demonstrated that among individuals with current ideation, suicide attempters exhibited a more positive latent ΔN2 amplitude relative to nonattempters during a complex go/no-go task. Follow-up analyses indicated that this effect was driven by a more positive no-go N2 amplitude and that this simple group difference remained when accounting for psychiatric comorbidity and age. Furthermore, these group differences only emerged when the latent N2 component was extracted using temporospatial PCA, perhaps owing to the greater signal-to-noise ratio provided by this technique relative to raw amplitude approaches (47). No group differences emerged for indices of motor inhibition in either the behavioral (accuracy, reaction time) or neurophysiological (ΔP3a amplitude) units of analysis. This pattern of results suggests that suicide attempters differ from nonattempters on a specific impairment in the ability to detect when inhibition is needed (i.e., a more positive ΔN2 and no-go N2), but not necessarily in the allocation of cognitive resources toward motor inhibition (i.e., P3a). These findings dovetail with research linking suicidal behavior with poor dorsal anterior cingulate cortex functional connectivity during conflict detection in other forms of cognitive control (25,26) and violence directed toward others (29,30). Because the dorsal anterior cingulate cortex is believed to be the neural generator of the no-go N2 (27), it is plausible that these findings represent a similar underlying deficit of conflict detection that differentiates ideators with a history of SAs and those with no history of SAs.

There are several nonmutually exclusive pathways through which poor conflict detection could contribute to suicide risk. First, difficulty detecting when to engage inhibitory control could contribute to enacting a suicide plan when confronted with suicidal desire. This interpretation is consistent with the predictions of existing theories of suicide that poor inhibitory control serves as a distal risk factor that amplifies more proximal affective or interpersonal disruptions such as thwarted belongingness and perceived burdensomeness (6), similar

Table 4. Three Separate Hierarchical Linear Regressions Assessing Unique Effects of SA History on Latent N2 Amplitudes

Model	Independent Variables	β	*t*	*p*	R^2
DV: Latent ΔN2					
Step 1	SA history	.33	2.82	.007	.106
	Depressive disorder	.09	0.74	.465	.007
	PTSD	.23	1.90	.062	.048
	SUD	−.03	−0.26	.793	.001
Step 2	SA history	.30	2.46	.017	.084
	Depressive disorder	.07	0.54	.590	.004
	PTSD	.22	1.85	.070	.046
	SUD	−.03	−0.24	.811	.001
	Age	.09	0.73	.471	.007
DV: Latent No-Go N2					
Step 1	SA history	.33	2.90	.005	.109
	Depressive disorder	.17	1.45	.151	.028
	PTSD	.18	1.48	.143	.029
	SUD	−.09	−0.82	.418	.009
Step 2	SA history	.29	2.44	.017	.077
	Depressive disorder	.14	1.14	.250	.017
	PTSD	.17	1.42	.162	.026
	SUD	−.09	−0.78	.436	.008
	Age	.13	1.08	.286	.015
DV: Latent Go N2					
Step 1	SA history	.17	1.36	.178	.028
	Depressive disorder	.15	1.18	.243	.021
	PTSD	.05	0.36	.722	.002
	SUD	−.10	−0.77	.443	.009
Step 2	SA history	.14	1.07	.288	.017
	Depressive disorder	.13	0.97	.336	.014
	PTSD	.04	0.31	.757	.001
	SUD	−.09	−0.75	.458	.008
	Age	.10	0.74	.464	.008

Latent N2 components were extracted using temporospatial principal components analysis. Depressive disorders include presence/absence of major depressive disorder, persistent depressive disorder, or mood disorder not otherwise specified.

ΔN2, residualized difference score of no-go N2 regressed on go N2; DV, dependent variable; PTSD, posttraumatic stress disorder; SA, suicide attempt; SUD, substance use disorder.

to dual-process models in related psychopathologies (60–64). Tangential evidence implicating a blunted N2 with the commission of impulsive acts of aggression toward others (29,30) also supports this view insofar as aggression toward others might reflect an underlying biobehavioral suite related to aggression toward oneself (i.e., an SA) (28). Engagement in interpersonal violence among those with poor conflict detection (29,30) also supports the view that these individuals may engage in more painful and provocative events, thereby reducing fearlessness about death (65,66). Alternatively, it is possible that poor conflict detection may contribute to legal, financial, or interpersonal stressors through an inability to withhold inappropriate behaviors, thereby triggering or escalating a suicidal crisis (67,68). Future research should utilize longitudinal designs to better understand the role of conflict detection in the development and/or escalation of suicidal crises within an ideation-to-action framework. Regardless of the mechanism, individuals with poor conflict detection may benefit from targeted interventions designed to mitigate this deficit. For instance, there is some evidence supporting the use of neurostimulation (69,70) or contextual cues (71) to enhance conflict detection.

There are several limitations to address. Although the expected negative-going N2 appeared in the raw waveforms (see Figure 1), the latent N2 factor derived by the PCA was positive-going (see Figure 2). This has also been found in other studies using complex go/no-go tasks (53,72). All other component properties (trial type, spatial/temporal characteristics) and correlations between PCA factors and raw waveforms suggested that the N2 in the present study reflects a typical N2 in function and supports the conclusion that suicide attempters have poor conflict detection. A benefit of using this complex go/no-go task is that it may better approximate daily life, which necessitates working memory utilization. However, this task could have also contributed to the N2 factor componentry differing from the N2 appearing in simple go/no-go tasks (13–15). Given the utility of PCA to better understand other ERP components (73,74), it is possible that the negativity in raw no-go (relative to go) trials appears because of a lack of positivity, which may represent an action selection process that is revealed only with PCA, which could also suggest a different function for the N2 (24). Also, the duration of the intertrial intervals were not jittered in the current study. This limitation could influence trial-to-trial carryover effects and therefore impact responding. Future research should employ both simple and complex go/no-go tasks with jittered intertrial interval durations to clarify the observed effects. The current study also did not test a distinction between reactive and proactive control (75) among suicide attempters, leaving another avenue for future research.

Regarding the sample, future research should seek to increase sample size and to match groups based on relevant demographics as opposed to accounting for group differences statistically, which may bias results (76). Lastly, the current study utilized a BSS score > 0 as a cutoff for current SI, which captures a heterogeneous presentation of ideation (e.g., active ideation, passive ideation, planning). While there is precedent for the use of such a cutoff (31,32), future research would benefit from expanding on these novel findings with a sample exhibiting more severe SI.

Despite these limitations, the current study provides further support for the role of inhibitory control impairments in the transition from SI to making an SA (11,12,19–21) and expands on prior work by demonstrating that impaired conflict detection, specifically, differentiates ideating attempters from non-attempters. Future research should evaluate whether poor conflict detection predicts future suicide risk and can thus be conceptualized within the ideation-to-action framework.

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