Event-Related Potential Markers of Emotional Provocation in Obsessive-Compulsive Disorder: A Centroparietal LPP Analysis

By

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Thesis

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Chapter 1: Introduction

1.1 Clinical Overview of Obsessive-Compulsive Disorder

Obsessive-compulsive disorder (OCD) is a neuropsychiatric disorder classically defined by the interplay between obsessions, or intrusive, unwanted thoughts or urges, and compulsions, or repetitive mental or physical acts performed to relieve distress or achieve a subjective sense of "completeness".[1] In the clinical context, this feedback loop can become self-reinforcing: compulsions temporarily reduce anxiety, but also strengthen the perceived importance of the obsession and increase reliance on ritualized behaviors over time.[2,3] Among U.S. adults, the lifetime prevalence of OCD is 2.3%,[4] with about half indicating serious impairment[5] due to their OCD as determined by scores on the Sheehan Disability Scale.[5]

1.2 Diagnosis and Current Treatment Approaches

Diagnosis and treatment of OCD require a careful and multifaceted clinical approach. Diagnosis is primarily based on a psychological evaluation that explores an individual's thoughts, emotions, symptoms, and behavior patterns to determine whether obsessions and/or compulsions are present and significantly impair quality of life.[1] In some cases, collateral information from family members may also be incorporated. A physical exam can be performed to rule out alternative medical causes of symptoms or related complications.[1] Diagnostic complexity arises because OCD symptoms can overlap with those of other psychiatric conditions, including anxiety disorders, major depressive disorder, schizophrenia, or obsessive-compulsive personality disorder, and comorbidity is common.[6,7] As a result, accurate diagnosis relies on thorough clinical assessment and ongoing collaboration between patients and clinicians to ensure proper

classification and future treatment planning.[8] Treatment for OCD is aimed at symptom management rather than cure and often requires long-term or intensive intervention depending on symptom severity. The most effective approach typically combines psychotherapy and medication,[9] with cognitive behavioral therapy (CBT), particularly exposure and response prevention (ERP), serving as a cornerstone of care. ERP involves gradual exposure to feared stimuli while preventing compulsive responses,[10] helping individuals reduce anxiety and break the obsessive–compulsive cycle over time; its primary challenges include high attrition rates and difficulty with sustaining patient engagement with the therapy.[11] Pharmacologic treatment most commonly includes antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs) and clomipramine which often require higher doses[12] than those used for major depressive disorder and longer treatment trials to achieve symptom control.[12] This dose–response relationship is supported by meta-analytic evidence demonstrating that higher SSRI doses are associated with greater symptom reduction in OCD,[12] although this benefit is accompanied by increased side-effect burden.

For individuals with treatment-resistant OCD,[13] additional options such as intensive outpatient programs, deep brain stimulation (DBS), or transcranial magnetic stimulation (TMS) may be considered. DBS[14] is a reversible and adjustable neuromodulation therapy in which implanted electrodes modulate dysfunctional cortico-striato-thalamo-cortical circuits to reduce symptoms in patients with severe, treatment-resistant obsessive-compulsive disorder.[13,14,15] While these interventions can be extremely beneficial for some patients,[14,15] with significant improvements in overall OCD symptom severity, including both obsessions and compulsions, as well as depressive symptoms and global functioning,[14,15] there remains a critical need to elucidate the

optimal anatomical targets, stimulation parameters, and neural mechanisms underlying treatment response in order to improve efficacy.

1.3 Symptom Dimensions and Heterogeneity in OCD

OCD encompasses a wide spectrum of symptom presentations, with considerable variability in both the content of obsessions and the behaviors or mental rituals used to manage them. Obsessive thoughts frequently involve concerns related to contamination, personal or interpersonal harm, intrusive sexual or violent imagery, or a strong need for symmetry and precision, though the specific form these thoughts take can differ substantially across individuals.[1,16] Compulsions may include repetitive cleaning, checking, ordering, counting, praying, or internal attempts to neutralize or control unwanted thoughts.[1] While OCD shares surface-level similarities with other psychiatric conditions, it is distinguished by the persistence and salience of its symptoms: obsessive fears tend to extend well beyond immediate triggers and often dominate daily functioning through prolonged rumination, ritual performance, and avoidance.[6] In contrast to disorders such as generalized anxiety disorder or major depressive disorder, OCD-related thoughts are typically experienced as intrusive and irrational, and they are closely linked to compulsive behaviors.[6] It is also important to differentiate OCD from obsessive-compulsive personality disorder (OCPD), in which traits such as perfectionism and rigidity are generally *ego-syntonic* and perceived as appropriate or desirable, rather than distressing or intrusive.[1,8]

Given the breadth of symptom expression, OCD is commonly described using multidimensional frameworks that organize symptoms into broader categories rather than relying on a single diagnostic profile. Empirical work using symptom-based assessments has identified recurring

clusters[16,17,18,19] such as contamination and cleaning, symmetry and ordering, doubt and checking, and taboo or intrusive thoughts, while hoarding has since been recognized as a separate diagnostic entity.[1] Other models focus less on symptom content and more on underlying motivational drives, distinguishing between behaviors primarily aimed at preventing harm and those driven by a chronic sense of incompleteness or discomfort when things feel "not quite right".[18,19]

Despite differences in symptom expression, individuals with OCD often share overlapping cognitive features, including difficulties with executive control, attention, and working memory.[16,20] These cognitive patterns are thought to reflect core processes, such as pathological doubt, altered risk evaluation, and heightened sensitivity to errors or incompleteness, that cut across symptom subtypes and contribute to the chronic and self-reinforcing nature of the disorder.[6]

1.4 Neurobiological Models of OCD

Neuroanatomical differences between individuals with OCD and healthy controls remain most consistently characterized by dysfunction within cortico–striato–thalamo–cortical (CSTC) circuits,[21,22] which are involved in cognitive control, behavioral regulation, and error processing. Previous neuroimaging studies identified elevated metabolic and hemodynamic activity within the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), caudate nucleus, and thalamus in OCD compared with healthy controls,[23] and these abnormalities have been shown to attenuate following successful pharmacologic or behavioral treatment, supporting a direct association with symptom expression.

However, other structural and functional imaging and machine learning studies have expanded this perspective to dimensional analyses of gray matter and white matter microstructure within CSTC loops and to connectivity differences across broader networks, including the default mode, salience, and frontoparietal networks.[24,25,26] These network-level studies confirm that while CSTC dysfunction remains central, OCD is also marked by widespread alterations in how large-scale brain systems interact and coordinate activity, making this a more complex disorder than early region-specific models alone would suggest.

More recent functional connectivity research indicates that key nodes of executive control and salience networks show aberrant coupling in individuals with OCD.[27] For example, studies have reported altered resting-state functional connectivity not only within CSTC pathways but also between the default mode network (DMN), central executive network (CEN), and salience network (SN), suggesting disruptions in how networks responsible for internally generated thought, attentional control, and stimulus salience are integrated in OCD.[27,28,29]

These alterations can manifest as increased synchronization between certain network pairs (e.g., DMN–SN) and decreased dynamic connectivity between striatal subregions and widespread cortical regions, highlighting both hyperconnectivity and context-dependent dysregulation within and between large-scale networks in OCD compared to controls. Such findings align with the view that OCD involves not only local hyperactivity[21] within CSTC loops but also broader network reconfiguration, where interactions among networks that support cognitive flexibility, self-referential processing, and executive functioning are altered in a manner that corresponds to symptom severity and behavioral rigidity.[30]

Additional evidence suggests that these network alterations are not restricted to classic CSTC nodes but include systems such as the cerebellum and regions implicated in salience detection, top-down control, and self-monitoring.[31] Emerging work has shown expansion of the salience network representation into frontostriatal regions in OCD and concomitant contraction of executive networks like the frontoparietal network, a pattern not seen in healthy controls that may reflect imbalance in functional network boundaries.[31] Moreover, structural MRI and diffusion-based studies continue to document gray and white matter alterations within CSTC and adjacent pathways, reinforcing that both microstructural integrity and functional coupling contribute to the neurobiology of OCD.[32]

Taken together, these findings support a model in which OCD arises from distributed dysconnectivity across multiple interacting systems rather than from isolated regional dysfunction alone, extending classic CSTC circuit frameworks toward a more integrated[33] understanding of the disorder's neural basis.

1.5 Rationale for Electroencephalography in OCD Research

Although scalp EEG cannot directly resolve activity from deep brain structures implicated in OCD, it is well suited to capturing oscillatory dynamics and event-related activity within cortical regions that play a central role in obsessive-compulsive symptom processes, including the orbitofrontal cortex and anterior cingulate cortex.[33] Importantly, these regions are consistently implicated in error monitoring, threat valuation, and cognitive control, processes that are disrupted in OCD and are increasingly understood to be expressed through dynamic changes in cortical network activity.[34] By comparing EEG-derived measures between individuals with OCD

and healthy controls, it is therefore possible to identify cortical correlates of network engagement that could reflect downstream effects of circuit dysfunction.[35]

Analyzing EEG data in conjunction with structured behavioral paradigms further strengthens its utility for biomarker discovery.[36,37,38] Task-based EEG allows neural activity to be examined during controlled transitions between neutral and symptom-relevant states, enabling within-subject contrasts that reduce inter-individual variability and enhance sensitivity to disorder-specific signals.

Event-related potentials (ERPs) derived from EEG recordings allow researchers to examine time-locked neural responses to emotionally salient stimuli. One ERP component of particular relevance is the Late Positive Potential (LPP). The LPP is a sustained positive deflection in the ERP waveform that typically emerges approximately 300–1000 ms following stimulus onset and is most prominent over centroparietal scalp sites, including electrodes[39,40] CP1, CPz, and CP2. The amplitude of the LPP has been shown to increase in response to emotionally salient or motivationally relevant stimuli, reflecting processes related to sustained emotional engagement, attentional allocation, and emotion regulation.[40]

Previous research suggests that the LPP may index cortical processes related to CSTC dysfunction,[39,40,41,42] which are strongly implicated in OCD pathophysiology. Dysregulation within these circuits has been associated with persistent threat appraisal and difficulty disengaging from distressing thoughts or stimuli, which are core features of obsessive–compulsive symptomatology. Consequently, the LPP represents a potential neurophysiological marker of symptom-related emotional processing.

1.6 Task-Based EEG and State-Dependent Biomarker Discovery

In the broader field of psychiatry, biomarkers[35,36] are objective biological measures that can improve diagnosis, predict treatment response, and guide individualized therapeutic strategies, addressing a challenge in mental health where clinical signs and symptoms remain the primary basis for diagnosis rather than underlying biology.[36] For OCD specifically, research over the past decade has explored a wide range of potential biomarkers, from neurocognitive variables and neuroimaging measures to electrophysiological signatures, but none have yet demonstrated sufficient sensitivity and specificity to serve as standalone diagnostic tools; rather, the most promising work suggests that biomarkers may be more useful for predicting clinical outcomes or subtyping within OCD than for diagnosis alone.[37]

Recent reviews integrating multimodal data indicate that distinct OCD symptom subtypes may be underpinned by divergent neurobiological pathways, implicating dysregulation in glutamatergic, serotonergic, and dopaminergic systems alongside differential network engagement, and highlighting the need for subtype-aware studies to validate these signals.[38] In the electrophysiological domain, emerging research has begun to identify candidate neural signatures, including both intracranial local field potentials linked to core compulsive states and interpretable EEG features that may distinguish OCD from healthy controls within machine learning frameworks,[39] underscoring the potential for EEG-based biomarkers to contribute to precision interventions such as adaptive neuromodulation.

1.7 Thesis Objectives and Overview

The primary objective of this thesis is to investigate electrophysiological differences between individuals with OCD and healthy control participants using scalp electroencephalography during neutral and symptom-provocation tasks. By integrating EEG recordings with structured behavioral paradigms designed to elicit disorder-relevant neural states, this work aims to identify candidate cortical biomarkers associated with obsessive-compulsive symptom expression and cognitive control processes, with potential relevance for future biomarker-guided neuromodulation and precision psychiatry approaches.

The present study investigates neural responses during a computer-based symptom provocation task (PROVOC) designed to elicit manageable levels of symptom-relevant distress. During this task, participants view images that are either neutral or tailored to evoke OCD-relevant concerns while EEG is recorded. By comparing neural responses to provoking versus neutral stimuli, it is possible to assess whether symptom-related stimuli produce enhanced emotional processing as indexed by the LPP.

This study focuses specifically on LPP amplitude measured at centroparietal electrodes CP1, CPz, and CP2, where the component is typically strongest and least contaminated by earlier visual ERP components. Mean ERP amplitude within the 300–1000 ms post-stimulus interval is compared between provoking and neutral image conditions.

The primary hypotheses were as follows:

H1: Provoking images will elicit a significantly larger centroparietal LPP than neutral images across all participants.

H2: The magnitude of the LPP difference (provoking minus neutral) will differ between individuals with OCD and healthy control participants.

By examining the LPP during individualized symptom provocation, this study aims to evaluate whether EEG-derived neural responses can serve as candidate biomarkers of emotional engagement in OCD and contribute to the development of objective physiological measures of symptom reactivity.

Chapter 2: Methods

2.1 Electrophysiological Monitoring Setup

Participants were recruited through multiple channels including flyers distributed to the general population, EPIC/MyChart portal messages sent to Butler Hospital patients within relevant clinical groups, and referrals from other ongoing research projects at Butler Hospital. EPIC/MyChart recruitment messages were targeted to individuals with documented OCD diagnoses for the OCD cohort. All participants were screened by trained clinicians to confirm eligibility and cohort placement prior to participation in the in-clinic phase of the study.

Participants were adults aged 18–65 years, assigned to either an OCD group or a healthy control group. Individuals in the OCD group met DSM-5 diagnostic criteria for obsessive–compulsive disorder and demonstrated clinically significant symptom severity defined as a minimum score of 16 on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS).[43] Healthy control participants had no current or lifetime diagnosis of OCD or other major DSM-5 psychiatric disorders.

Exclusion criteria for all participants included:

- lifetime history of psychotic disorders
- current symptoms of psychosis or mania
- known intracranial pathology
- epilepsy or seizure disorders
- traumatic brain injury
- implanted medical devices incompatible with EEG
- substance use disorder within the previous six months (excluding nicotine)
- pregnancy
- elevated suicide risk

All participants provided written informed consent in accordance with Institutional Review Board approval and HIPAA regulations.

The study consisted of three primary assessment visits. Visit 1 involved informed consent procedures and comprehensive baseline clinical assessments and was conducted either in person or via HIPAA-compliant video conferencing. Visit 2 consisted of a brief virtual follow-up assessment to reassess symptom severity and affective state prior to experimental testing. Visit 3 was conducted in person at Butler Hospital and included physiological setup and administration of behavioral and provocation tasks while EEG and autonomic data were collected.

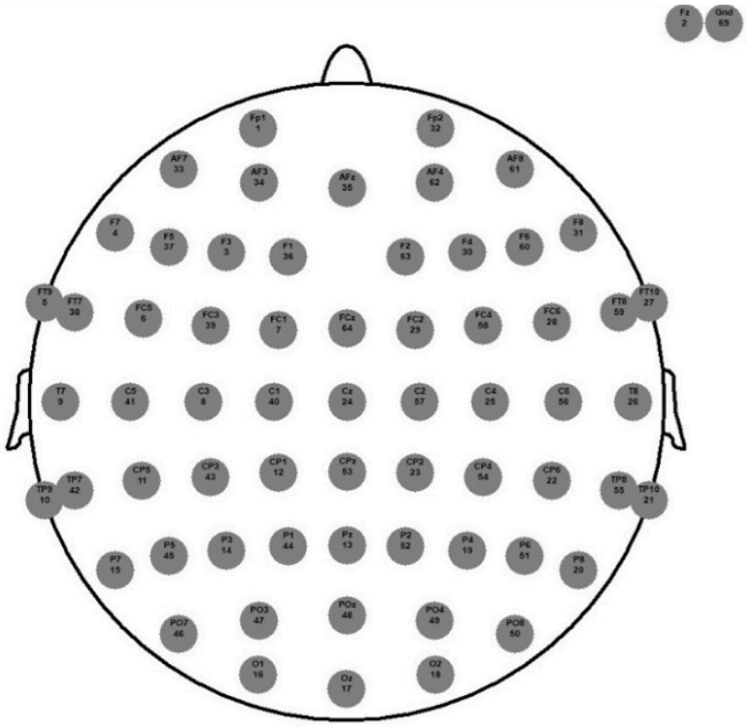

Figure 1. BrainVision 64-channel EEG.[44,45]

Figure 2. Visit 3 Participant Setup During Conveyor Belt Task

During the in-clinic visit, participants were fitted with a 64-channel BrainVision actiCAP EEG system connected to a BrainVision actiCHamp amplifier.[44,45] Additional physiological monitoring included galvanic skin response (GSR) and electrocardiogram (ECG) sensors to capture autonomic activity during task performance. Virtual tasks (Resting State, MSIT, Beads, and Computer PROVOC) were performed on a Windows Surface Pro tablet. Participants began with a resting state task to establish baseline EEG, GSR, and ECG recordings. Participants then completed the MSIT task, Beads task, Computer PROVOC task, an additional resting state task, and finally the Conveyor Belt PROVOC task, concluding the in-clinic phase of the study.

Following preprocessing and data quality checks, EEG datasets from thirteen participants were included in the final analysis. The final sample consisted of nine individuals with OCD and four healthy control participants.

2.2 Experimental Tasks

Resting-State Recording[46]

Participants completed resting-state recordings at the beginning of Visit 3 and before the conveyor belt task. During these periods, participants followed standardized instructions involving visual fixation, eye movements, blinking, and eyes-open/eyes-closed conditions while EEG, ECG, and GSR were recorded. These recordings served as baseline reference measurements for subsequent task-evoked analyses.

Beads Task[47]

The Beads Task[47] was administered to assess decision-making under uncertainty and information-gathering behavior. Participants were required to infer the majority category of stimuli drawn from probabilistic distributions while balancing reward value and information quantity. Behavioral responses and reaction times were recorded concurrently with EEG to examine neural correlates of uncertainty, evidence accumulation, and decision commitment.

Multi-Source Interference Task (MSIT)[48]

The Multi-Source Interference Task (MSIT)[48] was used to probe cognitive control and conflict processing. Participants identified target stimuli under congruent and incongruent conditions designed to elicit response conflict and engage fronto-cingulate circuitry.

Neural and autonomic data were recorded throughout task performance in order to characterize electrophysiological and affective responses associated with conflict monitoring.

Obsessive-Compulsive Symptom Provocation[49]

OCD symptoms were elicited using individualized provocation paradigms[49] designed to induce manageable levels of disorder-relevant distress. Two complementary PROVOC tasks were employed: a conveyor-based *in vivo* exposure task and a computer-based image provocation task.

During the conveyor task, participant-specific triggering objects were gradually presented while 0–10 Subjective Units of Distress Scale (SUDS)[50] ratings were collected at multiple exposure steps. Before the task, the research team selected three objects per participant, one non-distressing and two tailored to trigger OCD-related distress, using the participant's individual answers from the prior visits' assessments to make these decisions. Objects were

chosen to evoke general distress for healthy controls. For example, a control participant had a ball as their neutral object, and a bloody tissue and dirty sponge as their provoking objects.

Figure 3. Conveyor Task Objects for Control Participants. Objects shown: (left, provoking) bloody tissue in Ziploc bag, (middle, provoking) moldy kitchen sponge with maggot, (right, neutral) toy ball with smile. *The same neutral object is used as the "control object" across all participants, both OCD and controls.

Figure 4. Sample Conveyor Task Objects for Provoking Participants. Provoking objects shown: (left) several plastic bugs, (middle) filled pill bottle, (right) sample of fake urine

The computer-based PROVOC task consisted of two blocks of 60 trials, separated by a guided breathing period. Each trial began with the presentation of a white fixation cross for approximately two seconds (with jitter), followed by a green fixation cross displayed for 0.2 seconds to indicate trial onset. A tailored image, selected to be either neutral or symptom-provoking for the participant, was then presented for 3.5 seconds.

Stimuli consisted of 10 neutral and 10 provoking images, individualized for each participant based on a clinical interview conducted during the initial assessment visit. Each image was repeated three times per block, resulting in a total of 60 trials per block. Following the image presentation, participants rated their level of distress using the SUDS[50] scale via a computer interface.

All participants completed the task under the supervision of at least one clinically trained research staff member to ensure safety and provide support if distress became intolerable.

2.3 EEG Data Preprocessing

EEG data were preprocessed by a member of the research team using MATLAB (MathWorks, Natick, MA) and the EEGLAB toolbox. Preprocessed datasets were stored in EEGLAB (.set) format within the project's derivative data directory.

Continuous EEG data were downsampled to 250 Hz and cleaned using ZapLine-plus to remove line noise artifacts. Data were then bandpass filtered between 1 and 30 Hz. Channels

corresponding to non-EEG signals were removed, and bad channels were identified and interpolated using spherical interpolation. Data were re-referenced to the common average reference. Independent component analysis (ICA) was performed using the extended infomax algorithm, and artifactual components were identified using ICLabel and removed based on probability thresholds.

Continuous EEG recordings were segmented into stimulus-locked epochs from −1 to 3 seconds relative to stimulus onset, defined by event markers associated with image presentation during the computer-based provocation task. Baseline correction was applied using the −1000 to 0 ms pre-stimulus interval.

Each epoch was labeled according to stimulus type using the task variable *pType*, where *pType* = 1 indicated provoking stimuli and *pType* = 0 indicated neutral stimuli. Only epochs corresponding to the computer-based provocation task were included in the present analysis.

Channels of interest were identified using electrode labels from the standard 10–10 electrode system.

2.4 ERP Quantification

Event-related potential analyses focused on the Late Positive Potential, a sustained positive ERP component associated with emotional engagement.

The LPP was measured at centroparietal electrodes CP1, CPz, and CP2, where the component is typically strongest[40].

For each epoch, mean voltage amplitude was calculated within a predefined 300–1000 ms post-stimulus window, corresponding to the canonical LPP time range[40].

Epoch-level mean amplitudes were averaged across trials within each stimulus condition to produce subject-level estimates for:

- provoking trials
- neutral trials

A condition difference score was computed for each participant as:

$$\textbf{LPP difference} = \textbf{LPP}_{\textbf{provoking}} - \textbf{LPP}_{\textbf{neutral}}$$

This difference score served as the primary dependent variable used in statistical analyses.

Grand-average ERP waveforms were also generated separately for provoking and neutral conditions at electrodes CP1, CPz, and CP2. In addition, a centroparietal cluster waveform was computed by averaging across the three electrodes to visualize the overall LPP response.

2.5 Statistical Analysis

Statistical analyses were performed using MATLAB.

One-sample *t*-tests were conducted to determine whether the provoking-minus-neutral difference score differed significantly from zero across all participants, indicating a reliable LPP response to symptom-provoking stimuli.

Independent-samples t-tests were conducted to compare LPP difference scores between the OCD and healthy control groups. These tests were performed assuming equal variances. However, given the unequal sample sizes (n = 9 OCD, n = 4 controls), this assumption may be violated. Welch's t-test, which does not assume equal variances, may provide a more robust alternative and should be considered in future analyses.

Follow-up one-sample t-tests were conducted separately within each group on the provoking-minus-neutral difference scores to determine whether LPP amplitudes differed significantly between conditions.

Grand-average ERP waveforms were generated to visualize condition differences across the 300–1000 ms LPP interval.

Chapter 3: EEG Analysis

Event-related potential analyses focused on the LPP at centroparietal electrodes CPz, CP1, and CP2 during the computer-based provocation task. Mean amplitudes were extracted from the 300–1000 ms post-stimulus interval and averaged separately for provoking and neutral trials for each participant. Statistical analyses were conducted on a final sample of thirteen participants, consisting of nine individuals with OCD and four healthy control participants.

Across the full sample, provoking images elicited significantly greater mean amplitudes than neutral images at all electrodes of interest. One-sample *t*-tests comparing the provoking-minus-neutral difference score against zero revealed significant LPP effects at each electrode. At CPz, provoking stimuli produced significantly greater amplitudes than neutral stimuli, $t(12) = 3.191$, $p = .0078$. A similar effect was observed at CP1, $t(12) = 3.473$, $p = .0046$,

and at CP2, $t(12) = 3.280$, $p = .0066$. These results indicate that, across participants, symptom-provoking stimuli elicited a robust enhancement of centroparietal ERP amplitude within the LPP time range.

To visualize these effects, grand-average ERP waveforms were first generated across all participants. Figure 5 illustrates the ERP waveforms recorded at CPz. Across the epoch, provoking trials exhibited a more pronounced positive-going waveform than neutral trials, with the greatest divergence between conditions occurring approximately between 400 and 800 ms post-stimulus, consistent with the canonical LPP interval.

To further examine whether this pattern was consistent across diagnostic groups, grand-average ERP waveforms were then generated separately for individuals with OCD and healthy control participants (Figures 9–14). In both groups, provoking trials elicited greater positive amplitudes than neutral trials within the LPP window, consistent with the within-group statistical effects observed at centroparietal electrodes.

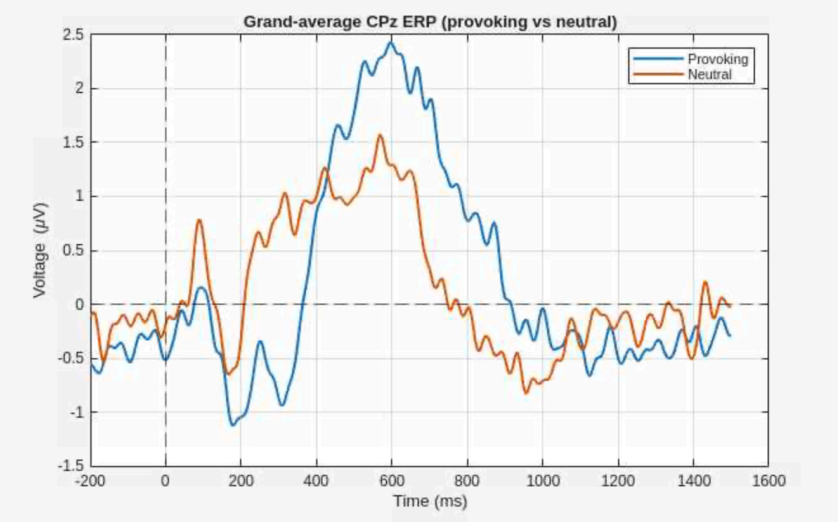

Figure 5. Grand-average ERP waveforms at CPz for provoking and neutral trials during the computer-based provocation task. Provoking trials elicited a larger sustained positive deflection than neutral trials, particularly over the 300–1000 ms interval corresponding to the LPP.

A similar pattern was observed at CP1, where provoking stimuli produced a sustained increase in positivity relative to neutral stimuli across the late post-stimulus interval (Figure 6). The divergence between conditions was again most prominent within the 300–1000 ms window, corresponding to the LPP.

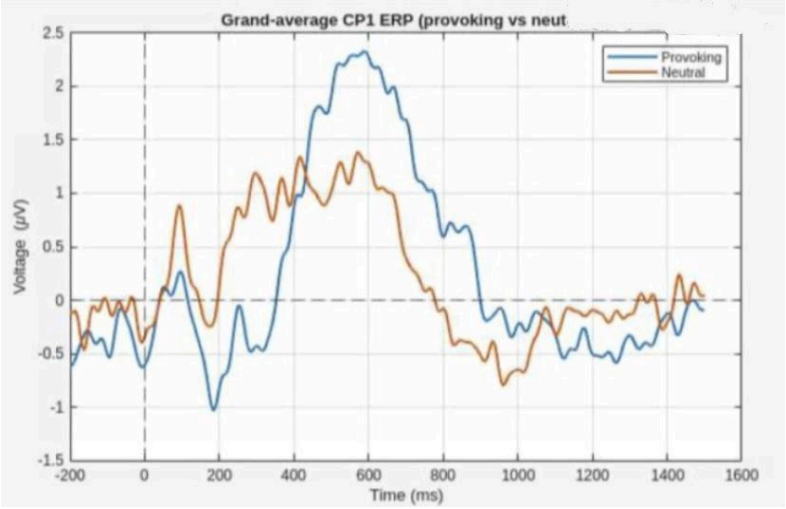

Figure 6. Grand-average ERP waveforms at CP1 for provoking and neutral trials during the computer-based provocation task. Provoking stimuli produced greater positivity than neutral stimuli within the centroparietal LPP time range.

At CP2, ERP waveforms also demonstrated enhanced positivity for provoking relative to neutral stimuli during the late post-stimulus interval (Figure 7). The overall morphology of the waveform was consistent with the pattern observed at the other centroparietal electrodes.

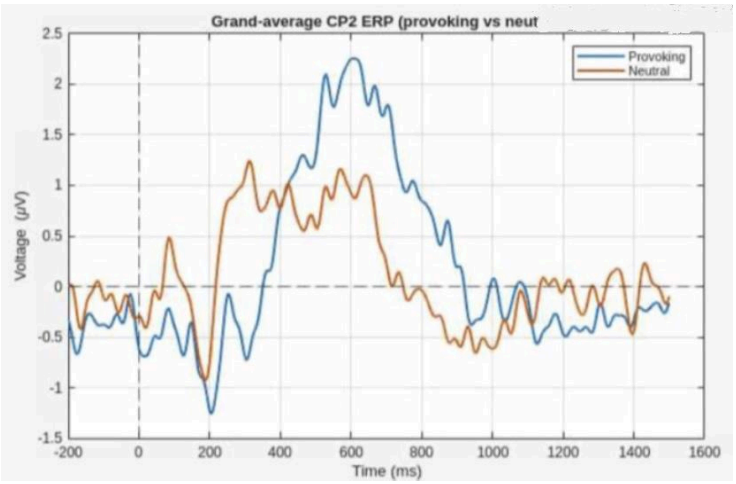

Figure 7. Grand-average ERP waveforms at CP2 for provoking and neutral trials during the computer-based provocation task. The provoking condition showed enhanced positive amplitude relative to the neutral condition across the late post-stimulus interval.

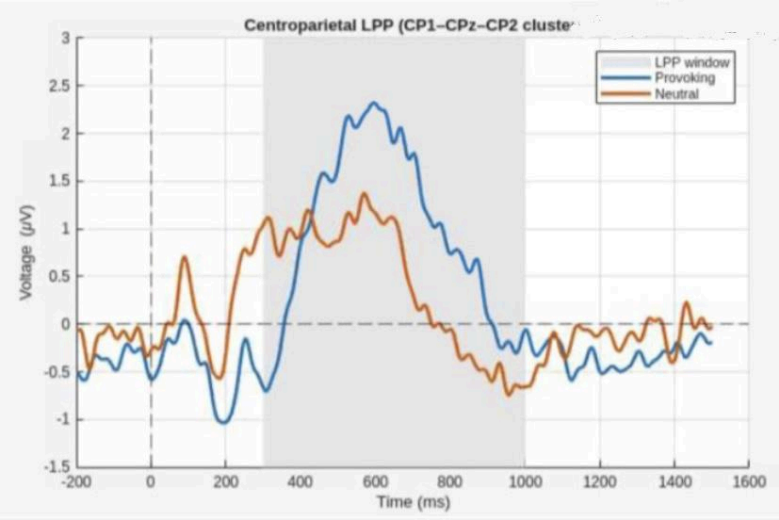

Figure 8. Grand-average centroparietal cluster waveform averaged across CP1, CPz, and CP2. The shaded region indicates the LPP analysis window from 300 to 1000 ms. Provoking trials showed greater mean positivity than neutral trials throughout much of this interval.

To further characterize within-group effects, grand-average ERP waveforms were plotted separately for HC and OCD participants at centroparietal electrodes (CPz, CP1, CP2). For each group, waveforms reflect mean responses to provoking and neutral trials, allowing visualization of the LPP across conditions.

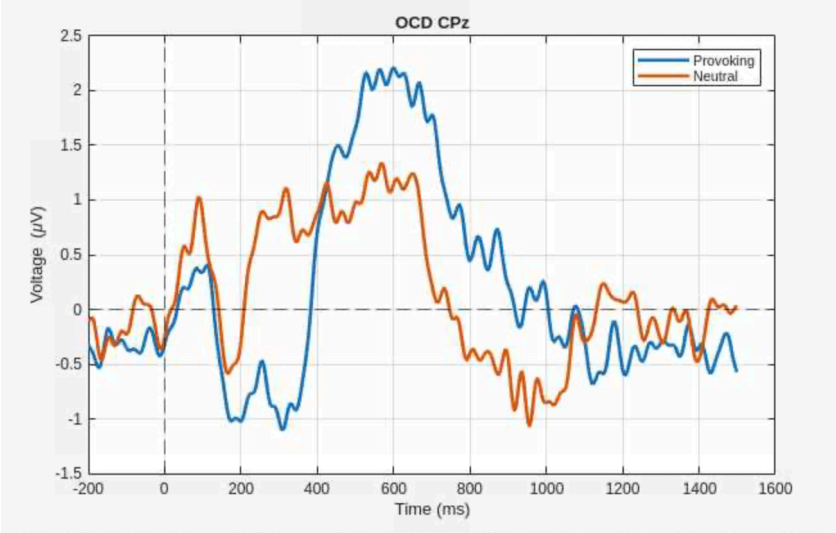

Figure 9. Grand-average ERP waveforms at CPz for individuals with OCD during the computer-based provocation task. Blue represents provoking stimuli and orange represents neutral stimuli.

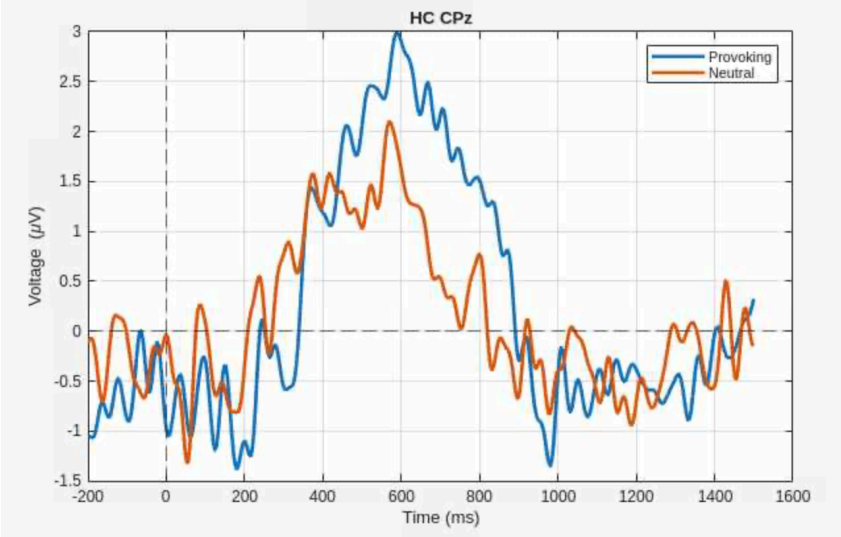

Figure 10. Grand-average ERP waveforms at CPz for healthy controls during the computer-based provocation task. Blue represents provoking stimuli and orange represents neutral stimuli.

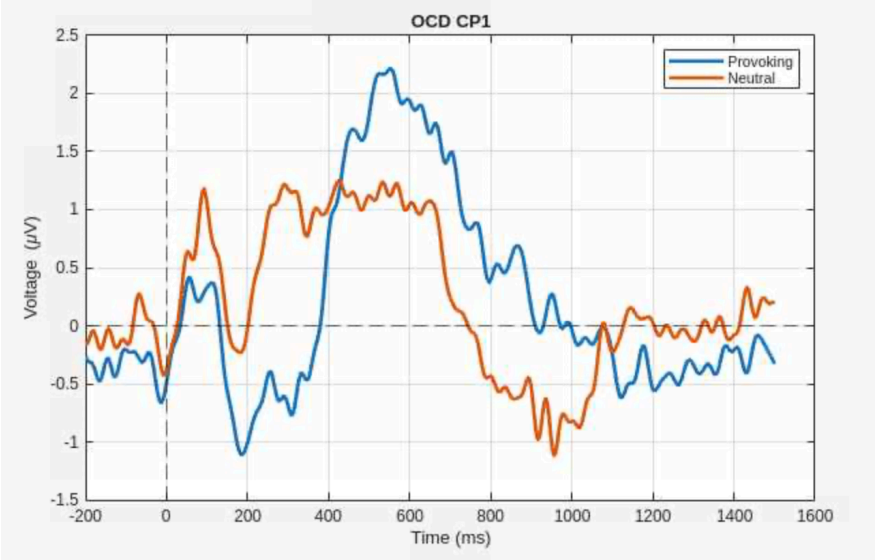

Figure 11. Grand-average ERP waveforms at CP1 for participants with OCD during the computer-based provocation task. Blue represents provoking stimuli and orange represents neutral stimuli.

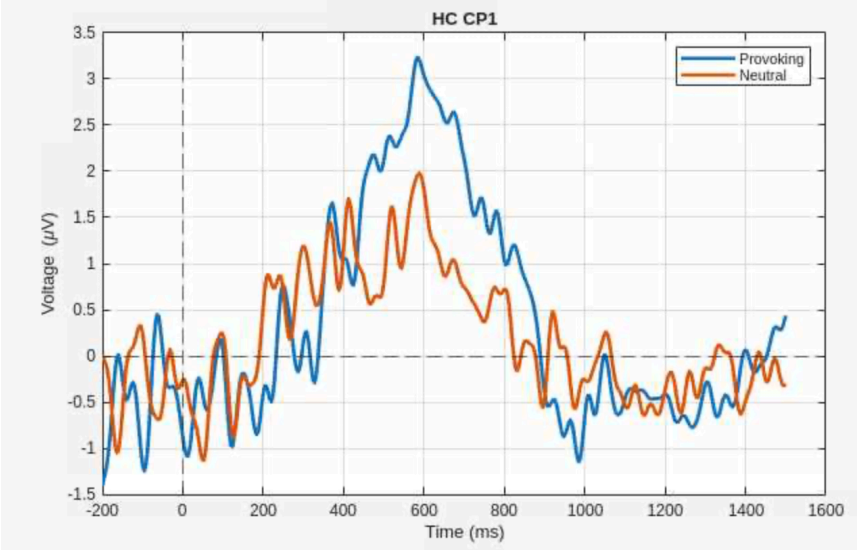

Figure 12. Grand-average ERP waveforms at CP1 for healthy controls during the computer-based provocation task. Blue represents provoking stimuli and orange represents neutral stimuli.

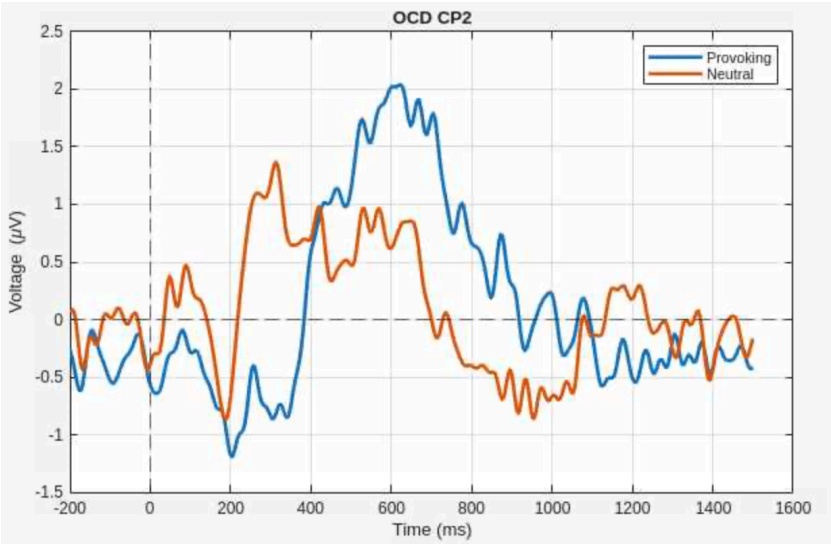

Figure 13. Grand-average ERP waveforms at CP2 for participants with OCD during the computer-based provocation task. Blue represents provoking stimuli and orange represents neutral stimuli.

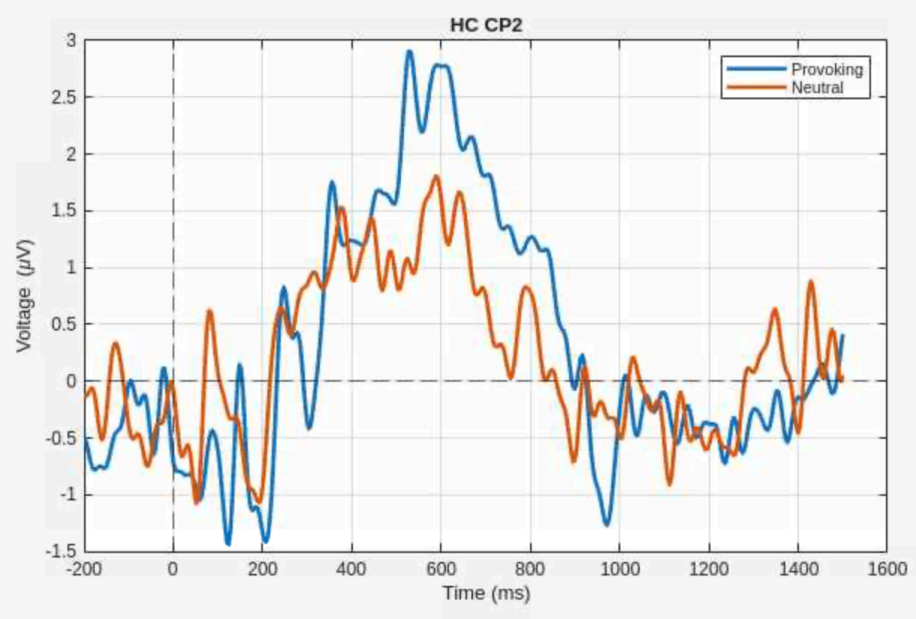

Figure 14. Grand-average ERP waveforms at CP2 for healthy controls during the computer-based provocation task. Blue represents provoking stimuli and orange represents neutral stimuli.

Although provoking stimuli elicited a significant LPP response across the full sample, the magnitude of the LPP difference score did not differ significantly between the OCD group and the healthy control group.

Independent-samples *t*-tests comparing the two groups revealed no significant differences at any electrode. At CPz, the group comparison yielded $t(11) = 0.088$, $p = .9316$. Similarly, the comparison at CP1 was non-significant, $t(11) = -0.119$, $p = .9073$, as was the comparison at CP2, $t(11) = -0.139$, $p = .8923$. These findings indicate that the magnitude of the LPP response to provoking stimuli did not significantly differ between individuals with OCD and healthy control participants in this sample.

Follow-up within-group analyses were conducted to examine whether provoking stimuli elicited greater LPP amplitudes than neutral stimuli within each diagnostic group separately.

Within the healthy control group, one-sample *t*-tests indicated significant provoking-versus-neutral amplitude differences at CPz, $t(3) = 3.845$, $p = .031$, and CP1, $t(3) = 3.440$, $p = .041$. At CP2, the provoking-versus-neutral difference did not reach statistical significance, $t(3) = 2.386$, $p = .097$.

Within the OCD group, provoking stimuli also elicited increased LPP amplitudes relative to neutral stimuli. This difference reached statistical significance at CP1, $t(8) = 2.412$, $p = .042$, and

CP2, $t(8) = 2.457$, $p = .039$. At CPz, the effect approached significance but did not meet the conventional threshold, $t(8) = 2.191$, $p = .060$.

Visual inspection of the grand-average ERP waveforms demonstrated a sustained positive-going deflection for provoking relative to neutral trials across centroparietal electrodes, with the clearest condition separation occurring within the 300–1000 ms interval corresponding to the LPP. Averaging activity across electrodes CP1, CPz, and CP2 yielded a similar waveform pattern, further supporting the presence of a centroparietal LPP enhancement during symptom provocation in both healthy control and OCD groups.

Chapter 4: Discussion, Conclusions, and Future Directions

4.1 Summary of Findings

The primary objective of this study was to examine electrophysiological responses during symptom provocation in individuals with OCD using scalp EEG. Specifically, the present work investigated whether the LPP would differ between neutral and symptom-provoking stimuli during the computer-based PROVOC task.

Across all participants, provoking images elicited significantly larger mean amplitudes than neutral images within the 300–1000 ms post-stimulus interval at centroparietal electrodes CPz, CP1, and CP2. This pattern is consistent with the well-established association between the LPP and emotional or motivational salience.[40,41,42] The enhanced positivity observed for provoking stimuli suggests that the PROVOC task successfully engaged neural processes related to sustained attention and affective processing.

Grand-average ERP waveforms further supported these findings. Across centroparietal electrodes, provoking trials produced a clear positive deflection relative to neutral trials within the canonical LPP time window. Averaging activity across CP1, CPz, and CP2 yielded a similar waveform pattern, indicating that the effect was spatially consistent across the centroparietal scalp region where the LPP is typically maximal.

However, despite the presence of a robust within-subject LPP effect, no significant differences were observed between the OCD and healthy control groups. Independent-samples tests comparing the magnitude of the LPP difference score (provoking minus neutral amplitude) revealed no reliable group differences at any electrode of interest.

Within-group analyses showed that both groups exhibited increased LPP amplitudes during provoking trials relative to neutral trials. Healthy controls demonstrated significant effects at CPz and CP1, while the OCD group showed significant effects at CP1 and CP2, with non-significant differences in the same direction at other electrodes. Taken together, these results suggest that the computer-based PROVOC paradigm successfully elicited emotionally salient neural responses across participants but did not reveal clear electrophysiological differences between groups in the present sample.

4.2 Interpretation of Findings

The present results provide evidence that symptom-provoking stimuli reliably engage late centroparietal neural processes associated with emotional salience and sustained attention. This finding is consistent with previous work linking the LPP to emotionally meaningful stimuli, including threat-related or motivationally relevant cues.[40]

Within the context of OCD research, these results are notable because they demonstrate that the PROVOC task is capable of eliciting measurable neural responses associated with symptom-related emotional processing. In this respect, the findings support the broader goal of the PROVOC project: creating behavioral paradigms that evoke distinct neural states associated with obsessive–compulsive symptoms. Earlier internal analyses conducted within the PROVOC project emphasized that the primary aim of early investigations was task validation rather than definitive biomarker discovery, and the present results align with that objective.

The observation of consistent LPP differences between provoking and neutral conditions suggests that the task structure is effective at eliciting measurable neural responses to symptom-relevant stimuli. However, interpretation of group-level findings should be made with caution given the small sample size (N = 13; 9 OCD, 4 healthy controls), which substantially limits statistical power to detect between-group differences.

In this context, the absence of significant differences between OCD and healthy control groups may reflect insufficient power rather than a true lack of disorder-specific effects. While the LPP response observed here may reflect general emotional engagement, the current findings cannot definitively rule out the presence of OCD-specific neural differences.

Accordingly, it remains possible that electrophysiological distinctions between groups would emerge in a larger, adequately powered sample or through complementary neural measures beyond the LPP.

4.3 Limitations

Several limitations should be considered when interpreting these findings.

The most important limitation of the present study is the relatively small sample size. The final dataset included thirteen participants (nine OCD, four healthy controls). While this sample size is sufficient to detect strong within-subject ERP effects, it provides limited statistical power for detecting between-group differences. In particular, the small number of healthy control participants restricts the reliability of group comparisons and increases the likelihood that subtle effects may remain undetected.

In addition, group comparisons were conducted using standard independent-samples t-tests assuming equal variances. Given the unequal group sizes and small sample, this assumption may not hold, and the use of Welch's t-test would provide a more robust estimate. Although the observed group differences were far from statistical significance, future studies should confirm these findings using variance-robust statistical approaches.

Additionally, the individualized nature of symptom-provoking stimuli introduces variability in the emotional intensity of stimuli across participants. Although personalization improves the likelihood that stimuli will meaningfully engage OCD-related concerns, it also introduces heterogeneity that may reduce statistical sensitivity when comparing across individuals.

Finally, the present analysis focused specifically on ERP amplitude within a predefined time window. Additional analyses examining time–frequency dynamics, network connectivity, or other ERP components may reveal additional differences between groups that were not captured in the current analysis.

4.4 Future Directions

Future research should aim to expand the sample size and further refine electrophysiological analyses of symptom provocation paradigms. Increasing the number of participants, particularly healthy control participants, would improve statistical power and enable more reliable detection of potential group differences.

Future work could also explore individual differences in symptom subtype and distress responses. OCD is a heterogeneous disorder with multiple symptom dimensions. Neural responses to symptom-provoking stimuli may vary across these dimensions, suggesting that biomarker discovery efforts may benefit from subtype-specific analyses.

Future work should also incorporate analyses of subjective distress during the provocation task, as indexed by Subjective Units of Distress Scale (SUDS) ratings. Examining SUDS responses alongside electrophysiological measures may help clarify the relationship between self-reported emotional intensity and neural activity during symptom provocation. This approach could provide important validation for the task paradigm by confirming that provoking stimuli elicit meaningful subjective distress, while also allowing for the investigation of individual variability in distress sensitivity. Additionally, integrating behavioral and neural indices may strengthen efforts to identify clinically relevant biomarkers, particularly if specific patterns of neural activity correspond with heightened subjective distress. Such multimodal analysis may ultimately enhance the interpretability and translational relevance of task-based findings within OCD research.

Finally, continued development of task-based neural markers may contribute to emerging efforts in neuromodulation. One long-term goal of the PROVOC project is to identify neural signatures that could inform adaptive neuromodulation strategies, such as closed-loop deep brain

stimulation systems designed to detect and respond to pathological neural states in real time. As additional participants are recruited and analyses are expanded, the PROVOC paradigm may help clarify how electrophysiological signals reflect symptom engagement and how these signals could potentially guide future therapeutic interventions.

4.5 Conclusion

In summary, the present study demonstrates that the computer-based PROVOC task successfully elicits enhanced centroparietal LPP responses to symptom-provoking stimuli. This finding indicates that the task reliably engages neural processes associated with emotional salience and sustained attention.

Although no significant differences were observed between OCD and healthy control groups in the present sample, the results provide further support for the feasibility of using task-based EEG paradigms to probe neural responses to obsessive–compulsive symptom provocation. With continued data collection and expanded analyses, this line of research may contribute to future biomarker work capable of improving our understanding of OCD neurobiology and informing future neuromodulation-based treatments.

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