N2 as a Neural Marker of Cognitive Conflict: An ERP Study Using the Simon Task

Fernanda González
maria.gonzalez14@estudiant.upf.edu

March 4, 2025

Contents

1 Introduction

1.1 Background

1.1.1 Cognitive control and conflict monitoring

Cognitive control is the ability to regulate thoughts and actions in line with internal goals rather than automatic responses. **Cognitive conflict** arises when there is coactivation of mutually incompatible responses, requiring the brain to resolve the conflict in favor of goal-directed behavior. To resolve the conflict, automatic responses must be suppressed and overridden.

Botvinick proposed the **Conflict Monitoring Theory** [2]. According to this model, the brain continuously monitors for conflict and adjusts cognitive control accordingly. When conflict is detected, control mechanisms are upregulated to prevent subsequent errors.

1.1.2 The neural basis of conflict resolution

Activity in the **anterior cingulate cortex (ACC)** has been found to increase in response to conflict, suggesting its role as a conflict **detector** identifying situations where multiple competing responses are active [1]. See Figure 1.

Once conflict is detected by the ACC, the **prefrontal cortex (PFC)** engages top-down control mechanisms to **suppress** automatic but incorrect responses [2]. This suppression reduces interference.

The level of cognitive control recruited by the PFC scales with conflict intensity detected by the ACC. In high-conflict trials, reaction time (RT) is longer. However, across multiple trials, RT decreases due to conflict adaptations like interference reduction. This is known as the **Gratton effect**.

Besides automatic but incorrect response suppression, the PFC also modulates the **posterior parietal cortex (PPC)** and the **temporoparietal junction (TPJ)**. These posterior cortical regions contribute to attention reallocation and stimulus evaluation, facilitating the selection and execution of the correct response [4].

1.1.3 The Simon task and effect

In a typical Simon task, participants respond to a feature of a stimulus (e.g., the meaning of a word) while ignoring another feature (e.g., the color of the word). Reaction time (RT) is significantly faster when the relevant feature (e.g., word meaning) corresponds to the irrelevant feature (e.g., color). This is known as the **Simon effect** [5].

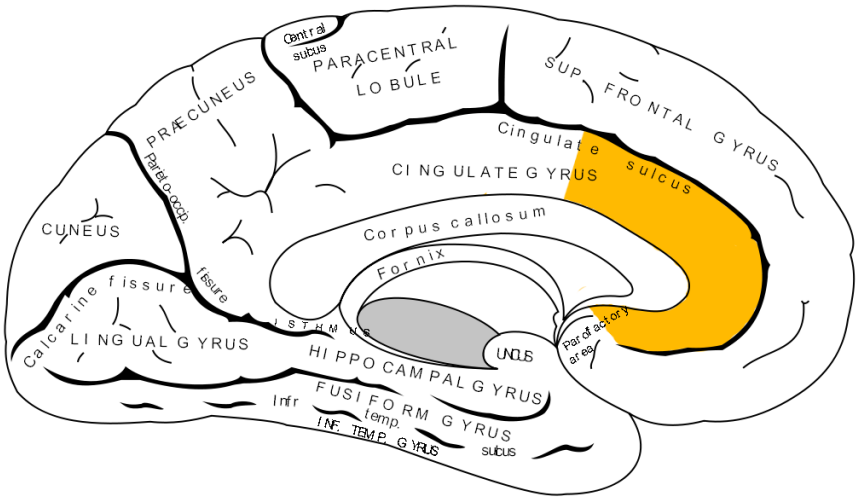

Figure 1: Anterior cingulate cortex (ACC) highlighted [7].

1.1.4 Measuring cognitive control

Several scalp potentials can be used to study different aspects of cognitive control. However, the **N2 component** (∼200–350 ms) of frontocentral electrodes (Fz, FCz, and Cz, see Figure 2) is a well-established **marker of ACC conflict detection prior to response**, making it the most appropriate EEG marker for this Simon task study [3].

For a schematic comparison of N2 against other measures, see Table 1.

Table 1: Comparison of different EEG components and their role in cognitive control studies.

EEG component	**Brain region**	**Function**	**Time window (ms)**	**Best task type**
N2	ACC	Conflict detection before response execution	∼200–350	Conflict tasks (Simon, Stroop, Flanker)
P300	PPC & TPJ	Stimulus evaluation, response selection	∼300–500	Oddball tasks, decision-making
FRN (Feedback-Related Negativity)	ACC & dopaminergic system	Feedback evaluation, learning signal	∼250–350 after feedback onset	Reinforcement learning tasks
ERN (Error-Related Negativity)	ACC & basal ganglia	Error detection, response monitoring	∼50–100 after an error	Error trials in cognitive tasks
Theta Power (4–7 Hz)	ACC-PFC coordination	Sustained cognitive control, adaptation	Frequency-domain analysis, not time-domain	Adaptive control tasks

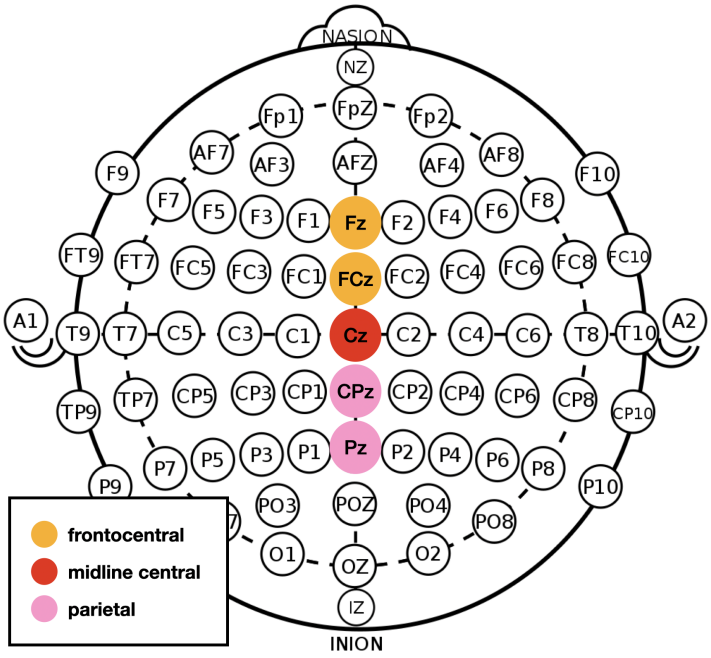

Figure 2: Electrodes of interest in cognitive control studies.
This study focuses on Fz, FCz, and Cz.

1.2 Hypothesis

IF response conflict is present in incongruent Simon task trials,
THEN the ACC will detect this conflict,
DUE TO the need to resolve interference between competing response tendencies.

1.3 Prediction

N2 amplitude at frontocentral electrodes (FCz, Cz, Fz) will be larger (more negative) in incongruent trials (S2) compared to congruent trials (S1).

2 Methods

2.1 Dataset

The Simon task EEG recordings used in this study were taken from Singh et al. (2022) [6], and are publicly available here. For this analysis, two subjects (sub-100 and sub-101) were selected.

The EEG data was recorded at a sampling rate of 500 Hz using a 64-channel electrode cap following the 10-20 standard system. Data includes event markers for stimulus onset, where "S1" corresponds to congruent trials and "S2" to incongruent trials.

2.2 Preprocessing pipeline

The preprocessing pipeline for this study was designed to prepare EEG data for event-related potential (ERP) analysis. It consists of two stages:

1. First, sub-100 and sub-101 datasets were manually cleaned for rejection of defective channels and artifacts. The code to replicate this manual cleanse was extracted and can be found here.
2. Then, automated steps followed for filtering, re-referencing, epoch extraction, and baseline correction. The code to replicate this automated cleanse can be found here.

See Table 2 for parameter values.

2.3 Analysis pipeline

The analysis pipeline follows a time-domain approach, focusing on Event-Related Potentials (ERPs). It extracts and compares N2 waveforms (∼00–350 ms) at frontocentral electrodes (FCz, Cz, Fz) in the Simon task, evaluating differences between congruent (S1) and incongruent (S2) trials across two subjects. The analysis pipeline code is available here.

For a single-run, unified pipeline (manual preprocess, automated preprocess, and analysis) use this code.

Table 2: Preprocessing pipeline specifications.

Parameter	Value	Description
Bad channel removal	Manual	Noisy electrodes were manually removed
Artifact rejection	Manual	Segments containing muscle artifacts, blinks, or drifts were rejected
Filter type	FIR (Kaiser Window)	Linear phase filtering, avoiding latency shifts
High-pass cutoff frequency	1 Hz	Removes slow drifts and DC offsets
Low-pass cutoff frequency	30 Hz	Removes high-frequency noise
Max passband deviation ripple	0.01	Ensures accurate filter response within the passband
Transition bandwidth	0.5 Hz	Controls the smoothness of frequency transition
Re-referencing	Average reference	Signals were re-referenced to the global average
Epoch extraction	-300 to 700 ms (time-locked to S1 and S2)	Extracted segments around stimulus onset
Baseline correction	-300 to 0 ms	Mean voltage in this period was subtracted to normalize the data
Interpolated channels	Spherical interpolation	Previously removed bad channels were reconstructed

3 Results

The number of valid trials per condition was:

Table 3: Number of trials per subject.

Subject	**Trials$_{S1}$ (Congruent)**	**Trials$_{S2}$ (Incongruent)**
sub_100	82	86
sub_101	80	87

In Figure 3, variability **across-subjects** is observed for the N2 component. Waveforms follow different patterns for each subject, most notable at electrode FCz. In Figure 4, the averaged N2 waveforms further highlight individual variability: sub-100 exhibits one peak, while sub-101 seems to exhibit two.

Whether the predicted pattern **across conditions** (S1 vs S2) is observed, is not clear by sheer visualization. Amplitude during S2 conditions was expected to be significantly more negative, but that's not observed in either Figure 3 nor figure 4. Therefore, the mean N2 amplitudes calculated in Table 4 are preferred for interpretation:

- For subject sub-100, N2 amplitude was slightly more negative in incongruent trials (-0.49 µV) than in congruent trials (-0.40 µV). This pattern aligns with the expected conflict detection mechanism. However, the effect size appears mild compared to prior literature.
- For subject sub-101, an unexpected and opposite pattern is observed, where the N2 amplitude was more negative in congruent trials (-2.15 µV) than in incongruent trials (-0.51 µV).

The original study did report significant N2 amplitudes for S2 conditions [6]. The contrast with these results is attributed to the **small size of the sample**, only 2 subjects being individually analyzed instead of 147. The characteristics of the 2 selected subjects was checked, and they are classified as part of the control group instead of the PD patients, discarding neurological conditions as a possible confounder.

Table 4: Mean amplitudes of N2 component.

Subject	**N2$_{S1}$ (Congruent)**	**N2$_{S2}$ (Incongruent)**
sub_100	-0.40 µV	-0.49 µV
sub_101	-2.15 µV	-0.51 µV

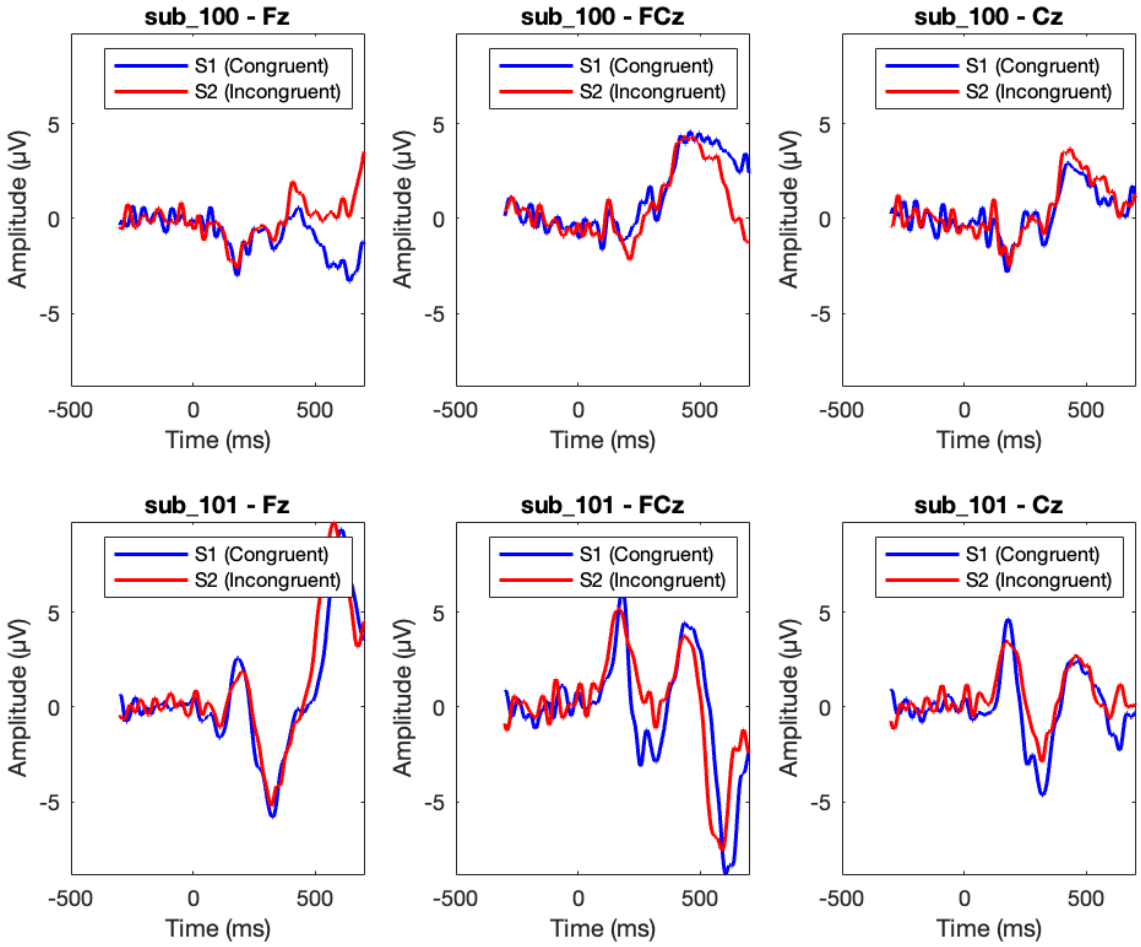

Figure 3: N2 component for each separate electrode.

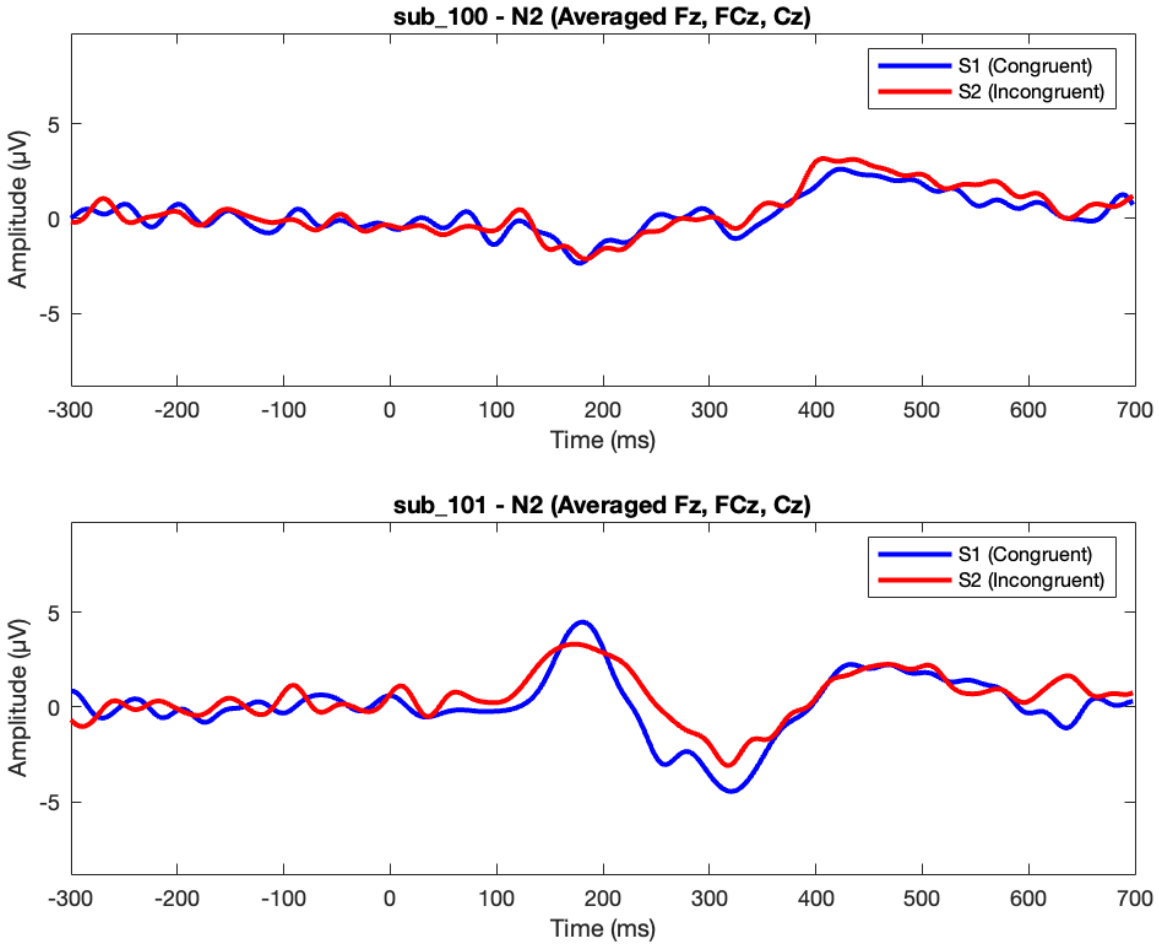

Figure 4: N2 component averaged across all electrodes.

References

[1] Botvinick, Matthew et al. (1999). "Conflict monitoring versus selection-for-action in anterior cingulate cortex". In: *Nature* 402.6758, pp. 179–181. DOI: 10.1038/46035. URL: https://doi.org/10.1038/46035.

[2] Botvinick, Matthew M. et al. (2001). "Conflict monitoring and cognitive control". In: *Psychological Review* 108.3, pp. 624–652. DOI: 10.1037/0033-295X.108.3.624. URL: https://doi.org/10.1037/0033-295X.108.3.624.

[3] Folstein, Jonathan R. and Cyma Van Petten (2008). "Influence of cognitive control and mismatch on the N2 component of the ERP: A review". In: *Psychophysiology* 45.1, pp. 152–170. DOI: 10.1111/j.1469-8986.2007.00602.x. URL: https://doi.org/10.1111/j.1469-8986.2007.00602.x.

[4] Polich, John (2007). "Updating P300: An integrative theory of P3a and P3b". In: *Clinical Neurophysiology* 118.10, pp. 2128–2148. DOI: 10.1016/j.clinph.2007.04.019. URL: https://doi.org/10.1016/j.clinph.2007.04.019.

[5] Simon, J. Richard and Alan P. Rudell (1967). "Auditory S-R compatibility: The effect of an irrelevant cue on information processing". In: *Journal of Applied Psychology* 51.3, pp. 300–304. DOI: 10.1037/h0020586. URL: https://doi.org/10.1037/h0020586.

[6] Singh, Arun et al. (2022). "Evoked midfrontal activity predicts cognitive dysfunction in Parkinson's disease". In: *medRxiv*. Preprint. DOI: 10.1101/2022.07.26.22278079. URL: https://doi.org/10.1101/2022.07.26.22278079.

[7] Wikimedia Commons Contributors (2024). *Gray727 Anterior Cingulate Cortex Image*. Accessed: February 27, 2025. URL: https://commons.wikimedia.org/wiki/File:Gray727_anterior_cingulate_cortex.png.