Case Study Poster

EEG and ERP Biomarkers Across Modifiable Risk Factors for Cognitive Decline

Metabolic, Cardiovascular, and Sleep-Related Case Findings

J. A. McCallen, MD[1]; Jeffrey Boone, MD[2]; S. Yathiraj, MD[3]; Marta Spada, BS, RPSGT[4]; Salina Yathiraj[5]; David S. Oakley[6]; Francis X Palermo, MD, MPH, MBA[7]

[1] Cenegenics Medical Institute, Denver, CO, [2] Boone Heart Institute, Denver, CO, [3] Palma Sola Neurology Associates; Roskamp Institute, [4] Sleep 360, [5] University of Miami, [6] WAVi Research,

Three clinical risk domains. Six patients. One recurring observation: measurable electrophysiological differences accompanied changes in modifiable health factors.

Background

Metabolic dysfunction, cardiovascular disease, and impaired sleep are established risk factors associated with cognitive decline. These risks are typically evaluated using laboratory testing, vascular assessment, and sleep studies, but such measures do not directly characterize concurrent electrophysiological activity.

Quantitative EEG and event-related potentials provide objective measurements of brain electrical activity and stimulus-evoked responses. Across these case reports, EEG and ERP metrics were evaluated alongside metabolic, cardiovascular, and sleep-related clinical findings to examine whether electrophysiological changes accompanied management of potentially modifiable risk factors.

Objective

To explore whether quantitative EEG and ERP biomarkers can reveal changes not fully captured by symptoms or conventional clinical measures during management of modifiable risk factors for cognitive decline.

Case 1- Cardiovascular Risk Management

An older adult previously diagnosed with Alzheimer's disease underwent additional evaluation that identified elevated blood pressure and vascular plaque burden. Baseline EEG showed reduced dominant frequency, while P300 measures provided additional context for reassessment of the clinical picture. Following physician-directed cardiovascular risk management, longitudinal EEG and ERP changes were documented alongside reported clinical improvement.

Case 1 Continued-

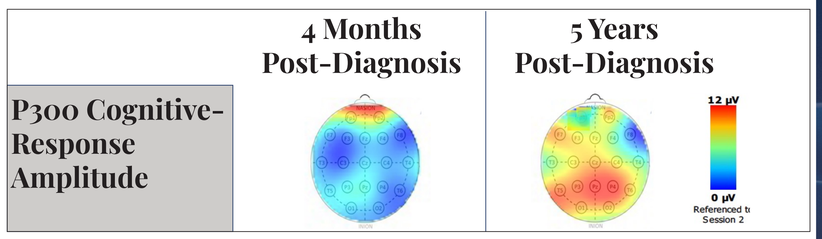

Figure 1. P300 amplitude topographs obtained four months and five years after diagnosis. The later assessment demonstrates greater central-parietal response voltage.

WAVi EEG/QEEG Biomarkers	4 Months Post-Diagnosis	5 Years Post-Diagnosis	Reference Ranges
Frontal			
F3/F4 Eyes Closed Alpha (Power)	1.4	1.0	0.9–1.1
Peak Frequency (7.0–13.0 Hz)	7.0 Hz	? 7.0 Hz	8.5–10.0 Hz
Test/Retest Change	-	0.0 Hz	±0.2 Hz
Central-Parietal			
Audio P300 Delay	352 ms	252 ms	322–419 ms
Test/Retest Change	-	-100 ms	±11 ms
Audio P300 Voltage	6.4 µV	11.5 µV	6–14 µV
Test/Retest Change	-	5 µV	±2 µV
Peak Frequency (7.0–13.0 Hz)	7.0 Hz	7.2 Hz	8.5–10.5 Hz
Test/Retest Change	-	0.2 Hz	±0.2 Hz
CZ Eyes Open Theta/Beta (Power)	N/A	N/A	0.6–1.4
CZ Eyes Closed Theta/Beta (Power)	1.0	⬓ 1.1	0.6–1.4
Occipital			
Peak Frequency (7.0–13.0 Hz)	7.0 Hz	12.5 Hz	9.0–11.0 Hz
Test/Retest Change	-	5.5 Hz	±0.2 Hz

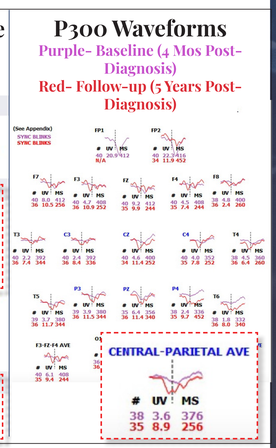

Figure 2. Longitudinal comparison of EEG and ERP measures. P300 latency decreased from 352 to 252 ms, P300 amplitude increased from 6.4 to 11.5 µV, and occipital peak frequency increased from 7.0 to 12.5 Hz between assessments. The waveform overlay shows baseline and follow-up responses across electrode sites.

Case 2- Sleep Apnea Management

Two adults with mild cognitive impairment and obstructive sleep apnea underwent EEG and ERP assessment. One accepted treatment for sleep apnea; the other declined treatment. The treated patient demonstrated greater P300 amplitude, shorter latency, and stronger coherence patterns than the untreated patient, together with better performance on selected reaction-time

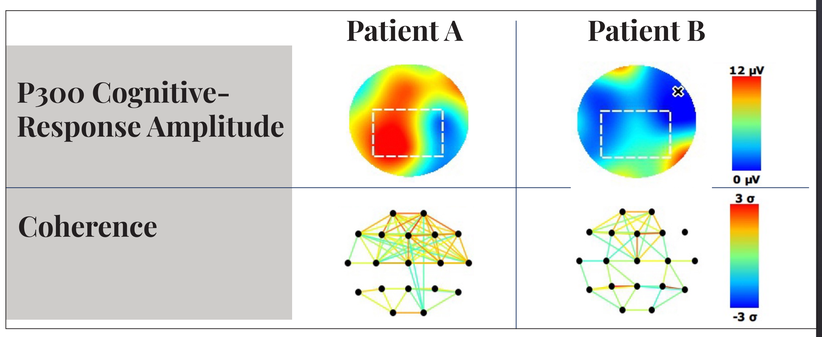

Figure 3. P300 amplitude topographies and coherence maps for two patients with obstructive sleep apnea. Patient A, who underwent treatment, demonstrated greater central-parietal response voltage and denser coherence patterns than Patient B, who declined treatment. The comparison is descriptive and does not establish treatment causality.

Case 3- Metabolic Risk Management

Three cognitively asymptomatic men entered a physician-directed wellness program with elevated metabolic risk markers, including HbA1C, fasting insulin, excess body fat, and low free testosterone. Following individualized dietary, exercise, hormonal, and clinical interventions, metabolic markers moved toward target ranges and body fat declined. P300 amplitude increased beyond expected test-retest variation in all three patients.

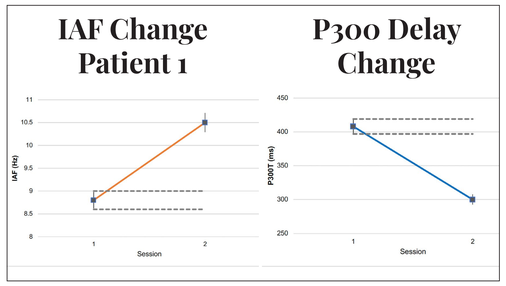

Figure 4. Individual alpha frequency and P300 latency in Patient 1 before and after intervention. Alpha frequency increased from 8.8 to 10.5 Hz, while P300 latency decreased from 408 to 300 ms, both exceeding expected within-person variation.

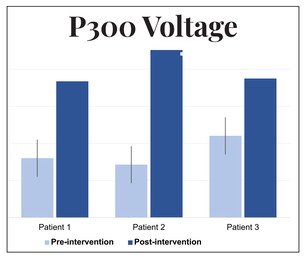

Figure 5. P300 amplitude increased across all three patients following intervention. Error bars indicate expected test-retest variation.

Objective EEG markers identified abnormalities that were not fully reflected by symptoms or standard cognitive testing. Especially with older patients in the study.

P300 Voltage and Clinical Outcomes

	Patient 1		Patient 2		Patient 3	
Marker (range)	**74.5 years**	**75.8 years**	**62.6 years**	**64.8 years**	**61.2 years**	**62.8 years**
HbA1C (< 5.1)	5.7	**5.3**	5.0	5.0	5.8	**5.1**
Insulin (< 5)	13.5	**2.8**	7.1	**3.4**	9.3	**4.8**
Free test (155–220)	60	**152**	27	**298**	25	**140**
Body fat	37%	**24%**	42%	**30%**	42%	**33%**
CNS VS percentile	35%	36%	56%	63%	49%	52%
P300V (6–16 µV), ΔP300V ± 2 µV	6 µV	**15 µV**	6 µV	**19 µV**	9 µV	**15 µV**

Figure 6. Metabolic and electrophysiological measures before and after intervention. Reductions in fasting insulin and body fat were accompanied by increased P300 amplitude across all three patients. The topographies illustrate the distribution of evoked response voltage across the scalp.

Conclusion

Across patients ranging from cognitively asymptomatic to previously diagnosed with Alzheimer's disease, quantitative EEG and ERP added objective electrophysiological context to metabolic, cardiovascular, sleep, and cognitive assessments. They documented longitudinal change during physician-directed management and, in one asymptomatic patient, identified abnormal P300 latency and individual alpha frequency despite largely normal neurocognitive testing.

These cases do not establish causation or treatment efficacy, but they raise a practical question: If clinicians routinely measure HbA1C, blood pressure, and sleep, should cognitive-risk assessment also include objective measurement of brain electrical activity?