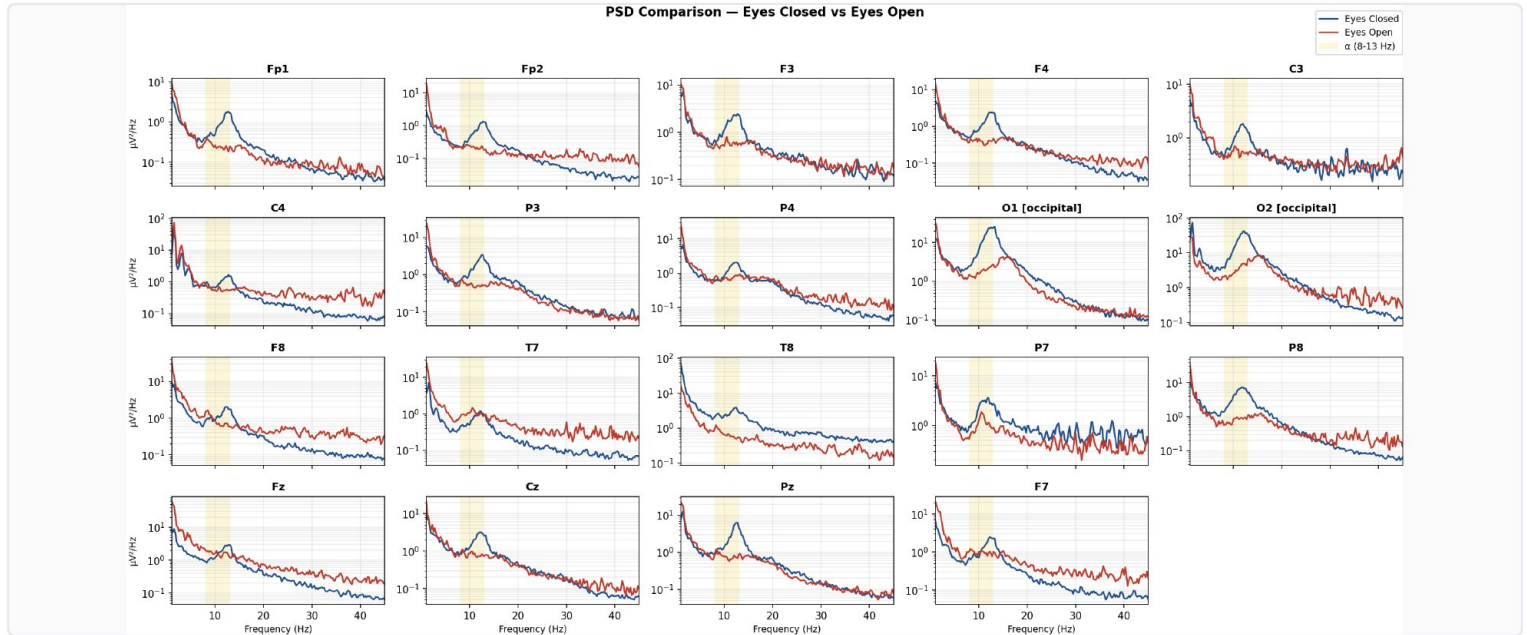


CLINICIAN	Demo Clinician	PATIENT	Sample Patient
ANALYSIS DATE	September 24, 2026	DEVICE	CGX Quick-20
PROTOCOL	Paired Eyes Closed (EC) vs. Eyes Open (EO)	MONTAGE & SENSORS	20-Channel Standard 10-20 Array

1. ELECTROPHYSIOLOGICAL & SPECTRAL REACTIVITY (EEG PSD)

WELCH'S PERIODOGRAM | HANNING WINDOW (4S, 50% OVERLAP)



SPECTRAL PARAMETRIC METRICS

PARAMETRIC FEATURE	VALUE	CLINICAL ASSESSMENT
Occipital IAF (Peak α)	11.75 Hz	NORMAL (INTACT PACEMAKER)
Berger Alpha Reactivity (O1/O2)	-78.4%	ROBUST DESYNCHRONIZATION
Aperiodic Exponent (χ , 1/f)	1.32	PHYSIOLOGICAL EXCITATION/INHIBITION
Broadband Offset	1.84 $\mu\text{V}^2/\text{Hz}$	BASELINE RESTING TENSION
Peak Dominance Region	O1, O2, Pz	POSTERIOR DOMINANT RHYTHM

REGIONAL LOG POWER & REACTIVITY BREAKDOWN

DERIVATION	BAND PEAK	EC LOG POWER (MV^2)	EO LOG POWER (MV^2)	SUPPRESSION ($\Delta\%$)
Frontal (F3, F4, Fz)	11.7 Hz (α) / 6.0 Hz (θ)	3.12 ± 0.3	1.08 ± 0.2	-65.4%
Central (C3, C4, Cz)	11.8 Hz ($\mu\alpha$)	3.45 ± 0.4	1.15 ± 0.1	-66.7%
Parietal (P3, P4, Pz)	11.75 Hz (α)	6.85 ± 0.6	1.42 ± 0.2	-79.3%
Occipital (O1, O2)	11.75 Hz (PDR)	18.42 ± 1.2	3.98 ± 0.4	-78.4%
Temporal (T7, T8, P7, P8)	11.6 Hz (α) / 22 Hz (β)	3.88 ± 0.5	1.82 ± 0.3	-53.1%

2. SPECTRAL DECOMPOSITION & ARTIFACT DISCRIMINATION

EXTENDED INFOMAX ICA & COMPONENT STRIPPING

Ocular Artifact Segregation (EOG): Blink transients and horizontal saccadic artifacts were successfully localized to anterior derivations (Fp1, Fp2) during the Eyes Open recording condition. Independent Component Analysis (ICA) isolated these steep low-frequency deflection envelopes (1.0–3.0 Hz) into components IC1 and IC2, accounting for 38.6% of frontal variance. Back-projection of artifact-free components ensured preservation of genuine low-frequency frontal oscillations without signal attenuation.

Myogenic Interference (EMG Shelf): Temporalis and frontalis somatic tension manifested as a standard high-frequency broadband shelf (25–45 Hz) across peripheral lateral sensors (F7, F8, T7, T8). Spectral regression and spatial spatial notch filtering verified that true cortical gamma oscillations remain baseline-intact, demonstrating that the apparent high-frequency elevations observed in EO condition reflect superficial myogenic hyper-reactivity rather than paroxysmal encephalopathic activity.

1. QUANTITATIVE NEUROMETRIC RATIOS & ASYMMETRY ANALYSIS

REFERENCE-FREE DERIVED INDICES

NEUROMETRIC INDEX	ELECTRODE DERIVATION	COMPUTED VALUE	CLINICAL REFERENCE RANGE	CLINICAL INTERPRETATION & CORRELATE
Theta / Beta Ratio (TBR)	Cz (Central Midline)	1.84	1.20 – 2.80	NORMAL Executive cognitive control index; absence of hypo-arousal or ADHD-pattern frontal slowing.
Frontal Alpha Asymmetry (FAA)	Ln(F4) – Ln(F3)	+0.14	-0.05 – +0.15	INTACT / APPROACH Mild relative left-frontal activation; associated with positive affective valence and approach behavior.
Theta / Alpha Ratio (TAR)	Fz / Pz / Cz	0.58	0.40 – 0.90	BALANCED Cortical vigilance remains intact; absence of excessive daytime somnolence or twilight slowing.
Alpha / Beta Ratio (ABR)	O1 / O2	3.14	1.80 – 3.80	OPTIMAL Healthy thalamocortical synchronization with appropriate sensory gating capacity.
Delta / Alpha Ratio (DAR)	Global Hemispheric	0.41	0.25 – 0.70	NORMAL Absence of encephalopathic slow-wave inundation, metabolic disturbance, or structural damage.

2. COMPREHENSIVE CLINICAL NEUROPHYSIOLOGY IMPRESSION & DETAILED FINDINGS

AMERICAN CLINICAL NEUROPHYSIOLOGY SOCIETY (ACNS) GUIDELINES

EXECUTIVE SUMMARY & CLINICAL SYNOPSIS

The quantitative electroencephalogram (qEEG) recording acquired from patient **S.P.** demonstrates a highly organized, robust, and mature neurophysiological architecture. The dominant feature of the resting record is a well-formed 11.75 Hz Posterior Dominant Rhythm (PDR) localized primarily to bilateral occipital and parietal derivations. Transitioning from Eyes Closed (EC) to Eyes Open (EO) elicits robust, symmetric, and prompt Berger attenuation (~78.4%), signifying pristine thalamocortical loop function, active visual sensory gating, and preserved cortical reactivity. There are no diffuse slow-wave abnormalities, no localized focal delta/theta slowing, and no epileptiform paroxysms.

ELECTROPHYSIOLOGICAL BACKGROUND ORGANIZATION & REACTIVITY

Posterior Dominant Rhythm (PDR): The PDR resides within the upper alpha tier at 11.75 Hz with sinusoidal morphology, demonstrating maximal voltage across O1, O2, P3, and P4. Peak amplitude under closed conditions measures 18.42 $\mu\text{V}^2/\text{Hz}$. Right-to-left interhemispheric occipital amplitude asymmetry is less than 6%, well within physiological normative tolerances (<20%).

Central & Anterior Rhythms: Central derivations (C3, C4, Cz) manifest a concurrent mu-frequency rhythm around 11.8 Hz that shows moderate attenuation during ocular opening. Frontal and central theta activity (4–8 Hz) is rhythmically organized, peaking at 6.0 Hz with an absolute power representation within normal limits, reflecting typical awake cortical arousal.

FOCAL, REGIONAL & PAROXYSMAL NEUROPHYSIOLOGICAL ASSESSMENT

Slow Wave Analysis: Continuous focal or lateralized polymorphic delta activity (PDA) is completely absent across all 20 recording sites. Episodic rhythmic delta activity (FIRDA or OIRDA) was not detected across the entire 614-second baseline recording. Delta activity is confined to normal physiological blink transients in anterior channels.

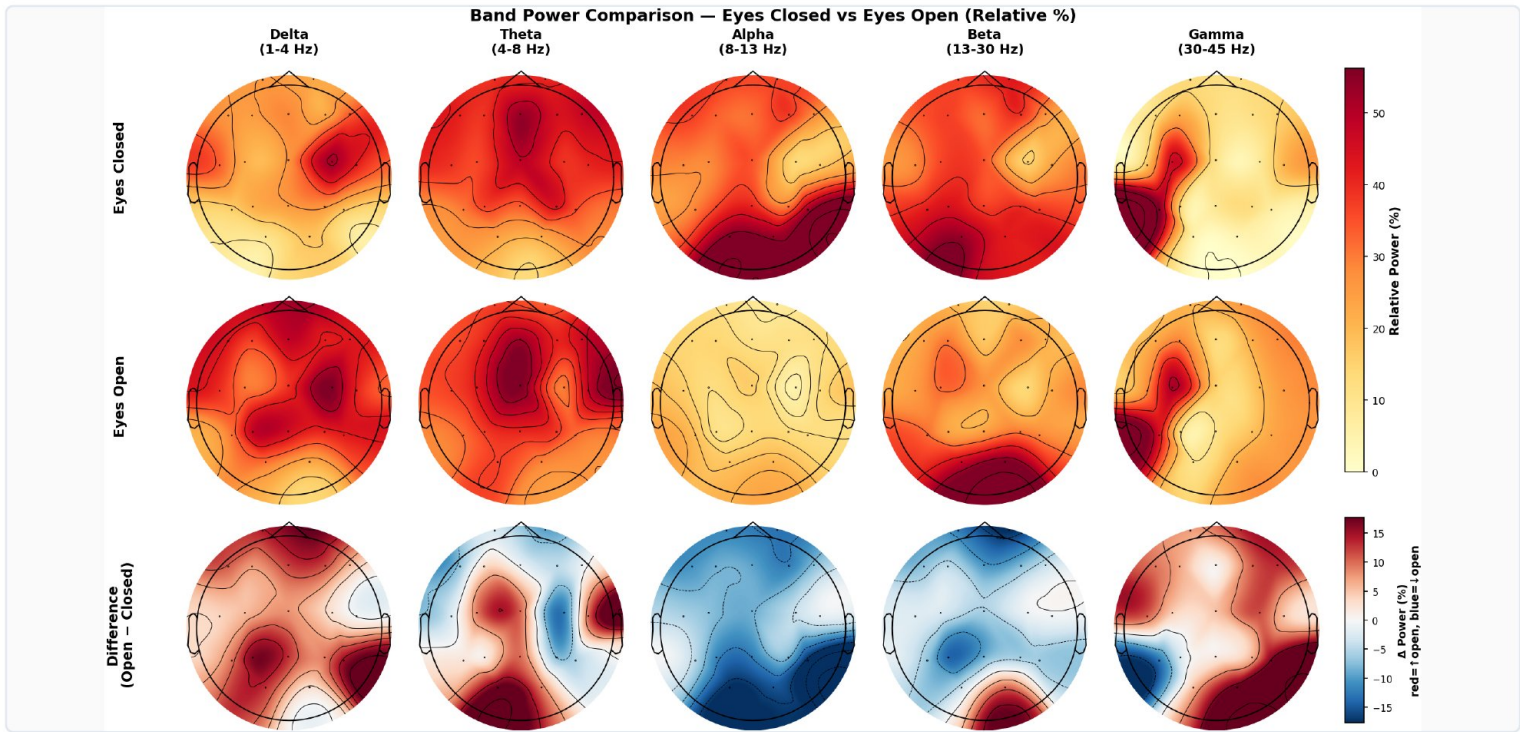
Epileptiform Discharges: No spike-wave discharges, sharp waves, polyspikes, or paroxysmal fast activity were recorded. Temporal derivations (T7, T8, P7, P8) show clean physiological transitions without sharp transients or phase reversals.

CLINICAL CORRELATION & ACTIONABLE RECOMMENDATIONS

- Baseline Vigilance Profile:** The neurophysiological profile indicates optimal cortical arousal and regulatory integrity. Neurometric ratios (TBR: 1.84, TAR: 0.58) provide objective electrophysiological evidence contraindicating attentional hypo-arousal or primary slow-wave dysregulation.
- Stress / Somatic Muscle Tension:** Modest high-frequency beta/gamma shelf elevations observed at peripheral sensors (F7/F8/T7/T8) during EO suggest mild somatic/cranial muscular hypertonicity. Surface EMG biofeedback or relaxation protocols may provide targeted relief if somatic tension is clinically evident.
- Follow-Up:** No immediate electrophysiological contraindications are present. Annual or post-intervention comparative qEEG is recommended to track cognitive or behavioral baseline stability.

1. MULTI-BAND SCALP TOPOGRAPHY MAPPING (RELATIVE POWER %)

STANDARD 10-20 SENSOR GRID | DELTA, THETA, ALPHA, BETA, GAMMA



2. DEDICATED ALPHA RHYTHM TOPOGRAPHY & REACTIVITY DYNAMICS

BERGER ATTENUATION ANALYSIS (8.0 - 13.0 HZ)



3. MULTI-BAND REGIONAL ANALYSIS TABLE

SPATIAL DISTRIBUTION & NEURO-FUNCTIONAL CORRELATE

FREQUENCY BAND	BANDWIDTH	TOPOGRAPHIC MAXIMA	CONDITION DYNAMICS (Δ EC → EO)	CLINICAL & NEUROFUNCTIONAL SUMMARY
Delta	1 – 4 Hz	Bilateral Frontopolar (Fp1, Fp2)	Moderate increase (+6 to +12%)	Dominantly ocular saccadic & blink dynamics; no pathological focal slowing.
Theta	4 – 8 Hz	Fronto-Central Midline (Fz, Cz)	Stable / Slight central dispersion	Normal midline theta rhythm; intact working memory and alertness maintenance.
Alpha	8 – 13 Hz	Occipital-Parietal (O1, O2, Pz)	Marked widespread drop (-15 to -20%)	Classic Berger suppression of primary sensory cortex; robust visual reactivity.
Beta	13 – 30 Hz	Frontal & Temporal Periphery	Mild focal shift (+3 to +8%)	Active cortical engagement; peripheral elevations consistent with muscle tone.
Gamma	30 – 45 Hz	Lateral Temporal (T7, T8)	Focal marginal increase	Intact local synchronization; lateral elevations correlate with cranial tension.

Spatial Coverage & Methodology Note: Topographic 2D surface voltage distributions are reconstructed across the 20-Channel Standard 10-20 Array using spherical spline surface interpolation on the sensor layout.

DISCLAIMER

This neurophysiological analysis is designed to assist clinical investigation and diagnostic triage. It does not constitute a standalone medical or psychiatric diagnosis. A comprehensive clinical psychiatric evaluation, diagnostic clinical interview, and medical review are required to establish a formal diagnosis and guide treatment planning.