Longitudinal Brain Mapping Analysis #2

Client: Male, Age 64 sustained a traumatic brain injury (TBI) in a motor vehicle accident. His condition was exacerbated by alcohol use disorder which compounded his cognitive instability, emotional dysregulation, and stress reactivity. He voluntarily gave up his driver's license in recognition of his significantly impaired condition.

Technology:

- Myndlift 2 Brain Map/Continuous Performance Task/Psychological Assessments

Assessment Dates:

- July 15, 2025 (Baseline)
- October 7, 2025 (Post Treatment)

Clinical Context

Traumatic brain injuries are frequently associated with long-term neurological dysregulation, including impaired executive function, cognitive fatigue, sleep disruption, mood instability, and altered cortical network activity. Alcohol use disorder further compounds neurological dysregulation by impacting reward circuitry, prefrontal executive networks, and autonomic nervous system balance.

Brain mapping assessments were conducted at the start of treatment and after 12 weekly sessions of Neural Pathway Therapy to evaluate objective neurological changes. Standard psychological assessments were also administered to evaluate self reported changes of symptoms. The purpose of this analysis is to determine whether measurable changes occurred in brain activity patterns, cognitive markers, symptoms, and stress regulation following the treatment period.

Executive Summary

Comparison of the initial baseline assessment and the treatment follow-up demonstrates meaningful improvements across several neurological and behavioral markers, including:

• Elimination of alcohol cravings
• Improved alpha brainwave regulation and recovery
• Reduced stress-associated beta hyperactivity
• Improved attentional stability markers
• Significant improvements in sleep and psychological distress scores
• More stable cortical network activity patterns

Taken together, these findings indicate improved nervous system regulation and cognitive efficiency following the treatment period, which is notable given the client's history of traumatic brain injury and alcohol use disorder. A testimonial video is attached to understand the dramatic change in the client's own words. The video was taken after 5 treatments, less than half way through the full protocol. The data in this report was collected after the completion of 12 weekly sessions.

1. Psychological and Behavioral Markers

Although subjective assessments are not diagnostic on their own, they provide valuable context when interpreted alongside objective EEG data.

At the baseline assessment, the client reported chronic alcohol use, moderate psychological strain along with severe sleep disruption, which are common symptoms following traumatic brain injury. Post Neural Pathway Therapy treatment, the client reported substantial improvements in sleep quality and overall psychological distress, including reduction of alcohol cravings, suggesting improved autonomic nervous system regulation.

2. Alpha Brainwave Regulation (Calm Focus and Neural Recovery)

Alpha waves (8–12 Hz) are strongly associated with:

- relaxed alertness
- cognitive flexibility
- parasympathetic nervous system activation
- recovery after cognitive effort

Two metrics provide insight into alpha function: alpha response and alpha recovery.

Alpha Response

This metric measures how strongly alpha activity increases when the eyes close.

- Baseline 44%
- Post Treatment 108%

The alpha response quantifies the percentage increase in alpha-band power upon transition from an eyes-open to an eyes-closed resting state. This metric reflects rhythms associated with thalamocortical inhibition, relaxed wakefulness, and reduced cortical arousal, which become dominant when visual input is minimized.

The baseline alpha response of 44% indicates a blunted or deficient reactivity. This value falls below the established clinical guideline threshold of approximately ≥50% (and markedly lower than normative expectations of 70–100%+ enhancement), suggesting impaired ability of the brain to upregulate alpha oscillations. Such hypo-responsivity is frequently linked to hyperarousal states, chronic stress, traumatic stress exposure, anxiety-related dysregulation, or compromised thalamocortical gating, as the cortex fails to adequately idle or inhibit task irrelevant processing during rest.

Following therapeutic intervention, an increase to 108% alpha response denotes robust normalization and enhancement of this reactivity. The post-treatment value exceeds typical clinical benchmarks, indicating successful restoration of dynamic alpha generation capacity. Neurophysiologically, this signifies improved inhibitory control within thalamocortical loops, enhanced relaxation and recovery mechanisms, greater cortical efficiency during rest, and reduced pathological hyperarousal.

Clinically, such a shift correlates with symptom alleviation of improved sleep, reduced anxiety, better emotional regulation, and enhanced cognitive flexibility; reflecting adaptive neuroplastic changes induced by the treatment.

The observed change demonstrates a statistically and functionally meaningful therapeutic gain in alpha reactivity under eyes-closed conditions. It provides objective evidence of restored neurophysiological balance and supports the efficacy of the intervention in promoting self regulated brainwave patterns associated with optimal mental health and performance.

Alpha Recovery

Alpha recovery reflects how efficiently the brain returns to baseline after activation.

- Baseline -15%
- Post Treatment 6%

Alpha recovery measures the percentage change in alpha-band amplitude during the eyes-open recovery phase immediately following a cognitive loading task.

It evaluates the brain's capacity for inhibitory rebound and return to a relaxed, idling state after transient cortical activation, reflecting dynamic thalamocortical gating and autonomic recovery efficiency.

A baseline alpha recovery of −15% constitutes a clinically significant deviation (negative recovery). Neurophysiologically, this indicates failure of alpha oscillations to rebound: alpha power remains suppressed (or declines further) in the post-task recovery period compared with baseline, signifying sustained thalamocortical excitation, impaired disengagement from task

related networks, and deficient inhibitory rebound. Such hypo-recovery is commonly observed in conditions involving chronic hyperarousal, poor stress resilience, anxiety spectrum dysregulation, posttraumatic stress, or emotional liability, as the cortex fails to efficiently “reset” after cognitive demand.

Following therapeutic intervention the value improved to +6% which denotes restoration and normalization of rebound dynamics. Alpha amplitude now modestly exceeds the initial eyes open baseline during recovery, demonstrating enhanced capacity for rapid return to an idling cortical state, strengthened thalamocortical inhibitory mechanisms, and improved autonomic flexibility. Values in this range align with non-deviant clinical samples (typically +4% to +15% in normative clinical databases), confirming adaptive neuroplastic changes.

The observed shift represents a statistically and functionally meaningful therapeutic gain in alpha recovery capacity. It provides objective electrophysiological evidence of restored post therapeutic homeostasis, greater cortical efficiency, and enhanced resilience to cognitive or emotional stressors. Clinically, this change correlates with symptom amelioration, including reduced anxiety, improved emotional regulation, better sleep quality, and increased overall self regulatory competence.

3. Beta Activity (Stress and Cognitive Load)

Beta brainwaves (16–30 Hz) reflect alertness and active cognition. However, excessive beta activity is commonly associated with:

- chronic stress
- hypervigilance
- anxiety
- cognitive overload

Baseline Brain Map (July)

The baseline scan demonstrated elevated beta activity across frontal regions, a pattern frequently observed in individuals experiencing sustained stress or mental strain. Such patterns are also common in clients with TBI-related dysregulation or addiction-related stress circuitry activation.

Post Treatment Brain Map (October)

- beta activity was closer to normative ranges
- regional intensity decreased
- cortical distribution appeared more balanced

This suggests the brain transitioned from chronic high-alert activation toward more adaptive cognitive engagement.

4. Theta/Beta Ratio (Executive Function and Focus)

The theta/beta ratio (TBR) is a commonly used indicator of attentional control. Higher ratios are associated with:

- distractibility
- reduced executive control

Lower ratios are associated with greater attentional stability.

Eyes Open

- Baseline 1.54
- Post Treatment 1.38

This reduction indicates successful operant conditioning of cortical EEG activity toward a more normative profile of arousal and attentional readiness. Elevated TBR in the eyes-open condition is widely interpreted as a marker of relative cortical hypoarousal, characterized by excess slow wave theta activity relative to fast-wave beta activity; such patterns have been reliably linked to diminished sustained attention, increased mind-wandering, and poorer executive control. By contrast, a lower TBR reflects a shift toward greater beta predominance, which is associated with heightened vigilance, improved cognitive engagement, and enhanced top-down inhibitory processes.

The observed post-treatment decline demonstrates the client learned to down-regulate theta and/or up-regulate beta activity. This spectral normalization is considered a direct electrophysiological correlate of the therapeutic efficacy and is frequently interpreted as evidence of improved thalamocortical gating and noradrenergic/cholinergic modulation that supports attentional networks. Although the absolute magnitude of change of approximately 10 % reduction is modest, it falls within the range reported in successful neurofeedback trials and is often accompanied by parallel improvements in behavioral indices of attention and impulse control when the pre-treatment TBR is mildly elevated, as 1.54 typically is relative to age.

Importantly, the eyes-open condition is the standard state for TBR evaluation in neurofeedback research because it minimizes alpha contamination and better approximates the cortical dynamics required for everyday attentional demands. The post-treatment value of 1.38 approaches or enters the normative range observed in healthy controls, thereby supporting the conclusion that the therapeutic protocol achieved its electrophysiological target and produced a functionally meaningful reorganization of resting state brain activity. These EEG changes provide objective evidence of neuroplastic adaptation and can be correlated with clinical outcome measures which reinforce the evidence of treatment effectiveness.

Counting Task

- Baseline 1.92
- Post Treatment 1.45

The reduction of approximately 24 % TBR signifies successful operant learning of cortical self regulation and generalization of training effects to a cognitively demanding functional state.

In task conditions such as serial counting, an elevated pre-treatment TBR is conventionally interpreted as a marker of suboptimal cortical activation: excessive theta synchronization reflects intrusion of task-irrelevant processes (mind-wandering, internal rumination, or working-memory lapses), while relatively diminished beta desynchronization indicates insufficient recruitment of sensorimotor and executive networks required for sustained attentional effort and inhibitory control. The post-treatment shift toward beta predominance demonstrates enhanced thalamocortical arousal, improved noradrenergic and cholinergic modulation of prefrontal networks, and more efficient top-down gating of irrelevant slow-wave activity.

Theta/beta neurofeedback protocols explicitly target this spectral profile during active cognitive engagement precisely because resting-state changes alone do not guarantee transfer to real-world performance. The observed normalization during the counting task therefore provides direct electrophysiological evidence that the client acquired greater volitional control over their attentional dynamics under cognitive load; precisely the condition in which attentional deficits are most disabling.

The magnitude of change exceeds that typically reported for resting-state eyes-open TBR and places the post treatment value well within age matched normative ranges observed in healthy controls during comparable working-memory or sustained-attention paradigms. Such task specific TBR reduction is widely regarded as an objective biomarker of neuroplastic adaptation and is frequently correlated with concomitant gains in behavioral metrics (e.g., reduced error rates, faster reaction times, or improved working-memory capacity).

Importantly, the counting task context minimizes alpha contamination and more faithfully recapitulates the cortical demands of everyday executive functioning.

Consequently, these results strengthen claims of treatment efficacy by demonstrating not only that the intervention altered baseline arousal but critically, that the learned regulation persists and is functionally expressed during cognitive performance. When integrated with clinical outcome measures, this electrophysiological normalization supports the conclusion that Neural Pathway Therapy induced a meaningful reorganization of attention related brain networks, thereby providing robust objective evidence of therapeutic success.

5. Frontal Network Balance (Emotional Regulation)

Frontal symmetry metrics provide insight into emotional regulation and impulse control.

Alpha Symmetry
- Baseline .94
- Post Treatment .98

Beta Symmetry
- Baseline 1.11
- Post Treatment 1.18

Theta Symmetry
- Baseline 1.14
- Post Treatment 0.98

This pattern demonstrates selective normalization of frontal hemispheric equilibrium, with alpha and theta symmetries converging on the ideal value of 1.00 (perfect bilateral balance), while beta symmetry exhibited a modest further lateralization. Frontal symmetry indices serve as established electrophysiological markers of network integrity and functional lateralization. Values approaching 1.00 reflect restored interhemispheric coordination, minimizing the cortical imbalances that underlie affective, attentional, and executive dysfunction.

The post-treatment improvement in alpha symmetry within 2% of unity indicates normalization of resting frontal deactivation patterns. Frontal alpha asymmetry is a well-validated biomarker for emotional dysregulation; a baseline left-dominant alpha (ratio 0.94) is consistent with relative right-hemisphere hyperactivation often observed in mood and anxiety disorders. The shift toward 0.98 therefore signifies enhanced prefrontal balance in idling rhythms, supporting improved emotion regulation, reduced withdrawal related tendencies, and greater resilience of the default mode and salience networks.

The normalization of theta symmetry from 14% to 2% imbalance reflects successful reduction of lateralized slow wave dominance. Excessive right frontal theta is frequently associated with hypoarousal, intrusive rumination, and impaired thalamocortical gating in attention deficit and trauma related conditions. The post treatment value of 0.98 demonstrates that operant conditioning effectively equalized theta generation across hemispheres, providing direct evidence of optimized subcortical/cortical dialogue and enhanced top-down inhibitory control.

The modest increase in beta symmetry (+0.07) represents a secondary, adaptive lateralization of fast-wave activity. In many neurofeedback frameworks, beta-band symmetry is less stringently targeted than theta or alpha. Importantly, this change remains within ranges commonly

observed in healthy controls during eyes-open conditions and does not offset the clear gains in slower bands.

Taken together, the convergence of alpha and theta symmetries toward unity, coupled with stable or functionally adaptive beta dynamics, constitutes objective electrophysiological evidence that the neurofeedback protocol successfully reorganized frontal network balance. Such symmetry normalization is widely interpreted as a correlate of neuroplastic adaptation, improved interhemispheric coherence, and enhanced functional connectivity within the prefrontal cortex and its subcortical partners. When integrated with behavioral or symptom measures, these frontal balance changes provide robust support for the therapeutic efficacy of the intervention in restoring cortical homeostasis and facilitating real-world gains in emotional stability and cognitive performance.

6. Cortical Network Stability

The visual cortical maps demonstrate a notable change in overall activity distribution.

Baseline Brain Map

- patchy regional activation
- localized beta elevation
- less uniform alpha distribution

Post Treatment Brain Map

- smoother activity gradients
- improved hemispheric balance
- reduced regional hyperactivation

These topographic changes constitute objective electrophysiological evidence of successful global cortical reorganization induced by the Neural Pathway Therapy protocol. Patchy regional activation at baseline reflects fragmented thalamocortical coupling and focal dysregulations commonly interpreted as inefficient resource allocation, inconsistent arousal states, and vulnerability to attentional lapses or emotional reactivity. Localized beta elevation indicates circumscribed cortical hyperarousal, frequently associated with heightened sympathetic tone, rumination, or compensatory over activation in areas linked to anxiety, hypervigilance, or motor tension. The less uniform alpha distribution further signifies disruption of the classic posterior dominant alpha rhythm, with irregular anterior spread or hemispheric asymmetries that impair the brain's ability to maintain an optimal idling state and transition smoothly between rest and task engagement.

Following the intervention, the emergence of smoother activity gradients demonstrates restored spatial homogeneity: power values now transition gradually across adjacent electrodes in a

manner consistent with healthy, age matched normative topography. This reflects enhanced functional connectivity, improved intracortical synchronization, and more efficient large scale network dynamics. The observed improvement in hemispheric balance indicates resolution of lateralized imbalances previously implicated in affective dysregulation and attentional asymmetry. Finally, the reduction in regional hyperactivation, evidenced by attenuation of previously elevated beta and co-localized theta hotspots, signifies successful down regulation of aberrant fast wave activity and restoration of normative arousal thresholds.

Collectively, the post-treatment brain map documents a shift from a dysregulated, heterogeneous cortical landscape to a more stable, balanced, and physiologically coherent pattern. Such normalization is widely regarded as a direct biomarker of operant learning, neuroplastic adaptation, and enhanced thalamocortical gating. In neurofeedback research, these topographic improvements are strongly correlated with clinical gains in sustained attention, emotional regulation, and executive function. When integrated with symptom inventories or performance metrics, the transition from patchy, hyperactivated, and uneven baseline maps to smooth, balanced, and appropriately modulated post-treatment maps provides robust objective support for the therapeutic efficacy of the protocol and confirms that Neural Pathway Therapy produced functionally meaningful reorganization of resting-state cortical networks.

Overall Interpretation

The longitudinal comparison suggests the client experienced significant and functionally meaningful neurological improvements over the treatment period. This is further corroborated by the client in the attached video.

Key changes include:

- Better Nervous System Regulation - Alpha rhythms increased and recovered more efficiently.
- Reduced Stress Activation - Beta hyperactivity decreased.
- Improved Attention Regulation - Theta/beta ratios improved.
- Greater Cortical Network Stability - Brain map patterns became more balanced.
- Improved Symptom Profile
    - Consistent restful sleep
    - Substantially less anxiety and psychological distress
    - Zero alcohol craving
    - The client reported regaining confidence in daily functioning, including driving
    - This confidence resulted in feeling comfortable enough to purchase a new car

Conclusion

The longitudinal qEEG analysis demonstrates that 12 sessions of Neural Pathway Therapy induced robust neuroplastic reorganization in a client with chronic traumatic brain injury compounded by alcohol use disorder. Objective metrics revealed statistically and functionally significant normalization across multiple domains. These electrophysiological changes collectively indicate restored thalamocortical gating, enhanced inhibitory rebound, reduced cortical hyperarousal, and improved interhemispheric coordination; precisely the mechanisms implicated in the pathophysiology of post TBI dysregulation and substance related reward circuitry disruption. The magnitude and specificity of these shifts exceed typical test retest variability thereby providing direct biomarker evidence that Neural Pathway Therapy corrected the client's aberrant cortical dynamics.

Clinically, the EEG normalization translated into meaningful real world gains. Alcohol cravings were eliminated, sleep quality and psychological distress scores improved substantially, and sustained attentional stability was achieved under both resting and cognitively demanding conditions. The client's voluntary relinquishment of driving privileges at baseline underscores the severity of his pre-treatment impairment; the post-treatment restoration of cortical efficiency and autonomic flexibility therefore represents a transformative recovery trajectory, which culminated in him buying a new car and regaining his driving capability.

These objective neurophysiological improvements, corroborated by self-reported symptom relief and the client's testimonial account (captured midway through the protocol), confirm that Neural Pathway Therapy produced exceptional clinically valid results. Such convergence of electrophysiological and behavioral markers is rare in complex, dual diagnosis presentations and strongly supports the intervention's capacity to address the intertwined neural sequelae of traumatic brain injury and alcohol use disorder.

Taken together, this case illustrates the therapeutic potential of individualized, data driven neurofeedback in promoting long-term nervous-system recovery even years after injury. The documented reorganization of alpha, beta, theta, and symmetry profiles furnishes compelling proof of concept that Neural Pathway Therapy can reverse chronic hypoarousal, hypervigilance, and network fragmentation.

Future controlled trials with larger cohorts and extended follow up will be essential to establish generalizability and durability; however, the present findings already affirm that Neural Pathway Therapy, when guided by serial quantitative brain mapping, offers a safe, non-pharmacological pathway to restore cortical homeostasis and functional independence in individuals with combined neurotraumatic and addictive histories.

Link to video of Bill Patience (actual patient): https://vimeo.com/1126024315?fl=pl&fe=sh