NEURORECOVERY ELITE: Accelerating Autonomic and Cerebral Post-Exertion Recovery Through Integrated Neurotechnological Interventions in Elite Sport

Alina ROBU, Alina-Diana NEMES

Abstract

In elite sport, congested competition schedules frequently require athletes to perform at maximal intensity with only 48–72 hours between matches. While traditional recovery strategies emphasize muscular and metabolic restoration, emerging evidence indicates that incomplete cerebral and autonomic recovery may compromise decision-making, emotional regulation, and tactical consistency. The present randomized controlled longitudinal study evaluated the efficacy of an integrated neurotechnological protocol designed to accelerate neurophysiological recovery following high-intensity competition. Forty professional athletes were randomly assigned to an experimental group receiving a five-component intervention (EEG neurofeedback, transcutaneous vagal nerve stimulation, HRV biofeedback for heart–brain coherence, transcranial photobiomodulation, and binaural auditory entrainment) or to a control group undergoing standard recovery procedures. Assessments were conducted pre-match and at 24, 48, and 72 hours post-match, including quantitative EEG (High Beta, SMR activity in prefrontal regions), heart rate variability (RMSSD, LF/HF ratio), HRV coherence, subjective cognitive fatigue, and decisional clarity ratings. Compared to controls, the experimental group demonstrated significantly greater increases in RMSSD (+15.77 ms), greater reductions in LF/HF ratio (–1.26), marked decreases in High Beta activity (–9.22 μV²), increased SMR power (+4.41 μV²), and enhanced heart–brain coherence (+0.44). These physiological improvements were paralleled by substantial reductions in perceived cognitive fatigue and improved decisional clarity, confirmed by coaching staff evaluations. The findings support the hypothesis that neurophysiological recovery can be accelerated to within 24–36 hours through integrated intervention, providing a reproducible and clinically applicable framework for sustaining performance in high-density competitive contexts.

Keywords: *Neurophysiological recovery; Heart rate variability (HRV); EEG neurofeedback; Heart–brain coherence; Vagal stimulation; Photobiomodulation; Sport performance*

1. Introduction and Scientific Rationale

In contemporary elite sport, competitive calendars have become increasingly congested, with athletes frequently required to perform at maximal intensity in matches separated by only 48 to 72 hours. Under these conditions, recovery is no longer a supportive process but a strategic determinant of performance. While traditional recovery paradigms have emphasized muscular repair, metabolic restoration, glycogen resynthesis, hydration, and sleep optimization, the neurophysiological dimension of recovery, encompassing central nervous system regulation and autonomic balance, has received comparatively limited systematic attention. Emerging evidence suggests that this imbalance in focus may contribute to unexplained fluctuations in decision-making quality, emotional stability, and injury risk during dense competition periods.

Cognitive fatigue has been shown to impair executive functioning, reaction time, inhibitory control, and tactical decision-making in athletes (Smith et al., 2018). These deficits may occur even when peripheral physiological markers such as creatine kinase levels or perceived muscle soreness have returned to baseline. Neurocognitive fatigue affects the prefrontal cortex and associated executive control networks, compromising working memory, attentional flexibility, and response inhibition, functions that are critical in high-speed, high-pressure match contexts. In team sports such as football, basketball, or handball, micro-decisions made within milliseconds often determine competitive outcomes. Thus, even subtle decrements in cortical efficiency may translate into tangible performance losses.

Parallel to cortical alterations, the autonomic nervous system (ANS) plays a fundamental role in post-exertional recovery. Heart rate variability (HRV), particularly the root mean square of successive differences (RMSSD), is widely recognized as a non-invasive index of parasympathetic (vagal) activity and autonomic adaptability (Shaffer & Ginsberg, 2017). Following intense physical and emotional exertion, HRV parameters typically decrease, reflecting sympathetic predominance and reduced vagal tone. Although peripheral physiological markers may normalize within 24 hours, HRV recovery can require 24–72 hours depending on emotional load, contextual stressors, and individual resilience (Plews et al., 2013; Stanley et al., 2013). Persistently reduced HRV is associated with impaired emotional regulation, decreased executive performance, and elevated injury risk (Thayer & Lane, 2007).

This dissociation between peripheral and central recovery suggests that athletes may appear physically ready while remaining neurophysiologically compromised. Incomplete autonomic restoration has been linked to increased impulsivity, diminished stress tolerance, and decreased tactical inhibition under pressure (Thayer & Lane, 2007). In practical terms, this may manifest as avoidable fouls, poorly timed actions, attentional lapses, or emotional reactivity during decisive phases of competition.

Electrophysiological research further supports the existence of prolonged cortical activation following demanding competition. Elevated power in the High Beta frequency band (22–30 Hz), particularly in prefrontal and anterior cingulate regions, has been associated with hyperarousal, sustained cognitive effort, and mental fatigue (Thun et al., 2015). Persistent High Beta activity beyond 24–48 hours post-competition may reflect ongoing executive strain and incomplete neural downregulation. Conversely, sensorimotor rhythm (SMR; 12–15 Hz) and Alpha activity are associated with calm alertness, efficient cortical inhibition, and optimized attentional control (Gruzelier, 2014). Failure to reestablish these adaptive oscillatory patterns may contribute to what can be termed “silent cognitive fatigue,” in which subjective readiness masks subtle neuroelectric inefficiencies.

Beyond isolated cortical regions, large-scale brain networks are central to understanding recovery dynamics. The Default Mode Network (DMN), typically active during introspection and self-referential processing, must appropriately deactivate during task engagement. Prolonged post-competition hyperactivity of the DMN has been associated with rumination and mental fatigue (Raichle, 2015). The Salience Network (SN), responsible for detecting behaviorally relevant stimuli and coordinating network switching, may remain in a state of hyperalertness following emotionally intense matches, sustaining sympathetic activation. Meanwhile, the Executive Control Network (ECN), encompassing dorsolateral prefrontal and anterior cingulate regions (including Brodmann areas 10, 24, 32, and 46), may exhibit transient functional depletion, impairing attention regulation and inhibitory control. Efficient recovery requires the recalibration of these interacting networks rather than merely the attenuation of muscle soreness.

Inflammatory processes provide an additional biological link between physical exertion and cognitive performance. Acute elevations in pro-inflammatory cytokines such as interleukin-6 (IL-6) and C-reactive protein (CRP) following intense exercise have been associated with transient reductions in cognitive

flexibility and executive function (Reynolds et al., 2020). Neuroinflammatory responses may influence prefrontal cortical functioning and interact with autonomic dysregulation, further delaying comprehensive recovery.

Taken together, current literature indicates that post-match recovery must be conceptualized as a multidimensional neurophysiological process involving autonomic recalibration, cortical oscillatory normalization, network reintegration, and inflammatory modulation. However, existing recovery protocols in professional sport predominantly emphasize passive or peripheral strategies, with limited integration of targeted neurotechnological interventions. The Neurorecovery Elite framework emerges from this gap. It proposes that recovery of the brain and autonomic system should be actively trained and clinically monitored in the same structured manner as strength or endurance. Neurofeedback targeting High Beta reduction and SMR enhancement, transcutaneous vagal nerve stimulation to accelerate parasympathetic reactivation, HRV biofeedback to restore heart–brain coherence (McCraty & Shaffer, 2015; Lehrer & Gevirtz, 2014), photobiomodulation to support mitochondrial and vascular recovery (Hamblin, 2016), and binaural auditory entrainment to consolidate adaptive oscillatory states (Orozco Perez et al., 2020) represent complementary mechanisms addressing distinct components of the recovery cascade. The central hypothesis underpinning this approach is that neurophysiological recovery can be accelerated by at least 40–50%, reducing the effective restoration window from 72 hours to approximately 24–36 hours without compromising functional stability. If validated, such acceleration would allow athletes competing in congested schedules to maintain executive clarity, emotional balance, and tactical precision across successive matches.

In modern elite sport, performance sustainability depends not only on training load management but also on precise neurophysiological recalibration. Recovery is no longer merely the absence of fatigue; it is the active restoration of neural efficiency, autonomic adaptability, and integrative heart–brain synchronization. The scientific rationale for Neurorecovery Elite is therefore grounded in converging evidence from psychophysiology, cognitive neuroscience, and sports performance research, all pointing toward a single conclusion: without complete cerebral and autonomic recovery, consistent high-level performance cannot be maintained.

2. Objectives of the NEURORECOVERY ELITE Study

The NEURORECOVERY ELITE study was designed to clinically validate an advanced, integrated neurotechnological protocol aimed at accelerating cerebral and autonomic recovery following high-intensity competitive matches. In professional sport, where athletes frequently compete at 48–72-hour intervals, maintaining consistent decision-making performance under repeated physical and emotional load represents a central performance challenge. The present study therefore moves beyond the traditional concept of “faster recovery” and instead investigates whether neurophysiological recalibration can be objectively measured, systematically accelerated, and functionally translated into sustained tactical clarity.

The general objective of the study was to evaluate the effectiveness of a combined neurotechnological intervention in three primary domains: (1) acceleration of autonomic recovery as indexed by HRV parameters (RMSSD, SDNN, LF/HF ratio); (2) reduction of neurocognitive fatigue as measured through quantitative EEG markers, particularly High Beta activity in prefrontal and cingulate regions (Brodmann areas 10, 24, 32, and 46); and (3) restoration of affective–decisional performance through improvement in heart–brain coherence and executive network stabilization.

Several specific objectives were derived from this overarching aim. First, the study sought to shorten the time required for autonomic markers to return to baseline levels. Given evidence that reduced RMSSD and

altered LF/HF ratios are associated with diminished executive control and emotional regulation (Shaffer & Ginsberg, 2017; Stanley et al., 2013), the intervention aimed to restore parasympathetic dominance within 24 hours post-match in the experimental group.

Second, the protocol targeted the reduction of cortical fatigability, operationalized as elevated High Beta power in prefrontal areas. Persistent High Beta activation following competition has been associated with hyperarousal and cognitive rigidity (Thun et al., 2015). The objective was therefore to achieve a significant decrease in High Beta activity alongside an increase in SMR (12–15 Hz), a rhythm linked to stable attention and efficient cortical inhibition (Gruzelier, 2014), within the first 24–36 hours post-effort.

Third, the study aimed to enhance heart–brain coherence, measured through HRV coherence indices centered around the 0.1 Hz resonance frequency. Increased coherence has been correlated with improved emotional regulation, psychophysiological resilience, and cognitive clarity (Lehrer & Gevirtz, 2014; McCraty & Shaffer, 2015). The objective was to elevate coherence values to clinically meaningful thresholds within 24 hours after competition.

Fourth, the intervention sought to reduce cumulative fatigue across autonomic, neuroelectric, and subjective dimensions, shortening the integrative recovery window from approximately 72 hours to 36 hours or less. This integrative approach recognizes that isolated normalization of either EEG or HRV is insufficient if not accompanied by perceived cognitive restoration and observable decisional readiness (Smith et al., 2018).

Finally, a strategic objective of the study was to validate a standardized, reproducible, and non-invasive recovery protocol that can be feasibly implemented within elite sports clubs during congested competitive cycles. The ultimate goal is not merely improved physiological metrics, but the preservation of stable executive performance and emotional balance between closely scheduled matches. Through these objectives, NEURORECOVERY ELITE positions neurophysiological recovery as a measurable, trainable, and strategically actionable component of high-performance sport.

3. Study Design of NEURORECOVERY ELITE

The NEURORECOVERY ELITE study was developed as a clinically rigorous and ecologically valid investigation aimed at testing an integrated neurotechnological recovery protocol under real competitive conditions. The central premise of the design was that post-match neurophysiological recovery must be assessed longitudinally and multidimensionally in order to capture both the speed and the depth of cerebral and autonomic recalibration.

Two guiding principles informed the study architecture. First, the intervention required strong clinical and neurophysiological validity, supported by standardized, objective, and reproducible measurements. Second, the protocol needed to demonstrate practical feasibility within elite sports environments, ensuring that it could be integrated into existing microcycles without interfering with physical or tactical planning.

3.1 Type of Study

NEURORECOVERY ELITE was structured as a randomized, longitudinal, controlled clinical study with two parallel arms:

- **Experimental group**: received the complete integrated neurotechnological protocol within 24 hours after the match.

- **Control group**: underwent identical assessment procedures but followed standard recovery strategies (active rest, hydration, stretching), without targeted cerebral or autonomic interventions.

This dual-arm design enabled isolation of the intervention's specific neurophysiological effects while controlling for spontaneous recovery processes and placebo-related influences. By comparing objective markers (EEG, HRV, coherence indices) across groups, the study aimed to determine whether accelerated recovery could be attributed to the intervention rather than natural physiological restitution.

The study adhered to CONSORT principles for randomized clinical trials (Schulz et al., 2010), particularly regarding allocation transparency, standardized measurement points, and parallel outcome assessment.

3.2 Intervention Window

Each participant was monitored over a 72-hour post-match recovery window, corresponding to the typical interval between competitive fixtures in elite sport. The intervention schedule was structured into three intervention sessions, each lasting 60 minutes, conducted at 12 hours, 36 hours, and 60 hours post-match. Assessment points were standardized across both groups:

- **T0** – Pre-match baseline
- **T1** – 24 hours post-match
- **T2** – 48 hours post-match
- **T3** – 72 hours post-match

These time points were selected to reflect the real competitive rhythm in professional leagues and to allow precise tracking of autonomic and cortical recovery trajectories. This structure enabled the analysis of early acceleration effects (within 24–36 hours) as well as sustained normalization across the 72-hour window.

3.3 Study Population and Participant Selection

The study enrolled a total of **40 professional athletes**, randomly assigned to two groups of 20 participants each.

Targeted athlete profiles included football, basketball, and handball players competing in schedules characterized by at least two matches per week. The inclusion criteria ensured homogeneity in competitive load and neurophysiological strain:

- Age between 18 and 35 years
- Participation in an official match within the previous 24 hours
- Regular exposure to congested competition cycles (≥2 matches/week)
- No neurological, psychiatric, or cardiovascular disorders
- RMSSD < 35 ms at 24 hours post-match and/or High Beta activity >20% in BA10/BA24 regions

Exclusion criteria included:

- Use of stimulants or medication influencing EEG or HRV
- Absence from any post-match session
- Recent traumatic injury or severe sleep deprivation

This selection strategy ensured that participants exhibited measurable post-match neurophysiological strain while minimizing confounding medical factors.

3.4 Experimental Intervention

Each intervention session consisted of five sequential components applied in an integrated manner. The structure remained constant across sessions, while parameters were individualized based on baseline (T0) EEG and HRV measurements. The intervention structure is presented below:

Component	Duration	Primary Clinical Objective
Neurofeedback (EEG)	20 min	Reduction of cortical fatigability; increase in SMR
Transcutaneous Vagal Stimulation	10 min	Parasympathetic activation; LF/HF reduction
HRV Coherence Training	15 min	Heart–brain synchronization; affective restoration
Photobiomodulation	10 min	Cortical inflammation reduction; neural reset
Binaural Audio Therapy	5 min	Alpha–SMR rhythmic stabilization; cognitive calm

The sequential order was designed to first reduce cortical hyperactivation, then recalibrate autonomic tone, restore integrative coherence, support biological recovery processes, and finally consolidate adaptive oscillatory patterns.

3.5 Control Group Procedures

Participants in the control group underwent conventional recovery practices commonly implemented in professional clubs. These included:

- Guided active rest and assisted stretching sessions
- General breathing exercises without real-time biofeedback
- No EEG-based intervention
- No vagal stimulation
- No photobiomodulation or auditory entrainment

The aim was not to deprive participants of recovery but to replicate current standard practice, thereby providing an ecologically valid comparison condition.

3.6 Evaluation Measures

Assessments were conducted at T0, T1, T2, and T3 and included objective, subjective, and observational indicators. The primary instruments were:

- **Comprehensive HRV analysis**: RMSSD, SDNN, LF/HF ratio, HRV coherence index
- **19-channel quantitative EEG (qEEG)**: High Beta, Alpha, and SMR activity in BA10, BA24, BA32, BA46
- **Subjective cognitive fatigue scale (0–10)**
- **Perceived mental and affective clarity ratings**

- **Coaching staff observational score** for decisional performance at 48 hours

This multidimensional measurement system allowed triangulation of recovery across autonomic, cortical, inflammatory, and functional domains. The inclusion of both subjective and staff-based evaluations provided ecological validation of neurophysiological findings.

The study was implemented through mobile EEG clinical units or designated recovery spaces within club training facilities. All devices used for neurofeedback, HRV monitoring, vagal stimulation, and photobiomodulation were CE-certified medical-grade systems.

The design of NEURORECOVERY ELITE was intentionally structured to evaluate not merely whether athletes recover, but how rapidly and how completely they recover at the cerebral and autonomic levels. By integrating methodological rigor with practical applicability, the study provides a framework for translating neurophysiological science into competitive advantage within elite sport.

4. Structure of the NEURORECOVERY ELITE Intervention

The NEURORECOVERY ELITE intervention was conceived as an integrated neurotechnological sequence targeting the principal mechanisms underlying post-match neurophysiological fatigue. Rather than functioning as a collection of isolated techniques, the protocol operates as a coordinated recovery cascade designed to recalibrate cortical excitability, restore autonomic balance, re-establish heart–brain synchronization, and biologically support neural restoration. Each session lasts 60 minutes and follows a standardized five-stage structure, with parameters individualized according to baseline EEG and HRV measurements.

The intervention begins with **targeted EEG neurofeedback** (20 minutes), aimed at reducing excessive High Beta activity (22–30 Hz) in prefrontal and anterior cingulate regions, particularly Brodmann areas 10, 24, and 46. Persistent High Beta activation following competition reflects sustained executive strain and hyperarousal (Thun et al., 2015). Through operant conditioning mechanisms, athletes are trained to downregulate maladaptive oscillatory patterns while enhancing sensorimotor rhythm (SMR; 12–15 Hz), a frequency band associated with calm attentional stability and improved inhibitory control (Gruzelier, 2014). This phase targets the functional recalibration of executive control networks and reduces residual cognitive rigidity.

The second phase consists of **transcutaneous vagal nerve stimulation (tVNS)** (10 minutes), applied auricularly to activate parasympathetic pathways. Vagal stimulation influences central autonomic nuclei and facilitates rapid restoration of vagal tone, reflected in improved HRV parameters (Breit et al., 2018; Frangos et al., 2015). By attenuating sympathetic dominance and promoting parasympathetic re-engagement, this component accelerates autonomic stabilization during the critical early recovery window.

Following autonomic activation, the protocol integrates **HRV biofeedback focused on heart–brain coherence** (15 minutes). Using guided resonance breathing around 0.1 Hz, athletes train synchronization between cardiac oscillations and prefrontal regulatory circuits. Increased coherence has been associated with enhanced emotional regulation, improved executive functioning, and greater psychophysiological resilience (Lehrer & Gevirtz, 2014; McCraty & Shaffer, 2015). This phase operationalizes integrative recovery by unifying cortical and autonomic dynamics into a coherent physiological state.

The fourth component involves **transcranial photobiomodulation (PBM)** (10 minutes), delivered at near-infrared wavelengths (approximately 810 nm) to frontal and parietal regions. Photobiomodulation has been shown to enhance mitochondrial ATP production, support cerebral blood flow, and modulate inflammatory signaling pathways (Hamblin, 2016; Iaccarino et al., 2016). Within the recovery context, PBM

is intended to biologically facilitate cortical restoration and reduce neuroinflammatory burden associated with intense exertion.

The session concludes with **binaural auditory entrainment** (5 minutes), using Alpha (≈10 Hz) and SMR-range frequencies to consolidate adaptive oscillatory states. Binaural stimulation can induce frequency-following responses that stabilize cortical rhythms associated with calm alertness and cognitive integration (Orozco Perez et al., 2020). This final phase serves to reinforce the neuroelectric patterns achieved during neurofeedback while promoting a balanced and sustainable post-session state.

Together, these five components form a sequential and synergistic intervention model. EEG regulation addresses cortical fatigue, vagal stimulation restores autonomic equilibrium, HRV coherence training integrates heart and brain dynamics, photobiomodulation supports biological neural recovery, and auditory entrainment consolidates adaptive oscillatory stability. The structure reflects a systems-level approach to recovery, recognizing that optimal post-match restoration requires coordinated recalibration across neural, autonomic, and integrative regulatory networks.

5. Results

The results demonstrate a consistent and clinically meaningful acceleration of neurophysiological recovery in the experimental group compared to standard recovery procedures. Across autonomic, cortical, integrative, and behavioral domains, the intervention produced effects that were not only statistically substantial but also functionally relevant within the 48–72-hour competitive window.

Table 1. Summary of Post-Match Changes (Mean ± SD)

Parameter Evaluated	Experimental Group	Control Group
Δ RMSSD (vagal activation)	+15.77 ± 3.11 ms	+3.46 ± 2.13 ms
Δ LF/HF (sympathetic balance ↓)	–1.26 ± 0.33	–0.33 ± 0.22
Δ EEG High Beta (BA10/24/46)	–9.22 ± 1.66 μV²	–2.29 ± 1.45 μV²
Δ EEG SMR (calm focus ↑)	+4.41 ± 1.89 μV²	+0.98 ± 0.85 μV²
Δ HRV Heart–Brain Coherence	+0.44 ± 0.09	+0.11 ± 0.05
Δ Subjective Cognitive Fatigue (0–10)	–5.02 ± 1.51 points	–0.96 ± 0.81 points
Δ Decisional Clarity (Self-report)	+2.57 ± 0.47 / 7	+0.73 ± 0.41 / 7
Δ Decisional Clarity (Staff rating)	+1.89 ± 0.48 / 5	+0.39 ± 0.37 / 5

Autonomic Recovery: RMSSD and LF/HF

The increase in RMSSD of +15.77 ms in the experimental group represents a robust restoration of parasympathetic activity within the first 24–36 hours post-match. RMSSD is widely recognized as a reliable index of vagal tone and autonomic flexibility (Shaffer & Ginsberg, 2017). In elite athletes, suppressed RMSSD values following competition have been associated with delayed recovery, impaired emotional regulation, and reduced executive functioning (Stanley et al., 2013). The comparatively modest increase observed in the control group (+3.46 ms) reflects natural autonomic restitution but does not indicate full parasympathetic recalibration within the same timeframe.

The reduction in LF/HF ratio (–1.26 in the experimental group) further confirms the shift from sympathetic dominance toward autonomic balance. Elevated LF/HF ratios following competition are linked to persistent physiological hyperarousal and diminished cognitive inhibition (Thayer & Lane, 2007). The magnitude of change observed suggests that the integrated protocol effectively attenuated residual stress activation, enabling athletes to exit the post-competition alert state more rapidly than through passive recovery alone.

Cortical Oscillatory Normalization: High Beta and SMR

Quantitative EEG findings revealed a pronounced decrease in High Beta power (–9.22 μV^2) in prefrontal and anterior cingulate regions (BA10, BA24, BA46) within the experimental group. Elevated High Beta activity is commonly interpreted as a biomarker of sustained cognitive effort, hypervigilance, and mental fatigue (Thun et al., 2015). Persistent High Beta following exertion may reflect incomplete executive disengagement and ongoing rumination related to tactical stressors. The substantially smaller decrease in the control group suggests that spontaneous downregulation is slower and less complete within the 72-hour window.

Concurrently, SMR power increased by +4.41 μV^2 in the experimental condition. SMR enhancement has been associated with stable attentional focus, improved motor inhibition, and reduced cortical noise (Gruzelier, 2014). In performance contexts, elevated SMR correlates with efficient executive control under pressure. The observed shift from High Beta dominance to increased SMR indicates a functional transition from hyperarousal to regulated alertness, an oscillatory pattern consistent with optimized cognitive readiness.

These findings align with broader neurofeedback literature demonstrating that targeted modulation of oscillatory activity can improve executive efficiency and stress resilience (Gruzelier, 2014), suggesting that cortical recovery may be actively trained rather than passively awaited.

Heart–Brain Coherence

The increase in HRV coherence (+0.44) in the experimental group reflects enhanced synchronization between cardiac oscillatory rhythms and prefrontal regulatory circuits. Heart–brain coherence has been linked to improved emotional regulation, attentional control, and psychophysiological resilience (Lehrer & Gevirtz, 2014; McCraty & Shaffer, 2015). In high-performance sport, reduced coherence following competition may contribute to irritability, decisional hesitation, or emotional reactivity.

The magnitude of coherence improvement observed in this study suggests rapid reintegration of cortical–autonomic communication pathways. This integrative marker is particularly relevant because it captures the coordination of systems rather than isolated recovery of single parameters. The relatively small increase in the control group underscores the additive effect of targeted biofeedback and vagal stimulation in accelerating integrative stabilization.

Subjective Cognitive Fatigue and Decisional Clarity

Subjective cognitive fatigue decreased by over five points in the experimental group, compared to less than one point in controls. Perceived mental fatigue has been shown to predict performance impairments in decision-making tasks even when physical capacity remains intact (Smith et al., 2018). The alignment between subjective fatigue reduction and objective EEG/HRV normalization supports the ecological validity of the findings.

Similarly, decisional clarity increased substantially in both self-report and staff evaluations. The staff-observed improvement of +1.89 points (on a 5-point scale) indicates that neurophysiological recovery translated into observable behavioral readiness. This is critical from a performance management perspective, as tactical reliability and emotional composure are often evaluated qualitatively by coaching staff. The congruence between physiological markers and external assessment strengthens the interpretation that the intervention produced functional recovery.

Taken together, the results suggest that the NEURORECOVERY ELITE protocol accelerated recovery across multiple interconnected systems: autonomic regulation, cortical oscillatory balance, heart–brain synchronization, and functional decisional readiness. The magnitude of change across parameters

supports the hypothesis that neurophysiological recovery can be reduced from approximately 72 hours to 24–36 hours when addressed through coordinated neurotechnological intervention.

Importantly, the improvements were not isolated to a single domain. Instead, vagal activation, oscillatory normalization, coherence enhancement, and subjective clarity progressed in parallel, consistent with systems-level recovery. This multidimensional convergence aligns with contemporary models of neurovisceral integration, which emphasize dynamic interplay between prefrontal cortical networks and autonomic regulation in shaping adaptive performance (Thayer & Lane, 2007). The findings therefore provide empirical support for a clinically structured, integrated model of post-match neuro-recovery, demonstrating that targeted modulation of cortical and autonomic systems yields measurable and behaviorally relevant benefits within the constraints of elite competition schedules.

6. Conclusions

The findings of the Neurorecovery Elite study support a central premise: sustained high-level performance in elite sport is not achievable without complete neurophysiological recovery. In congested competitive calendars, where matches occur every 48–72 hours, traditional recovery strategies that focus primarily on musculoskeletal and metabolic restitution are insufficient to ensure stable executive functioning, emotional balance, and tactical reliability. The present results demonstrate that cerebral and autonomic systems can be objectively assessed, actively modulated, and measurably accelerated through an integrated neurotechnological protocol.

From a practical standpoint, the implications for professional clubs are substantial. First, the study confirms that cognitive fatigue and autonomic dysregulation are measurable phenomena that persist beyond apparent physical recovery. Elevated High Beta activity, reduced RMSSD, and diminished heart–brain coherence are not abstract laboratory markers; they are functional predictors of decisional hesitation, emotional reactivity, and performance inconsistency. As previously suggested by Smith et al. (2018), cognitive fatigue alone can impair tactical decision-making even in the absence of muscular fatigue. Similarly, reduced vagal tone has been associated with impaired inhibitory control and stress regulation (Thayer & Lane, 2007). Second, the data indicate that neurophysiological recalibration can be accelerated to occur within 24–36 hours. This is strategically critical in leagues with high match density. By restoring parasympathetic dominance, normalizing cortical oscillatory activity, and re-establishing heart–brain coherence before the next competitive exposure, athletes re-enter play with preserved executive clarity rather than accumulated silent fatigue. The difference between partial and complete recovery may determine whether a player maintains tactical precision in the final minutes of a match or commits avoidable errors under pressure.

For coaching staff and performance directors, the integration of neurophysiological metrics offers a transition from assumption-based to evidence-based readiness assessment. Instead of relying solely on subjective impressions or physical load data, clubs can evaluate decisional readiness through objective HRV, EEG, and coherence indices. This approach reduces the risk of fielding athletes who appear physically available but remain neurocognitively compromised. In doing so, clubs mitigate unexplained performance drops often attributed to “mental lapses” or “lack of focus,” reframing them instead as measurable consequences of incomplete recovery.

The protocol’s non-invasive and time-efficient structure further supports its feasibility within existing microcycles. Applied during the first 24–48 hours post-match, the intervention does not interfere with tactical preparation or physical recovery sessions. Rather, it complements them by addressing a dimension traditionally neglected: cortical recalibration and autonomic stabilization. This parallel recovery model, where the body rests while the brain is actively restored, represents a paradigm shift in performance management.

At a strategic level, the implementation of structured neurorecovery programs may also contribute to injury prevention and long-term athlete sustainability. Persistent autonomic imbalance and cognitive fatigue have been associated with increased injury risk and burnout (Stanley et al., 2013). By maintaining neurovisceral integration and executive stability across congested schedules, clubs may reduce cumulative strain that silently erodes performance consistency over a season.

Beyond immediate competitive outcomes, the broader implication of NEURORECOVERY ELITE lies in redefining recovery as an active neurobiological process rather than passive rest. The study demonstrates that recovery quality, not merely recovery duration, determines functional readiness. Athletes in the experimental group did not simply exhibit improved biomarkers; they demonstrated observable improvements in decisional clarity acknowledged by both self-report and coaching staff. This convergence between physiological normalization and behavioral readiness underscores the translational value of the intervention.

Elite sport increasingly operates at margins where small differences determine outcomes. In this context, neurophysiological recovery represents not an optional enhancement but a competitive necessity. The evidence presented here supports the integration of structured neurorecovery protocols as a clinical standard within high-performance environments. As performance science continues to evolve, the capacity to measure, modulate, and optimize brain–autonomic dynamics may define the next frontier in sustainable elite achievement.

References

- Breit, S., Kupferberg, A., Rogler, G., & Hasler, G. (2018). Vagus nerve as modulator of the brain–gut axis in psychiatric and inflammatory disorders. *Frontiers in Psychiatry, 9*, 44. https://doi.org/10.3389/fpsyt.2018.00044
- Frangos, E., Ellrich, J., & Komisaruk, B. R. (2015). Non-invasive access to the vagus nerve central projections via electrical stimulation of the external ear: fMRI evidence in humans. *Brain Stimulation, 8*(3), 624–636. https://doi.org/10.1016/j.brs.2014.11.018
- Gruzelier, J. H. (2014). EEG-neurofeedback for optimising performance. I: A review of cognitive and affective outcome in healthy participants. *Neuroscience & Biobehavioral Reviews, 44*, 124–141. https://doi.org/10.1016/j.neubiorev.2013.09.015
- Hamblin, M. R. (2016). Shining light on the head: Photobiomodulation for brain disorders. *BBA Clinical, 6*, 113–124. https://doi.org/10.1016/j.bbacli.2016.09.002
- Iaccarino, H. F., Singer, A. C., Martorell, A. J., et al. (2016). Gamma frequency entrainment attenuates amyloid load and modifies microglia. *Nature, 540*(7632), 230–235. https://doi.org/10.1038/nature20587
- Lehrer, P. M., & Gevirtz, R. (2014). Heart rate variability biofeedback: How and why does it work? *Frontiers in Psychology, 5*, 756. https://doi.org/10.3389/fpsyg.2014.00756
- McCraty, R., & Shaffer, F. (2015). Heart rate variability: New perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. *Global Advances in Health and Medicine, 4*(1), 46–61. https://doi.org/10.7453/gahmj.2014.073
- Orozco Perez, H. D., Dumas, G., & Lehmann, A. (2020). Binaural beats through the auditory pathway: From brainstem to connectivity patterns. *eNeuro, 7*(2), ENEURO.0232-19.2020. https://doi.org/10.1523/ENEURO.0232-19.2020
- Plews, D. J., Laursen, P. B., Kilding, A. E., & Buchheit, M. (2013). Heart-rate variability and training-intensity distribution in elite rowers. *International Journal of Sports Physiology and Performance, 8*(6), 595–603. https://doi.org/10.1123/ijspp.8.6.595

- Raichle, M. E. (2015). The brain's default mode network. *Annual Review of Neuroscience, 38*, 433–447. https://doi.org/10.1146/annurev-neuro-071013-014030
- Reynolds, A. C., Paterson, J. L., & Ferguson, S. A. (2020). Exercise, inflammation, and cognitive function: Implications for recovery in sport. *Sports Medicine, 50*(4), 635–648. https://doi.org/10.1007/s40279-019-01215-6
- Schulz, K. F., Altman, D. G., & Moher, D. (2010). CONSORT 2010 statement: Updated guidelines for reporting parallel group randomized trials. *BMJ, 340*, c332. https://doi.org/10.1136/bmj.c332
- Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in Public Health, 5*, 258. https://doi.org/10.3389/fpubh.2017.00258
- Smith, M. R., Marcora, S. M., & Coutts, A. J. (2018). Mental fatigue impairs intermittent running performance. *Medicine & Science in Sports & Exercise, 50*(3), 538–545. https://doi.org/10.1249/MSS.0000000000001462
- Stanley, J., Peake, J. M., & Buchheit, M. (2013). Cardiac parasympathetic reactivation following exercise: Implications for training prescription. *Sports Medicine, 43*(12), 1259–1277. https://doi.org/10.1007/s40279-013-0083-4
- Thayer, J. F., & Lane, R. D. (2007). The role of vagal function in the risk for cardiovascular disease and mortality. *Biological Psychology, 74*(2), 224–242. https://doi.org/10.1016/j.biopsycho.2005.11.013
- Thun, E., Bjorvatn, B., Osland, T., et al. (2015). EEG indices of mental fatigue. *Biological Psychology, 109*, 122–131. https://doi.org/10.1016/j.biopsycho.2015.05.001