EMOTIACT: Regulation of Performance Anxiety and Consolidation of Emotional Control in High-Stakes Matches Through Integrated Brain-Based Training

Alina ROBU, Alina-Diana NEMES

Abstract

Performance anxiety represents a critical yet often underestimated determinant of failure in elite sport, particularly during high-stakes competitive moments. Contemporary neuroscience conceptualizes this phenomenon as a neurophysiological imbalance involving hyperactivation of the anterior cingulate cortex (Brodmann areas 24–32), dysregulation of the Salience Network, reduced vagally mediated heart rate variability (HRV), and impaired heart–brain coherence. The present study introduces and validates EMOTIACT, a randomized, longitudinal, controlled clinical protocol designed to recalibrate these mechanisms through an integrated multimodal intervention. Forty professional athletes were randomly assigned to an experimental group receiving the full EMOTIACT protocol (EEG neurofeedback, transcutaneous vagal nerve stimulation, HRV biofeedback, binaural auditory stimulation, and cerebral photobiomodulation) or to a control group receiving structured relaxation without active neurophysiological modulation. The intervention was delivered over six weeks (18 sessions), with assessments at baseline, mid-intervention, and post-intervention. Results demonstrated significant improvements in the experimental group compared with controls across all primary outcomes: reduced physiological stress (GP-8), increased vagal tone (RMSSD), decreased LF/HF ratio, reduced high-beta activation in BA24–32, enhanced HRV coherence, improved self-rated emotional control, and higher behavioral reliability in high-pressure matches. These findings support the hypothesis that performance anxiety reflects a modifiable neurophysiological state rather than a fixed psychological vulnerability. EMOTIACT provides a reproducible and scalable framework for integrating emotional regulation training into elite sport preparation. By restoring cortical–autonomic integration, the protocol enhances decisional clarity, emotional stability, and competitive reliability under pressure.

Keywords: *Performance anxiety; Neurofeedback; Heart rate variability (HRV); Anterior cingulate cortex; Heart–brain coherence; Vagal stimulation; Elite sport performance.*

1. Introduction and Scientific Rationale

In elite sport, performance failure cannot be explained solely by physical fatigue, biomechanical inefficiency, or tactical error. A substantial proportion of decisive mistakes occur in moments characterized by intense psychological pressure, when athletes must execute complex motor and cognitive actions under emotional load. In such contexts, performance anxiety emerges not as a transient emotional state, but as a multidimensional neurophysiological process that disrupts the functional integration of neural systems responsible for decision-making, attentional control, and emotional regulation. Contemporary sport neuroscience increasingly recognizes that performance anxiety reflects measurable alterations in brain–autonomic dynamics rather than a vague construct of "mental weakness" (Etkin et al., 2011; Thayer & Lane, 2007).

Performance anxiety in high-stakes competitions, such as semifinals, penalty shootouts, elimination rounds, or final minutes of tied matches, manifests as a psychophysiological overload state. At the cognitive level, it impairs working memory, narrows attentional focus in maladaptive ways, and biases appraisal toward threat anticipation (Eysenck et al., 2007). At the physiological level, it is characterized by sympathetic hyperactivation, reduced heart rate variability (HRV), elevated muscle tension, and heightened cortical arousal. Importantly, these changes are not epiphenomenal; they directly interfere with motor precision, timing, and tactical clarity. Empirical research on "choking under pressure" demonstrates that excessive self-monitoring and anxiety-driven cortical interference can degrade well-learned motor skills, particularly in expert performers (Beilock & Carr, 2001).

Neuroimaging and electrophysiological studies provide further insight into the neural substrates of this phenomenon. Athletes experiencing heightened performance anxiety show exaggerated activation of the Salience Network (SN), particularly in the anterior insula and the anterior cingulate cortex (ACC), regions crucial for detecting emotionally relevant stimuli and mobilizing adaptive responses (Seeley et al., 2007). While the SN is essential for prioritizing salient environmental cues, its hyperactivation under perceived threat can lead to overestimation of risk and excessive internal monitoring. In competitive sport, this may translate into tactical rigidity, impulsive reactions, or decisional paralysis in critical moments.

Within this framework, Brodmann areas 24 and 32, corresponding largely to the dorsal and rostral anterior cingulate cortex, play a central regulatory role. These regions integrate affective signals, monitor conflict, and mediate top–down control over limbic structures such as the amygdala (Bush et al., 2000; Phillips et al., 2008). Under anxiety-provoking conditions, the ACC often exhibits increased high-frequency beta activity, reflecting heightened arousal and cognitive-emotional conflict. Such hyperactivation is associated with anticipatory worry, increased error sensitivity, and impaired flexibility in behavioral responses (Etkin et al., 2011). In athletes, this neural state may manifest as disproportionate emotional reactivity after mistakes, excessive fear of failure, or hesitation in decision-making sequences that normally require fluid execution. In parallel, dysregulation within the Default Mode Network (DMN), the network implicated in self-referential processing and internal narrative construction, can further exacerbate performance breakdown. Excessive DMN activation during task execution has been linked to rumination and maladaptive self-focus (Raichle, 2015). In competitive settings, such hyperactivation may generate intrusive thoughts about consequences, reputation, or past errors, diverting cognitive resources away from real-time tactical processing. The imbalance between the SN, DMN, and Executive Control Network (ECN) thus becomes a critical determinant of performance stability under pressure.

Autonomic markers provide an additional window into this imbalance. According to the neurovisceral integration model, HRV, particularly indices such as RMSSD, reflects the functional integrity of prefrontal–subcortical inhibitory circuits involved in emotion regulation (Thayer & Lane, 2000, 2007). Reduced HRV is consistently associated with anxiety, diminished emotional flexibility, and impaired stress adaptation. In athletes, low HRV prior to competition correlates with heightened anticipatory stress and poorer emotional recovery after errors (Laborde et al., 2017). A high LF/HF ratio further indicates sympathetic dominance, characteristic of hypervigilance and physiological rigidity. Importantly, these neural and autonomic disturbances are measurable through quantitative EEG (qEEG) and HRV assessment. Elevated high-beta activity in anterior cingulate regions, decreased alpha coherence, and reduced vagally mediated HRV constitute objective biomarkers of performance anxiety. Such findings challenge the outdated notion that emotional instability in sport is purely psychological or motivational. Instead, they suggest a modifiable neurophysiological profile that can be targeted through structured brain-based interventions.

Recent advances in applied neurotechnology support the feasibility of such interventions. EEG neurofeedback has demonstrated efficacy in modulating cortical oscillatory patterns and enhancing self-regulation capacities (Gruzelier, 2014; Ros et al., 2014). HRV biofeedback improves autonomic flexibility and emotional resilience (Lehrer & Gevirtz, 2014). Transcutaneous vagal nerve stimulation has been shown to influence limbic–prefrontal connectivity and reduce stress reactivity (Breit et al., 2018; Frangos et al., 2015). Moreover, emerging evidence suggests that photobiomodulation and rhythmic auditory stimulation can modulate cortical synchronization and promote neuroplastic adaptation (Hamblin, 2016; Orozco Perez et al., 2020).

Taken together, this body of literature converges on a critical insight: performance anxiety in elite athletes reflects a dysregulated interaction between salience detection systems, prefrontal regulatory circuits, and autonomic balance. This dysregulation compromises the athlete's ability to remain cognitively flexible, emotionally stable, and tactically precise under pressure. However, because these mechanisms are measurable and neuroplastic, they are also trainable. The EMOTIACT study is grounded in this integrative neuroscientific perspective. Rather than conceptualizing anxiety as a subjective vulnerability, it frames performance anxiety as a modifiable neurophysiological state arising from hyperactivation of the anterior cingulate cortex, salience network imbalance, and reduced vagal tone. The scientific rationale for EMOTIACT is therefore to intervene directly at the level of cortical oscillations, autonomic regulation, and heart–brain coherence, with the aim of restoring functional integration between emotion, physiology, and decision-making. In doing so, the study seeks to demonstrate that emotional stability in elite sport can be systematically trained and objectively validated, just as strength or endurance can be developed through structured physical conditioning.

2. Study Objectives

The EMOTIACT study was designed to develop and clinically validate an integrated neurophysiological intervention protocol aimed at reducing performance anxiety and enhancing emotional control in elite athletes exposed to high-pressure competitive contexts. Grounded in contemporary models of neurovisceral integration and large-scale brain network dynamics (Thayer & Lane, 2007; Seeley et al., 2007), the study conceptualizes performance anxiety as a measurable dysregulation of anterior cingulate activity, autonomic imbalance, and impaired heart–brain coherence.

The general objective of the study is to evaluate the efficacy of a multimodal intervention, combining EEG neurofeedback targeting Brodmann areas 24 and 32, transcutaneous vagal nerve stimulation, heart rate variability (HRV) biofeedback for heart–brain coherence, binaural auditory stimulation, and cerebral photobiomodulation, in reducing neurophysiological and behavioral markers of performance anxiety. The primary endpoints include reductions in physiological stress (GP-8 scores and LF/HF ratio), increases in vagally mediated HRV (RMSSD), normalization of high-beta hyperactivation in the anterior cingulate cortex, and improvements in observable emotional stability during competitive play.

Specific objectives include:

1. To decrease physiological stress levels and sympathetic hyperactivation, as indexed by reductions in GP-8 stress scores and LF/HF ratio, alongside increases in RMSSD, reflecting improved vagal tone and emotional flexibility (Shaffer & Ginsberg, 2017; Lehrer & Gevirtz, 2014).
2. To reduce hyperreactive cortical activation in Brodmann areas 24–32 by lowering excessive high-beta activity and enhancing alpha/SMR coherence through targeted EEG neurofeedback, thereby improving conflict monitoring and emotional inhibition capacities (Bush et al., 2000; Etkin et al., 2011).

3. To increase heart–brain coherence during stress exposure, operationalized through HRV coherence indices, facilitating synchronized interaction between prefrontal regulatory systems and autonomic feedback mechanisms (McCraty & Shaffer, 2015).
4. To stabilize emotional responses during high-pressure decision-making sequences, such as penalty situations or final-match phases, by aligning neurophysiological regulation with observable tactical behavior.
5. To enhance behavioral reliability under competitive stress, as evaluated by structured coach ratings and semi-structured athlete self-reports of emotional control and decisional clarity.

Ultimately, the study seeks to validate a reproducible, scalable, and clinically grounded protocol capable of transforming performance anxiety from a destabilizing factor into a trainable domain of neurophysiological optimization.

3. Study Design

The EMOTIACT study was conceived as a rigorous clinical investigation reflecting both the complexity of performance anxiety and the translational demands of elite sport. Its methodological architecture was structured to ensure internal validity, ecological applicability within professional competitive settings, and reproducibility across clubs and training environments. The design integrates strict experimental control with real-world implementation during an active competitive season, thereby bridging laboratory-based neuroscience and applied sport performance. EMOTIACT is a randomized, longitudinal, controlled clinical trial with two parallel arms, conducted in a multicentric framework in collaboration with professional sports clubs. This design was selected to isolate the specific contribution of the integrated neurophysiological intervention while minimizing confounding influences such as spontaneous improvement, motivational fluctuations, or placebo effects. Participants were randomly assigned to one of two groups:

- **Experimental group**: athletes receiving the full EMOTIACT multimodal intervention.
- **Control group**: athletes undergoing identical assessments and structured relaxation sessions without active EEG neurofeedback, physiological biofeedback, or neurostimulation components.

The randomized controlled structure aligns with international methodological standards for clinical research (Schulz et al., 2010) and is particularly suited for evaluating neurotechnological interventions in applied performance contexts. By maintaining equivalent monitoring and engagement across both groups, the design allows for a robust comparison of neurophysiological, psychological, and behavioral outcomes attributable specifically to the active protocol.

The intervention was delivered over a six-week period, embedded within a real competitive microcycle. This timeframe was chosen based on evidence indicating that targeted neuroplastic adaptations in fronto-limbic and autonomic circuits typically emerge after 10–15 structured training sessions (Enriquez-Geppert et al., 2017; Ros et al., 2014). The temporal structure was as follows:

- **Total duration**: 6 weeks
- **Session frequency**: 3 sessions per week
- **Total sessions**: 18
- **Session duration**: 60 minutes

- **Assessment points**:
  - T0 – baseline
  - T1 – mid-intervention (3 weeks)
  - T2 – post-intervention (6 weeks)

This schedule allowed sufficient intensity to induce measurable neurophysiological change while remaining compatible with athletes' physical training and competition calendars.

Study Population

The study included a total of 40 professional athletes, distributed equally between the experimental and control groups (20 per group). Participants were recruited from competitive teams in national and international leagues, with a particular focus on athletes frequently exposed to high-pressure decision-making roles (e.g., strikers, goalkeepers, playmakers).

Inclusion criteria were defined to ensure the presence of measurable performance anxiety and physiological dysregulation:

- Age between 18 and 35 years.
- Active participation in an ongoing competitive season.
- GP-8 stress score > 12 and/or RMSSD < 35 ms at screening.
- No diagnosed neurological, psychiatric, or cardiac disorders.

Exclusion criteria included:

- Current use of psychotropic medication.
- Concurrent participation in intensive cognitive therapy or neurostimulation programs.
- Significant absenteeism from scheduled sessions.

These criteria ensured a homogeneous sample characterized by functional performance anxiety without confounding clinical pathologies.

Structure of the Intervention

Each session followed a structured, integrative format designed to sequentially target cortical hyperactivation, autonomic imbalance, and heart–brain synchronization. The components were delivered in a standardized order to promote cumulative and synergistic effects.

Table 1. Structure of a Standard EMOTIACT Intervention Session

Component	Duration	Primary Functional Objective
EEG Neurofeedback (BA24–32)	20 min	Reduction of anterior cingulate hyperactivation; stabilization of emotional reactivity
Transcutaneous Vagal Stimulation (nVNS)	10 min	Parasympathetic activation; reduction of autonomic stress
Heart–Brain Coherence (HRV Biofeedback)	15 min	Synchronization of cardiac and cortical rhythms
Binaural Audio Therapy	10 min	Rhythmic cortical calming; limbic inhibition

Component	Duration	Primary Functional Objective
Cerebral Photobiomodulation	5 min	Modulation of fronto-limbic activation; vascular and mitochondrial support

The progression from active cortical modulation (EEG) to autonomic recalibration (nVNS and HRV) and consolidation (binaural stimulation and photobiomodulation) was intended to optimize neurophysiological integration within each session.

Participants in the control group underwent:

- Identical assessment procedures (qEEG, HRV, GP-8, interviews, behavioral scoring).
- Structured guided audio relaxation sessions without active neurofeedback or physiological modulation.

No vagal stimulation, HRV biofeedback training, or photobiomodulation was administered. This approach preserved participant engagement while controlling for expectancy and attention effects.

Assessments and Standardized Instruments

Evaluations were conducted at T0, T1, and T2 using a multimodal assessment battery capturing neural, autonomic, psychological, and behavioral domains.

The following measures were included:

1. **Quantitative EEG (19-channel system)**
    - Spectral power and coherence in BA24–32
    - High-beta (22–30 Hz) activation
    - Alpha and SMR coherence in fronto-limbic circuits
2. **Comprehensive HRV analysis**
    - RMSSD
    - SDNN
    - LF/HF ratio
3. **GP-8 Stress Profile**
    - Global score and subdimensions of physiological stress
4. **Heart–brain coherence index**
    - Derived from HRV biofeedback software
5. **Semi-structured emotional control interviews**
6. **Behavioral reliability score**
    - Rated by technical staff during high-pressure matches

This multidimensional system ensured that outcome evaluation extended beyond subjective reporting to include objective physiological markers and observable competitive behavior. All interventions were conducted in certified EEG cabins equipped with CE-compliant medical devices. Clinical-grade neurofeedback software, validated HRV systems, approved vagal stimulation devices, and photobiomodulation equipment

were employed. The professional team consisted of licensed clinical psychologists with specialization in applied neuroscience and accredited EEG technicians. Procedural fidelity was ensured through standardized protocols and session documentation.

The EMOTIACT design was constructed with a dual objective: first, to demonstrate that performance anxiety can be modulated at cortical, autonomic, and behavioral levels through structured neurophysiological training; and second, to offer professional sports organizations a reproducible, evidence-based framework for systematically enhancing emotional stability as a component of elite performance.

4. Intervention Structure

The intervention was conceptualized as a clinically integrated neuroregulatory training protocol rather than a collection of isolated techniques. Its structure reflects a systems-level approach, targeting the reciprocal interactions between cortical emotional regulation networks, autonomic balance, and heart–brain synchronization. Each session was organized to progressively recalibrate hyperreactive salience processing, strengthen prefrontal inhibitory control, and restore vagally mediated flexibility, thereby enabling athletes to maintain decisional clarity under competitive pressure. The intervention consisted of five synergistic components delivered in a standardized sequence during each 60-minute session.

4.1 Targeted EEG Neurofeedback (20 minutes)

The first phase of each session focused on EEG neurofeedback directed at Brodmann areas 24 and 32 within the anterior cingulate cortex. These regions play a central role in emotional conflict monitoring, impulse control, and integration of affective signals with executive decision-making (Bush et al., 2000; Etkin et al., 2011). The neurofeedback protocol aimed to reduce excessive high-beta activity (22–30 Hz), frequently associated with hyperarousal, anticipatory anxiety, and rigid cognitive control, while enhancing alpha and sensorimotor rhythm (SMR) activity linked to functional calmness and controlled attention. By providing real-time feedback, athletes were trained to recognize and modulate their cortical activation patterns, facilitating adaptive self-regulation. In performance terms, this component targeted the ability to experience competitive intensity without losing cognitive flexibility or emotional balance.

4.2 Transcutaneous Vagal Nerve Stimulation (10 minutes)

The second phase involved auricular transcutaneous vagal nerve stimulation (nVNS), delivered to enhance parasympathetic tone. The vagus nerve is anatomically and functionally connected to the anterior insula and anterior cingulate cortex, regions implicated in emotional salience and stress regulation (Breit et al., 2018). By stimulating afferent vagal pathways, the intervention sought to attenuate sympathetic dominance, reduce physiological hyperreactivity, and increase heart rate variability (HRV). Experimental research demonstrates that non-invasive vagal stimulation can modulate limbic–prefrontal connectivity and decrease stress responsivity (Frangos et al., 2015). Within the athletic context, this component functioned as a physiological anchor, training the athlete's nervous system to exit "fight-or-flight" states more rapidly during high-pressure scenarios.

4.3 Heart–Brain Coherence Training via HRV Biofeedback (15 minutes)

The third component consisted of HRV biofeedback aimed at enhancing heart–brain coherence. Guided breathing at resonance frequency was used to synchronize respiratory rhythms with cardiac oscillations, thereby maximizing vagally mediated HRV indices such as RMSSD. According to the neurovisceral integration model (Thayer & Lane, 2007), higher HRV reflects stronger functional connectivity between prefrontal regulatory circuits and subcortical emotional systems. Increased HRV coherence has been associated

with improved emotional stability, attentional control, and adaptive decision-making under stress (McCraty & Shaffer, 2015; Lehrer & Gevirtz, 2014). In practical terms, this phase trained athletes to cultivate a psychophysiological state characterized by calm alertness, maintaining access to emotional signals without being overwhelmed by them.

4.4 Binaural Auditory Stimulation (10 minutes)

The fourth phase incorporated binaural audio stimulation in theta (4–7 Hz) and alpha (8–12 Hz) frequency ranges. Binaural beats can promote cortical synchronization and reduce excessive cognitive noise by inducing rhythmic entrainment effects (Orozco Perez et al., 2020). The purpose of this component was to consolidate the regulatory gains achieved through neurofeedback and HRV training by facilitating a receptive yet stable cortical state. Theta–alpha entrainment is associated with relaxed attentional focus and decreased rumination, which are essential for mitigating maladaptive self-referential processing during competition (Wahbeh et al., 2007). For athletes, this phase supported the development of a consistent pre-performance mental state, calm, present-focused, and emotionally regulated.

4.5 Cerebral Photobiomodulation (5 minutes)

The final phase involved low-level light therapy applied to frontal regions using pulsed frequencies at 10 Hz (alpha range) and 40 Hz (gamma range). Photobiomodulation has been associated with increased mitochondrial activity, improved cerebral blood flow, and modulation of cortical oscillatory dynamics (Hamblin, 2016; Iaccarino et al., 2016). Within the EMOTIACT framework, photobiomodulation was intended to enhance neurovascular support in fronto-limbic regions and reinforce adaptive oscillatory synchronization achieved earlier in the session. The 10 Hz stimulation, in particular, was selected to promote alpha coherence in prefrontal–cingulate networks, facilitating a state of controlled relaxation with preserved cognitive readiness.

Collectively, the EMOTIACT intervention represents a structured neurophysiological training ecosystem. EEG neurofeedback addresses cortical hyperreactivity, vagal stimulation recalibrates autonomic balance, HRV biofeedback synchronizes heart–brain communication, binaural stimulation promotes rhythmic stabilization, and photobiomodulation supports metabolic and oscillatory optimization.Rather than merely reducing anxiety symptoms, the protocol aims to recalibrate the functional architecture underlying emotional regulation, enabling athletes to perform with composure, clarity, and decisional reliability when competitive pressure is at its peak.

5. Results

The intervention produced robust and clinically meaningful changes across all primary neurophysiological, psychological, and behavioral markers when compared with the control group, as observed in table 2. The magnitude and consistency of the observed effects support the hypothesis that performance anxiety in elite athletes can be modulated through an integrated neuro-autonomic protocol targeting anterior cingulate hyperactivation, autonomic imbalance, and heart–brain desynchronization.

Table 2. Pre–Post Changes (Δ T2–T0) in Experimental vs. Control Group

Parameter	Experimental Group (Mean ± SD)	Control Group (Mean ± SD)
Δ GP-8 Total Stress Score	–16.77 ± 2.83	–4.23 ± 2.61
Δ RMSSD (ms)	+14.45 ± 3.76	+2.86 ± 0.87
Δ LF/HF Ratio	–1.08 ± 0.43	–0.28 ± 0.30
Δ EEG High Beta (BA24–32)	–8.67 ± 1.58	–1.77 ± 1.14
Δ HRV Coherence Index	+0.42 ± 0.09	+0.11 ± 0.07
Δ Self-Rated Emotional Control	+2.18 ± 0.64	+0.63 ± 0.35
Δ Competitive Behavioral Reliability Score	+1.98 ± 0.47	+0.45 ± 0.36

These findings reflect substantial divergence between groups across all outcome domains, indicating that the intervention exerted effects not only at the subjective level but also across objective autonomic and cortical markers.

Reduction in Physiological Stress (GP-8)

The experimental group demonstrated a mean reduction of 16.77 points on the GP-8 Stress Profile, compared with a modest 4.23-point reduction in the control group. This magnitude of change suggests a systemic recalibration of psychophysiological stress. The GP-8 captures dimensions such as muscular tension, sleep disturbance, irritability, and decisional blockage, symptoms typically associated with chronic sympathetic overactivation. The reduction observed is consistent with findings showing that structured biofeedback and neurofeedback interventions can significantly reduce somatic and affective stress indicators in high-demand populations (Lehrer & Gevirtz, 2014; Ros et al., 2014). From a neurobiological standpoint, decreased perceived stress parallels improved inhibitory control within anterior cingulate circuits and enhanced vagal modulation, both of which are critical for adaptive emotion regulation (Thayer & Lane, 2007).

Increase in Vagal Tone (RMSSD)

RMSSD increased by 14.45 ms in the experimental group, compared with 2.86 ms in controls. RMSSD is a well-established index of vagally mediated HRV and reflects parasympathetic influence over cardiac function (Shaffer & Ginsberg, 2017). Elevated RMSSD values indicate greater emotional flexibility, faster recovery following stress, and improved resilience. The magnitude of this increase suggests not merely transient relaxation, but structural improvement in autonomic regulation capacity. Higher HRV has been consistently associated with better performance under stress and improved attentional control in athletes (Laborde et al., 2017). According to the neurovisceral integration model, enhanced HRV reflects strengthened functional connectivity between the medial prefrontal cortex and limbic regions, facilitating adaptive emotional inhibition (Thayer & Lane, 2000).

Decrease in Sympathetic Dominance (LF/HF Ratio)

The LF/HF ratio decreased by 1.08 points in the experimental group, indicating a shift away from sympathetic predominance toward balanced autonomic regulation. In contrast, the control group exhibited only a modest reduction (–0.28). Elevated LF/HF ratios are commonly observed in individuals with anxiety-related hypervigilance and anticipatory stress (Thayer & Lane, 2007). The reduction observed here suggests that the combined vagal stimulation and HRV coherence training effectively attenuated chronic physiological hyperarousal. Clinically, this translates into reduced anticipatory tension before decisive actions, diminished somatic rigidity, and expanded emotional tolerance windows during competition.

Reduction of High-Beta Hyperactivation in BA24–32

Quantitative EEG revealed a significant decrease in high-beta activity within Brodmann areas 24–32 (–8.67 units in the experimental group vs. –1.77 in controls). High-beta oscillations in anterior cingulate regions have been associated with heightened emotional vigilance, worry, and rigid cognitive control (Bush et al., 2000; Etkin et al., 2011). The observed reduction indicates normalization of salience network hyperreactivity. Excessive ACC activation under pressure has been linked to over-monitoring of potential errors and fear of negative evaluation, core components of performance anxiety. By reducing this hyperactivation, the intervention appears to have restored functional flexibility in fronto-cingulate circuits, enabling athletes to process emotionally salient stimuli without entering maladaptive threat states. Comparable reductions in maladaptive beta activity have been reported following neurofeedback interventions targeting emotional regulation networks (Gruzelier, 2014; Ros et al., 2014), supporting the mechanistic plausibility of the observed effects.

Increase in Heart–Brain Coherence

The HRV coherence index increased by 0.42 in the experimental group, compared with 0.11 in controls. Elevated coherence reflects synchronization between respiratory rhythms, cardiac oscillations, and cortical regulatory systems (McCraty & Shaffer, 2015). This state has been associated with improved attentional stability and reduced emotional volatility. Heart–brain coherence may represent the functional integration of cortical and autonomic processes necessary for stable decision-making in dynamic environments. Increased coherence suggests that emotional signals are being integrated rather than suppressed or amplified, permitting adaptive tactical execution during stress.

Improvement in Self-Perceived Emotional Control

Athletes in the experimental group reported a mean increase of 2.18 points (on a 1–7 scale) in emotional control, compared with 0.63 in controls. Self-efficacy in emotional regulation has been shown to predict behavioral consistency and resilience in high-pressure environments (Kober et al., 2015). The alignment between subjective improvements and objective HRV and EEG changes strengthens the ecological validity of the findings. Athletes did not merely demonstrate physiological recalibration; they experienced enhanced internal control over emotional states.

Increase in Competitive Behavioral Reliability

Perhaps most clinically relevant, the behavioral reliability score assigned by coaching staff increased by 1.98 points in the experimental group (vs. 0.45 in controls). This metric captured observable stability during decisive game sequences, including response after errors and consistency in tactical execution under pressure. Behavioral stability under stress is often compromised when anterior cingulate hyperactivation and low HRV coexist (Thayer & Lane, 2007). The convergence of cortical, autonomic, and behavioral improvements suggests that the intervention achieved systemic integration rather than isolated physiological change.

Collectively, the results demonstrate a coherent pattern: reductions in cortical hyperreactivity, restoration of autonomic flexibility, enhanced heart–brain synchronization, and parallel improvements in perceived and observed emotional control. The findings support the central hypothesis of EMOTIACT: performance anxiety in elite sport reflects a modifiable neurophysiological imbalance. Through targeted multimodal intervention, it is possible to recalibrate this imbalance, transforming emotional instability into decisional reliability in high-stakes competitive contexts.

6. Conclusions

The findings of the EMOTIACT study demonstrate that performance anxiety in elite athletes is not merely a subjective psychological state, but a measurable neurophysiological imbalance involving anterior cingulate hyperactivation, autonomic dysregulation, and impaired heart–brain synchronization. Through a structured multimodal intervention integrating EEG neurofeedback, vagal stimulation, HRV biofeedback, binaural auditory entrainment, and cerebral photobiomodulation, this imbalance can be systematically recalibrated.

From a neuroscientific perspective, the results reinforce contemporary models of emotion regulation that emphasize the functional integration of salience detection systems, prefrontal inhibitory circuits, and autonomic feedback loops (Etkin et al., 2011; Thayer & Lane, 2007). The significant reduction in high-beta activation within Brodmann areas 24–32 suggests normalization of anterior cingulate overreactivity, a region central to conflict monitoring and emotional integration. Concurrent increases in RMSSD and heart–brain coherence indices indicate strengthened vagally mediated control and improved neurovisceral integration. Together, these findings provide converging evidence that emotional regulation in sport can be enhanced through targeted modulation of identifiable neural and physiological mechanisms.

Clinically, EMOTIACT validates the premise that performance anxiety is trainable. Rather than attempting to suppress emotional arousal, the protocol fosters adaptive regulation, allowing athletes to experience competitive intensity without cognitive disorganization or impulsive reactivity. The observed improvements in behavioral reliability ratings further confirm that neurophysiological recalibration translates into tangible performance stability during decisive match situations. This integration of objective biomarkers with observable competitive behavior represents a critical advance in applied sport neuroscience.

For professional clubs, the implications are strategic. Performance breakdowns in high-stakes moments often determine season outcomes, financial returns, and career trajectories. Traditional psychological support models typically rely on motivational strategies or reactive interventions implemented after visible emotional collapse. EMOTIACT proposes a proactive alternative: systematic emotional regulation training embedded within the weekly microcycle, comparable in structure and rigor to strength or endurance conditioning. The strategic benefits for clubs include:

- **Increased decisional reliability under pressure**, reducing tactical errors driven by emotional dysregulation.
- **Enhanced recovery after mistakes**, minimizing cascades of impulsive or avoidant behavior.
- **Objective monitoring of emotional readiness**, using HRV and EEG metrics to guide individualized support.
- **Rehabilitation of emotionally vulnerable athletes**, without stigmatization or exclusion from competitive roles.
- **Creation of a coherent team culture**, in which physiological and emotional self-regulation are normalized as performance competencies.

Importantly, the protocol is reproducible, scalable, and compatible with ongoing physical training demands. By integrating neurophysiological optimization into performance strategy, clubs can reduce uncertainty regarding athletes' emotional stability during finals, elimination rounds, or decisive penalty sequences. Emotional regulation becomes not an unpredictable trait, but a trained and monitored capability.

The broader contribution of EMOTIACT lies in its reframing of competitive resilience. Emotional intensity is inherent to elite sport; success does not depend on eliminating it, but on regulating it efficiently.

Athletes who can maintain cortical stability and autonomic flexibility under stress are more likely to execute complex tactical decisions with clarity and composure. In this sense, heart–brain coherence is not metaphorical, it represents a measurable state of integrated regulation that supports high-level performance.

In conclusion, EMOTIACT provides empirical evidence that the neurophysiological substrates of performance anxiety can be modified through structured intervention. The protocol transforms emotional instability from a hidden vulnerability into a trainable domain of excellence. By aligning cortical regulation, autonomic balance, and behavioral expression, the study outlines a new paradigm in elite preparation, one in which competitive success is supported not only by physical strength and tactical intelligence, but also by coherent, adaptable emotional control when pressure is at its peak.

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