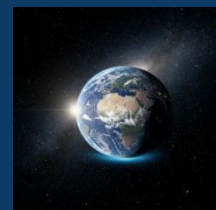




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THE NEUROBIOLOGY OF "CHOKING UNDER PRESSURE": MECHANISMS OF ANXIETY IMPAIRING WORKING MEMORY AND MOTOR AUTOMATISM IN ATHLETES

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ABSTRACT

Background. "Choking under pressure" — the paradoxical collapse of skilled performance when the desire to succeed is greatest — is central to sport psychology and cognitive neuroscience. Despite advances in fMRI, EEG, and brain stimulation, neural mechanisms by which incentive-induced arousal and anxiety undermine well-learned performance remain incompletely understood.

Aim: To synthesise neurobiological evidence on how performance anxiety impairs working memory and motor automatism in athletes, with focus on prefrontal cortex, ventral striatum, anterior cingulate, dopaminergic/noradrenergic systems, error-monitoring networks, and evidence-based interventions.

Material and methods: Narrative review of neuroimaging, EEG, and brain stimulation studies; meta-analyses; and systematic reviews published 2010–2025 from PubMed, SPORTDiscus, and PsycINFO.

Results: Three neural systems underlie choking: exaggerated ventral striatal incentive encoding producing hyperarousal; reduced prefrontal top-down control disrupting motor automaticity; and aberrant dACC activity impairing sequential performance — causally confirmed via rTMS. The ERN is potentiated in high-anxiety athletes during evaluation, supporting an explicit monitoring account; WMC predicts attentional control under anxiety, and WM training produces far-transfer benefits. Implicit learning, analogy learning, and external focus reduce declarative movement reliance and reinvestment vulnerability; cognitive reappraisal abolishes the sympathetic choking signature. Mindfulness, neurofeedback, and pre-performance routines offer further mechanism-specific avenues for prevention.

Conclusions: Choking is a neurobiologically heterogeneous phenomenon. Effective prevention requires mechanism-matched strategies spanning implicit learning, cognitive reappraisal, WM training, and psychological skills interventions. Multi-modal, ecologically valid neuroimaging research remains a priority.

Keywords: choking under pressure; performance anxiety; working memory; motor automatism; prefrontal cortex; anterior cingulate cortex; implicit learning; error-related negativity; neurofeedback

1. Introduction

The phenomenon of "choking under pressure" — defined as the paradoxical failure of skilled performance in high-stakes conditions when motivation to succeed is maximal and the threat of failure is acutely salient — has fascinated sport scientists, cognitive psychologists, and neuroscientists since its formal characterization in the 1980s [1]. Across competitive sport, high-stakes examinations, professional performance, and high-consequence occupations such as surgery and aviation, the observation is recurrent: individuals with demonstrably high skill levels can produce outcomes far below their established baseline when the consequences of failure are greatest [1][2]. The phenomenon contradicts the intuitive and economically canonical expectation that greater incentives should produce greater effort and superior performance [1].

The ability to perform under heightened pressure is one of the strongest discriminators between those who achieve success in competition and those who do not [2]. Choking belongs to a family of "paradoxical performances" — the occurrence of inferior performance despite striving and incentives for superior performance — that also includes the yips, a partly

overlapping psycho-neuromuscular impediment [2]. Athletes who experience choking frequently lose confidence, develop a fear of competition, and impose costs on their teams and sporting organizations, making choking a problem of considerable practical as well as scientific importance [3].

Behavioral research has established three major theoretical frameworks [1]. Distraction theories propose that high-pressure conditions draw working memory resources away from the task toward internal preoccupations — worries about failure, social-evaluative concerns, and outcome consequences — reducing the cognitive resources available for skilled performance [1][4]. Explicit monitoring theories argue that pressure raises self-consciousness and anxiety, prompting performers to micromanage the step-by-step execution of skills that would otherwise proceed automatically, thereby disrupting the fluid, proceduralized control characteristic of expertise [1][5]. The over-arousal or over-motivation framework draws on the Yerkes–Dodson law, holding that the excessive arousal generated by very high incentives degrades performance [1]. These accounts are not mutually exclusive, and mounting neuroimaging evidence suggests that different mechanisms operate in different task and performer contexts [1][6].

The neural underpinnings of choking remained largely unexplored until the application of functional magnetic resonance imaging (fMRI) to performance under pressure [1]. Initial studies implicated mesolimbic reward circuits — the ventral tegmental area, striatum, and medial prefrontal cortex — in encoding motivational salience during high-stakes performance [1]. Subsequent work extended these findings to the dorsal anterior cingulate cortex in anxiety-induced disruption of sequential motor execution [6], the dorsolateral prefrontal cortex in the regulatory failure associated with choking [4], and a distributed network of stress-responsive regions in the reorganization of brain function under acute psychological pressure [1][7].

Complementary neurophysiological approaches using electroencephalography have documented oscillatory signatures associated with expert performance that may serve as biomarkers of choking vulnerability [8]. The error-related negativity, an event-related brain potential reflecting rapid frontocentral monitoring of performance errors, is potentiated in athletes with high sport anxiety specifically under performance evaluation, providing neural evidence for explicit monitoring as a mechanism of choking [5]. Psychophysiological investigations have established that skin conductance responses during high-stakes motor performance are exaggerated on failed trials, providing the first direct experimental evidence that sympathetic hyperarousal accompanies choking rather than merely preceding it [9]. This review integrates this evidence to construct an account of the neural vulnerability to choking and of the neurobiological basis of interventions designed to protect performance under pressure.

2. Definition and Phenomenology of Choking Under Pressure

Choking under pressure is operationally defined as a performance decrement that occurs specifically under high-stakes conditions in individuals who are motivated to perform well and whose skill level — established through prior behavior under lower-pressure circumstances — would predict superior outcomes [1]. The defining criterion is that the decrement is specifically attributable to the psychological pressure of the high-stakes situation rather than to any deficit in skill, preparation, or physical condition [1]. This specificity makes choking both scientifically tractable — it can be experimentally induced through performance-contingent rewards, audience presence, and video-recording — and clinically meaningful, identifying a modifiable psychological vulnerability rather than an immutable skill limitation [5].

The phenomenology of choking spans a wide range of performance domains [1]. Sensorimotor examples include the collapse of golf putting in tournament conditions, the deterioration of basketball free-throw shooting in decisive moments, and the breakdown of precise coordination in championship-level individual sport [1]. Cognitive tasks such as mathematical problem-solving are equally susceptible, with high-stakes examination conditions producing decrements in individuals whose baseline ability would predict higher scores [1]. The breadth of choking across domains — from fine motor precision to abstract cognition — indicates that the underlying mechanisms operate at a level of generality transcending any particular skill type [1].

The historical and cultural salience of choking is illustrated by high-profile failures in elite competition, the most cited being an Olympic rifle shooter who, on two occasions, was a single shot from a gold medal yet produced a score far below his established standard at the decisive moment [1]. Such cases exemplify the central paradox: a maximally motivated and demonstrably capable athlete can nonetheless fail precisely when success matters most [1]. Survey data document the prevalence of these phenomena: in a study of 155 experienced golfers and archers, the prevalence of choking and yips reached 67.7% and 39.4% respectively, underscoring that paradoxical performance is not a rare aberration but a common feature of competitive sport [2].

Several features of the phenomenon have particularly guided the selection of neural targets for investigation [1]. First, the relationship between choking and incentive magnitude is non-monotonic: moderate incentives facilitate performance while very high incentives can degrade it, producing a function reminiscent of the inverted-U relationship posited by the Yerkes–Dodson law [1]. Second, well-learned, highly automatized skills appear especially vulnerable to disruption by conscious interference, suggesting a specific mechanism by which expertise itself creates vulnerability [1][10]. Third, susceptibility varies markedly across individuals, with characteristics including trait anxiety, fear of negative evaluation, self-consciousness, perfectionism, and impulsivity predicting differential vulnerability to performance decrements [2][11]. The phenomenon thus sits at the intersection of motivation, attention, emotion, and motor control, which is why its neural investigation has drawn on circuits spanning the mesolimbic reward system, the prefrontal executive networks, the cingulate-based action-monitoring system, and the subcortical stress-responsive structures [1][6].

3. Theoretical Models of Choking: Distraction, Explicit Monitoring, and Over-Arousal

The three dominant theoretical accounts generate distinct predictions about the neural systems differentially engaged during performance failure under pressure [1]. Distraction theories, grounded in working memory models and attentional control theory, propose that pressure creates a dual-task situation in which the primary task competes with intrusive task-irrelevant cognition for limited working memory capacity [1]. Because high-capacity working memory individuals rely more heavily on these resources for superior performance, they can be especially vulnerable to distraction-induced choking [1][12]. The distraction account predicts reduced engagement of goal-directed attentional control systems — specifically prefrontal-to-task-network connectivity — during choking [4].

Explicit monitoring theories propose an opposing mechanism: pressure increases attention to skill execution by heightening monitoring of the procedural details of well-learned motor skills [1]. Automatized skills are executed efficiently through procedural memory systems operating below conscious awareness; when anxiety and self-consciousness prompt explicit, step-by-step monitoring of these routines, the continuous flow of motor execution is fractured into isolated, effortful components resembling novice performance [1][5]. This theory predicts enhanced activity in executive-control regions governing deliberate monitoring of

action during choking, particularly in evaluative contexts [5]. Critically, it predicts that skill acquired through implicit, non-declarative learning should be more resistant to choking than skill acquired through explicit rule accumulation, because implicit knowledge is not available for reinvestment [10].

The over-arousal account treats the motivation-performance relationship as primarily mediated by physiological arousal, with arousal elevation eventually crossing an optimal point and degrading performance [1]. This theory implicates motivational circuits — mesolimbic dopaminergic pathways and the ventral striatum — as the proximate neural mediators of choking, predicting that exaggerated activation of these systems under high incentive should correlate with performance decrements [1]. Experiments combining fMRI with concurrent physiological recording have provided direct evidence that sympathetic arousal, indexed by skin conductance, is selectively elevated during failed performance on high-incentive trials, consistent with this account [9]. These three accounts are complementary rather than contradictory: the neural systems they implicate can co-occur and interact within the same individual and performance episode [1][4].

4. Neuroimaging Evidence: Task-Based fMRI Studies

The application of fMRI to choking has yielded convergent evidence implicating reward-sensitive motivational circuits and executive control networks [1]. An early influential investigation developed a Pac-Man-like game in which participants pursued a prey target for either low (£0.5) or high (£5) monetary rewards [1]. Participants caught the high-payoff prey less successfully and made more errors in the high-reward condition, constituting experimental choking [1]. As participants approached the prey, activity escalated in mesolimbic reward regions including the dorsolateral striatum, ventral striatum, medial orbitofrontal cortex, and rostral anterior cingulate cortex [1]. Midbrain activity spanning the ventral tegmental area and substantia nigra was significantly stronger in high- relative to low-reward conditions and correlated with performance decrements and error commission, whereas right ACC and medial prefrontal activity correlated with better performance and reduced susceptibility to incentive-induced errors [1].

Subsequent investigations examined loss aversion using a spring-mass visuomotor task with graded incentives [9]. Participants who appraised the incentive as a potential monetary gain exhibited significant choking at the highest incentive level, with performance at the \$100 level substantially worse than at its individual peak [9]. The ventral striatum showed a positive linear relationship with incentive magnitude during incentive presentation, and this neural encoding was significantly stronger in the baseline condition — in which choking occurred — than in a reappraisal condition [9]. Individual differences in the attenuation of this ventral striatal response predicted the degree to which choking was reduced [9].

In contrast, fMRI investigation of a challenging visuomotor task using the Snake paradigm favored a distraction rather than over-arousal account [4]. High rewards were associated with increased dorsolateral prefrontal cortex activity prior to movement onset, with functional connectivity between DLPFC and motor cortex likewise elevated [4]. However, the magnitude of this pre-movement connectivity increase was inversely related to choking propensity: participants with the smallest increases in frontal-motor connectivity were most likely to choke [4]. This inverse relationship suggests choking reflects a failure of top-down executive influence on motor cortex rather than excessive monitoring [4]. Loss aversion showed no significant correlation with choking in this paradigm, while trait impulsivity showed a significant positive relationship [4][1].

Further fMRI work has specifically implicated loss aversion in the neural genesis of choking [1]. When individuals perform a motor task for monetary incentives, striatal activity rapidly

switches between activation and deactivation as incentives increase, and decrements in performance together with striatal deactivations are directly predicted by an independent behavioral measure of loss aversion [1]. In a follow-up, behavioral loss aversion was correlated with performance decrements in high-reward contexts, strengthening the link between the fear of losing and choking [1]. When incentives were framed either as potential gains or as potential losses, the striatum was activated for both increasing prospective gains and increasing prospective losses at the time of incentive presentation, indicating that the region may encode the general level of arousal elicited by either the excitement of prospective reward or the fear of loss [1][9]. These observations help reconcile the apparently divergent findings of the neuroimaging literature, in which subcortical motivational over-activation and prefrontal regulatory under-activation have both been observed in association with performance failure [1][4].

A key insight from this literature is the importance of dissociating the anticipation phase, during which the incentive is presented and motivational systems engaged, from the execution phase, during which performance is produced [1]. The source of pressure determines which anticipatory circuits are recruited — reward-encoding regions by monetary incentive and negative-valence regions by fear of failure [1]. Studies of mathematics anxiety illustrate these dual mechanisms in a cognitive domain: anticipating mathematics increased frontoparietal activity in highly anxious individuals, and the relationship between this activity and performance deficits was fully mediated by activity in motivation-relevant subcortical structures — including the caudate, nucleus accumbens, and hippocampus — during task performance, while the mere prospect of a feared task activated the brain's pain network, comprising the mid-cingulate cortex and insula [1]. Importantly, the self-report measure of math anxiety was not correlated with the performance deficit itself, indicating that it is not poor performance that produces anxiety but rather that anxiety and its neural correlates drive the deficit [1][5]. These findings collectively demonstrate that both attentional distraction and over-arousal contribute to choking, and that the interaction between the two processes may be crucial to understanding the phenomenon [1].

5. Neural Networks of Stress: State-Based fMRI and Connectivity Analyses

Investigations of the neural state associated with acute psychological stress, induced through paradigms such as the Montreal Imaging Stress Task, reveal systematic reorganization of large-scale brain networks highly relevant to choking [1]. During social-evaluative stress combined with demanding cognitive performance, significant deactivation of the limbic system — hippocampus, hypothalamus, medio-orbitofrontal cortex, and anterior cingulate cortex — was observed in participants whose stress reactivity was characterized by elevated cortisol, with the magnitude of hippocampal deactivation directly proportional to the cortisol response [1]. The memory functions of the hippocampus, critical for tactical decisions requiring integration of past experience, may therefore be systematically impaired during high-pressure competition that activates the hypothalamic-pituitary-adrenal axis [1][7].

Large-scale network analysis demonstrated that acute stress promotes reorganization of functional connectivity such that a salience network — anterior insula, midbrain, thalamus, dorsal ACC, and temporoparietal cortex — increases in responsiveness and internal connectivity as a function of stress response magnitude [1]. This expansion occurs at the expense of the fronto-parietal executive control network encompassing the DLPFC, which enables working memory and deliberate attentional control [1]. The hypothesized consequence is a shift from top-down, goal-directed control toward bottom-up, stimulus-driven attention, at the cost of the flexible cognition that complex sport skills demand [1][13].

The neuromodulator noradrenaline was identified as the primary driver of this reorganization: beta-adrenergic receptor blockade, but not cortisol synthesis inhibition, diminished the salience network expansion, implicating the sympathetic-adrenal axis rather than the HPA axis as the primary mediator of acute network reconfiguration [1]. This aligns with the observation that skin conductance responses — indexing sympathetic activity — are selectively elevated during choking in high-incentive trials, and that cognitive reappraisal abolishes this hyperarousal while reducing behavioral choking [9]. A dual-system model distinguishes the salience network, associated with the sympathetico-adrenal axis, from the fronto-parietal executive network, associated with the HPA cortisol axis, proposing that optimal performance under pressure depends on a balanced activation of the two, with choking representing a state in which salience-driven emotional reactivity overwhelms executive-driven cognitive control [1].

The convergence between task-based neuroimaging of choking and large-scale network analyses is striking: both demonstrate that cortical regulatory activity is weakened while subcortical motivational and salience-processing responses are amplified under pressure [1]. A key question for future research is what differentiates "chokers" from those who thrive under pressure in terms of brain network configuration — whether choking-resistant individuals maintain a more favorable balance between salience and executive control networks, or a greater capacity to sustain prefrontal regulatory engagement in the face of stress-induced subcortical activation [1][13].

6. The Dorsal Anterior Cingulate Cortex and Sequential Motor Performance Under Anxiety

A significant advance came from work demonstrating a causal role of the dorsal anterior cingulate cortex (dACC) in the anxiety-induced deterioration of sequential motor performance — the form most characteristic of real athletic competition [6]. Unlike laboratory paradigms involving single-shot actions, sports and music require the assembly of sequential action components into longer performance streams, a structure that creates specific vulnerabilities [6].

A three-experiment investigation using a differential motor sequence learning paradigm showed that athletes who pre-learned two sub-sequences and then combined them (part learners) exhibited dramatically greater deterioration under anxiety than those who learned the complete sequence from the outset (single learners), precisely at the junction between pre-learned components [6]. Under anxiety induced by threat of electrical shock, galvanic skin response was significantly elevated above pre-test levels in both groups ($T(17)=2.35$, $p=0.03$ for part learners; $T(17)=2.17$, $p=0.04$ for single learners), confirming effective anxiety induction [6]. Part learners made significantly more errors ($T(17)=2.75$, $p=0.014$) and showed a specific increase in inter-press time variability at junctions that emerged under anxiety but not during practice [6].

Functional MRI revealed that dACC activity at junctions was positively correlated with inter-press time delay in part learners ($p=0.00017$) but negatively correlated in single learners ($p=0.0010$) [6]. The dACC activity was positively correlated with number of errors in part learners ($R=0.58$, $p=0.016$) but negatively correlated in single learners ($R=-0.64$, $p=0.0054$), suggesting that in part learners the dACC was engaged in maladaptive exploration of action alternatives at junctions, while in single learners it facilitated commitment to the already-learned sequence [6].

The causal role of the dACC was established through 1 Hz repetitive transcranial magnetic stimulation (rTMS), which produces inhibitory effects on the stimulated region, administered before the anxiety test [6]. TMS-stimulated part learners made significantly fewer errors than

SHAM-stimulated controls ($p=0.037$), and the junction-specific variability pattern was abolished, rendering their performance indistinguishable from single learners [6]. Crucially, anxiety levels did not differ between TMS and SHAM groups, confirming that TMS reduced the motor sensitivity to anxiety rather than attenuating anxiety itself — demonstrating that anxiety-induced motor deterioration can be neurobiologically decoupled from the subjective experience of anxiety [6].

These findings provide direct support for the self-focus theory of performance anxiety, which holds that anxiety reactivates the explicit cognitive processes used during initial learning, causing them to interfere with subsequently automatized control [6]. Part learners retained accessible declarative knowledge about the junctions that could be maladaptively reinvested under anxiety, whereas single learners, having acquired the sequence as a unified whole, had no such junction-specific knowledge to reinvest [6]. The dACC activity associated with the deterioration of sequential performance was anatomically distinct from the dACC activity correlated with the processing of the aversive shocks themselves, which was localized to a more posterior region, confirming that the dACC's role in choking is specifically related to the cognitive control of motor sequences under anxiety rather than to generic pain or threat processing [6]. The context-dependent contribution of the dACC — impairing performance in part learners while facilitating it in single learners — aligns with its established role in representing both the exploration of alternative actions and commitment to selected actions, with the balance determined by learning history and task structure [6].

This interpretation connects the dACC findings to the broader explicit monitoring framework and to the protective value of implicit learning approaches that minimize reinvestible declarative knowledge [10][5]. Computational modeling of the cortex–basal ganglia loop and cerebellum further suggests how excessive prefrontal activation under stress may disrupt the cortico-striatal stream that normally supports automatic execution of motor sequences [14]. In a systems-level model in which a cortex–basal ganglia motor loop learns through a novelty-based motor prediction error while the cerebellum minimizes the remaining aiming error, motor performance depends on the smooth interaction of these subsystems; the model reproduces human motor adaptation under cognitive control and illustrates how interference from higher cognitive processes can perturb the network that mediates automatic sequence production [14][6]. The dACC findings thus position this region as a central node in the neural circuitry of sport-relevant choking and identify it as a promising target for both diagnostic assessment and therapeutic intervention [6].

7. Error Monitoring, the Error-Related Negativity, and Sport Anxiety

The neural response to performance errors — indexed by the error-related negativity (ERN), a negative deflection in the event-related potential occurring approximately 50 milliseconds after error commission over frontocentral sites — has emerged as a sensitive marker of the interaction between anxiety and performance monitoring underlying choking [5]. The ERN is thought to reflect rapid evaluation of errors by an action-monitoring system centered on the anterior cingulate and dorsolateral prefrontal cortices, with amplitude reflecting the degree to which errors are experienced as aversive or motivationally significant [5].

A controlled investigation pre-screened 216 undergraduate athletes using the Sport Anxiety Scale-2 (mean 27.1, SD 7.1) to identify those with high (>35) and low (<20) sport anxiety, selecting 14 in each group [5]. Athletes performed a spatial Stroop task under two counterbalanced conditions: a performance-evaluation condition, in which a research assistant monitored performance from a laptop behind the participant, and a control condition, performed alone [5]. The primary finding was a significant interaction between sport anxiety group and evaluation condition in ERN amplitude ($F(1,26)=4.96$, $p=0.04$, $\eta^2=0.16$) [5]. For

the high anxiety group, the ERN was significantly larger under evaluation than control ($t(13)=2.75$, $p=0.02$); for the low anxiety group, no difference emerged ($t(13)=0.79$, $p=0.45$) [5]. Behavioral measures of reaction time and error rate did not differ between conditions, indicating that the neural differences emerged without manifest behavioral choking, consistent with the prediction that ERN enhancement reflects early neural stages of choking vulnerability [5].

These findings support explicit monitoring over distraction: if distraction were operating, attention would be drawn away from execution and the ERN — which is attenuated when less attention is allocated to monitoring — would decrease rather than increase under evaluation [5]. The enhanced ERN under evaluation instead indicates increased inward attention to performance monitoring specifically when performance is being assessed [5]. An unexpected finding was that high-anxiety athletes showed a smaller ERN than low-anxiety athletes in the control condition ($t(13)=2.20$, $p=0.04$), suggesting that choking-susceptible individuals attend less to errors when performing alone and engage excessive monitoring only under social-evaluative pressure [5]. The vulnerability is therefore not constant hypervigilance but a context-sensitive reactivity emerging under evaluation [5].

The ERN findings also connect to the developmental and environmental origins of choking vulnerability: children with more critical parents and those exposed to harsh parenting show larger ERN amplitudes, suggesting that punitive experiences surrounding errors may sensitize individuals to their mistakes across development [5]. Because the ERN can be measured non-invasively with high temporal precision, it holds promise both as a biomarker for identifying choking-susceptible athletes and as a target for interventions aimed at normalizing error reactivity under evaluative pressure [5][8].

8. Dopaminergic and Noradrenergic Mechanisms

Neurochemical evidence implicates the dopaminergic and noradrenergic systems as key mediators of the motivational and stress-related mechanisms underlying choking [1]. The mesocorticolimbic dopamine system — ventral tegmental area, nucleus accumbens, ventral striatum, and prefrontal cortex — is a primary substrate for reward processing and incentive salience, directly relevant to the over-arousal account [1]. Positron emission tomography evidence demonstrated that tonic dopamine synthesis capacity was positively associated with performance decrements in a Stroop task under high incentives, suggesting that monetary bonuses may impair cognitive control by over-exciting the dopaminergic system beyond optimal levels [1].

Noradrenaline, released by the locus coeruleus under acute stress, plays complementary roles [1]. Beta-adrenergic receptor blockade, which prevents noradrenergic signaling, specifically attenuates the stress-induced expansion of the salience network and the accompanying reduction in executive control network connectivity, identifying the sympathetic-adrenal axis as the primary driver of the network-level state changes accompanying acute pressure [1]. The interaction between catecholamines and glucocorticoids released via the HPA axis produces complex, time-dependent effects on prefrontal function [7][13]. Under optimal conditions, dopaminergic D1 receptor stimulation supports working memory by enhancing the signal-to-noise ratio of persistent prefrontal firing during delay periods; excessive catecholamine release under acute stress, combined with glucocorticoid elevation, disrupts this balance and impairs working memory [7].

The relationship between cortisol and working memory, however, is more complex than a simple impairment model [15]. An experimental within-subject study comparing acute physical activity and psychological stress found that vigorous exercise and the Trier Social Stress Test produced comparable, statistically indistinguishable increases in salivary cortisol,

yet neither manipulation significantly changed working memory performance measured immediately or after a 35-minute delay [15]. Grouping participants by their cortisol response showed that working memory performance was not associated with cortisol increases, leading the authors to conclude that cortisol elevation is not sufficient to impair working memory and that additional mechanisms — particularly concurrent catecholaminergic activity — are required [15]. This finding converges with meta-analytic evidence indicating that acute stress and exogenous cortisol produce different patterns of effects on executive function, pointing to a dominant role for catecholaminergic rather than glucocorticoid mechanisms in stress-induced cognitive impairment [13][15].

The dissociation between cortisol elevation and working memory impairment has important implications for the interpretation of choking research [15]. It implies that the physiological arousal accompanying high-pressure competition does not impair cognition through a single, uniform hormonal pathway, but rather through a specific configuration of catecholaminergic and glucocorticoid signaling whose effects depend on timing, task difficulty, the brain region involved, and individual differences [15][13]. This complexity helps explain why the cognitive consequences of competitive stress are variable across athletes and situations, and why interventions that simply reduce general arousal may be insufficient unless they target the specific neuromodulatory state that disrupts prefrontal function [7][15]. It also underscores that the elevated arousal of exercise and the elevated arousal of psychological threat, although superficially similar in their hormonal signatures, may differ in their consequences for the brain networks that govern skilled performance [15][1].

9. Working Memory Capacity, Attentional Control, and Performance Under Pressure

Working memory capacity (WMC) — defined not merely as storage but as the ability to maintain goal-relevant information while suppressing interference — is a central individual-difference variable in theories of choking [1]. High-WMC individuals show superior top-down attentional control and greater resistance to distraction, capacities that should buffer against pressure via the distraction mechanism, yet paradoxically they may be most vulnerable to explicit-monitoring choking when tasks rely heavily on procedural knowledge [1][12].

Experimental investigation of the relationship between WMC, state anxiety, and attentional control examined 56 student athletes under low and high state anxiety induced through a multi-source manipulation combining limited response windows, noise punishment, and electric-shock threat [12]. High state anxiety impaired attentional control indexed by antisaccade latency ($F(1,54)=12.988$, $p=0.001$, $\eta^2p=0.194$) and error rate ($F(1,54)=6.199$, $p=0.016$, $\eta^2p=0.103$) [12]. High-WMC individuals demonstrated shorter antisaccade latency than low-WMC peers ($F(1,54)=12.246$, $p=0.001$, $\eta^2p=0.185$), confirming the protective effect of WMC, though the interaction between state anxiety and WMC was not significant, suggesting partly independent mechanisms [12].

A critical extension used 15 sessions of adaptive spatial n-back training to directly manipulate WMC [12]. The training group showed improved performance on the trained task and a trend-level improvement on an untrained WMC measure, and, more importantly, significantly improved antisaccade latency relative to controls ($F(1,30)=7.012$, $p=0.013$, $\eta^2p=0.189$) — causal evidence that WMC enhancement produces far-transfer benefits to attentional control, the cognitive flexibility that distinguishes athletes who maintain performance under pressure [12]. The use of an antisaccade task to index attentional control is methodologically important, because eye-movement measures separate attentional control from task performance more cleanly than do reaction-time or accuracy measures, which can be maintained through compensatory effort even when attentional control is impaired [12][16]. Attentional control

theory frames these effects in terms of an imbalance between the goal-directed and stimulus-driven attentional systems: under anxiety, the influence of the goal-directed system diminishes while the stimulus-driven system gains influence, producing distraction [12].

The direct manipulation of working memory capacity through training, rather than the mere comparison of high- and low-capacity individuals, addresses a longstanding limitation of correlational designs and strengthens the causal inference that working memory capacity supports attentional control [12]. This finding has practical significance for choking prevention, because it suggests that the prefrontal regulatory machinery underlying resistance to pressure-induced distraction is trainable rather than fixed [12][7]. At the same time, the dissociation between latency and error-rate effects, and the absence of a state anxiety by working memory capacity interaction, indicate that state anxiety and working memory capacity influence partly independent components of attentional control, with state anxiety acting predominantly on the stimulus-driven system and working memory capacity on the goal-directed system [12][13].

Meta-analytic synthesis of acute stress effects on executive function found that stress impaired working memory and cognitive flexibility while producing more nuanced effects on inhibition, with enhancement of response inhibition but potential impairment of cognitive inhibition [13]. These effects depended on the delay between stress onset and assessment, reflecting distinct rapid nongenomic and slower genomic mechanisms of cortisol action, and on working memory load, with effects most apparent under high-load conditions — precisely those characteristic of high-pressure sport requiring concurrent tactical, technical, and self-regulatory demands [13]. The distinction between trait and state anxiety is central here: trait anxiety, a stable propensity to experience anxiety, consistently impairs attentional control across studies, whereas the effects of state anxiety have been more variable, an inconsistency that may reflect differences in the intensity of state anxiety induced in different experimental contexts [12]. The relationship between state anxiety intensity and performance appears to follow an inverted-U function, such that moderate state anxiety may facilitate attentional control through enhanced arousal and motivation while high state anxiety impairs it, consistent with the Yerkes–Dodson framework underlying the over-arousal account [12][1].

The disruptive effect of anxiety on goal-directed processing is also evident in dynamic perceptual-cognitive tasks [16]. In skilled and less-skilled table tennis players performing serve-anticipation under high and low anxiety, high cognitive state anxiety reduced processing efficiency — the ratio of accuracy to response time — without reducing anticipation accuracy itself, consistent with processing efficiency theory, which holds that anxiety degrades the efficiency of cognitive processing more than its ultimate effectiveness because performers compensate by recruiting additional effort [16]. High anxiety impaired anticipation efficiency specifically in early anticipation tasks that require athletes to shift attention flexibly between internal memory-based expectations and external kinematic cues, supporting attentional control theory's claim that anxiety disrupts the goal-directed attentional system and the shifting and inhibition functions of the central executive [16][13].

10. Motor Automatism and Implicit Learning: Protection Against Choking

A central prediction of explicit monitoring theories is that motor skills acquired through implicit learning routes, which minimize the accumulation of declarative, verbally articulable knowledge about execution, should be more resistant to reinvestment under pressure [10]. A systematic review of 25 controlled trials involving 1,040 participants examined whether implicit motor learning interventions — analogy learning, errorless learning, dual-task learning, and external focus learning — produced superior dual-task performance, an index of greater automatization [10]. Across 39 implicit-explicit comparisons, most showed no

significant group difference, but the remaining comparisons consistently favored implicit learning: several demonstrated superior absolute dual-task performance combined with less declarative knowledge, and others demonstrated lower dual-task costs with less declarative knowledge, with the overall evidence rated level 3 (unclear risk of bias) [10].

Analogy learning — providing a metaphorical instruction summarizing the global movement structure — emerged as a particularly effective implicit strategy: in all comparisons where declarative knowledge was assessed, analogy groups reported significantly less movement-specific declarative knowledge than explicit-instruction groups [10]. This reduction in verbally articulable movement knowledge is theoretically protective against reinvestment, as performers cannot consciously retrieve and apply knowledge they never explicitly formulated [10]. The neural plausibility of this protection is supported by computational modeling of motor learning, in which a cortex–basal ganglia reinforcement-learning loop and a cerebellar supervised-learning system jointly govern movement; stress and excessive prefrontal engagement can disrupt the cortico-striatal stream that mediates automatic sequence execution, undermining proceduralized control [14][6].

External attentional focus — directing attention to the environmental effects of movements rather than to bodily mechanics — represents a related implicit strategy [17]. Internally directed instructions provided early in practice impaired skill acquisition and produced decrements under stress, whereas externally directed instructions showed no such adverse effects regardless of when introduced [17]. The timing of instruction interacted with focus: early internal instructions were particularly detrimental, establishing a mode of conscious motor control that became a liability under pressure, while delaying or avoiding such instruction allowed automatic control to develop [17].

A detailed kinematic investigation of golf putting under anxiety found that participants who learned with an external focus continued to improve under pressure, while those given no focus instructions displayed classic choking [18]. Notably, participants who learned with an internal focus also maintained performance — through a different mechanism: habituation to the self-focused mode during learning desensitized them to the disruptive self-focus that anxiety induces, consistent with the specificity-of-learning principle and with the finding that self-consciousness training protects against choking [18]. The critical variable is thus the match between the attentional mode practiced during learning and that imposed by anxiety during performance [18].

The benefits of external focus extend to the physiological economy of movement [19]. In a simulated race-driving task, participants trained with a distal external focus outperformed those trained with an internal focus across both neutral and competitive conditions, and their superior performance was accompanied by smaller reductions in heart rate variability — a physiological index of mental effort — indicating more automatic, less effortful control [19]. The internal focus group's poorer performance was associated with larger heart rate variability reductions, reflecting greater investment of cognitive resources in conscious movement control [19]. These findings provide convergent psychophysiological support for the constrained action hypothesis, which holds that conscious attention to movement mechanics interferes with the automatic processes that normally regulate skilled movement, while external focus promotes a more unconscious, reflexive, automatic mode of task processing [19][18].

The kinematic analysis of golf putting under anxiety further clarified the mechanism by which attentional focus during learning shapes resistance to choking [18]. Participants who adopted an external focus reported less explicit knowledge regarding the movements of their own body than those who adopted an internal focus, and it was the nature rather than the sheer quantity of explicit rules generated during learning that governed reinvestment under anxiety

[18]. Skills are more likely to break down under anxiety when performers have accumulated accessible, conscious, task-relevant knowledge centered on body movements rather than on the external effects of those movements [18][10]. This refinement of reinvestment theory has direct coaching implications: practitioners should minimize the acquisition of explicit, body-focused movement knowledge by encouraging an external focus, or, alternatively, apply the specificity-of-learning principle by having athletes practice under the self-focused conditions that anxiety will later impose [18][17].

11. EEG Biomarkers and Brain Oscillations in Athletic Performance

Electroencephalography offers the temporal resolution to capture the rapid dynamics of brain activity during performance and to identify oscillatory signatures of optimal versus pressure-compromised states [8]. Alpha oscillations (approximately 8–12 Hz) have attracted attention as potential biomarkers of relaxed, focused readiness: in golfers, increased left-hemisphere alpha power was associated with reduced error in subsequent putts, and alpha power was higher for best versus worst shots in elite archers [8]. The relationship is complex and bidirectional, with alpha functioning as a pulsed-inhibition mechanism whose direction of change relative to optimal performance varies across tasks and individuals [8].

Frontal midline theta power (approximately 4–8 Hz) has consistently distinguished experts from novices in precision sports: expert golfers showed higher fronto-midline theta and parietal upper alpha during putting, while expert rifle shooters demonstrated increased frontal midline theta in the seconds before shooting, with inverse modeling locating its source in the medial frontal cortex and ACC, regions associated with focused attention [8]. These theta signatures index the focused attention and suppression of irrelevant thought that anxiety and performance pressure are liable to disrupt [8][16]. Beta oscillations (15–30 Hz) contribute to sensorimotor integration and serve as a binding mechanism linking motor cortex, premotor areas, and cerebellum during movement preparation, and have been observed to increase in relation to force-output decrement and neuromuscular fatigue in the context of exercise-induced muscle damage [8].

A working-memory model translating these dynamics to sport proposes that beta-gamma oscillations enable the gate-in phase during which target information is acquired, theta oscillations maintain a selective-gating mode that protects working memory contents against distractors during the delay, and alpha oscillations enable the gate-out phase in which working memory is efficiently cleared on task completion [8]. Anxiety-induced disruption of this orderly oscillatory sequence, through the stress-related amplification of the salience network at the expense of the executive control network, may explain the working memory dysfunction, attentional narrowing, and increased distractibility characterizing performance under pressure [8][1]. The framework of unified brain rhythms further situates these waking oscillations on a continuum with sleep-related activity, implying that the quality of an athlete's sleep — and the slow-wave and spindle activity that consolidate motor learning — may indirectly shape the oscillatory readiness of the brain for high-pressure performance [8].

The integration of EEG biomarkers with biomechanical and kinematic measurement holds particular promise for sport, as the optimized performance state achieved by elite athletes offers a privileged window onto the neuronal oscillations linked to sensorimotor and cognitive control [8]. The neural efficiency hypothesis — derived from the observation that more skilled performers exhibit lower, more focused brain activation than less skilled performers on the same task — has been extended from cognitive expertise to athletic expertise, suggesting that the pre-performance brain state of an expert is characterized by selective, economical engagement of task-relevant circuits rather than diffuse over-activation [8]. Anxiety and pressure, by promoting the diffuse, salience-driven activation pattern described

in the stress-network literature, may push the athlete's brain away from this efficient state and toward the pattern associated with performance failure [8][1].

The practical promise of EEG biomarkers is illustrated by neurofeedback, which uses real-time feedback of brain activity to train athletes to regulate specific frequency bands [20]. A systematic review of 24 studies published between 2016 and 2023, spanning golf, basketball, swimming, rifle shooting, football, volleyball, athletics, judo, ice hockey, triathlon, handball, fencing, taekwondo, and darts, found that neurofeedback targeting alpha, beta, theta, and sensorimotor rhythm bands produced improvements in sport and cognitive performance, emotional regulation, and anxiety management [20]. Notable improvements were observed in technical skills, scoring, attention, concentration, reaction time, working memory, self-regulation, and cognitive anxiety [20]. The review noted, however, considerable heterogeneity in the frequency bands trained and the protocols used, alongside limitations including small sample sizes, an under-representation of female athletes, frequent absence of control groups, and a predominance of laboratory-based settings, and it called for standardized, sport-specific protocols, more diverse samples, and field-based studies to validate these findings [20][8].

12. The Ventral Striatum, Loss Aversion, and Reappraisal

The ventral striatum — nucleus accumbens, putamen, and adjacent structures — occupies a central position in theories of choking, serving as a hub for encoding incentive magnitude and translating reward signals into action preparation [9]. Its activation under high incentives provides the neurobiological substrate for the over-arousal theory, with excessive striatal response representing the neural correlate of the motivational over-excitation that produces choking [9][1].

The investigation of reappraisal as a choking intervention provided the most direct demonstration of the causal role of the ventral striatum [9]. Participants performing a demanding motor task for incentives ranging from \$0 to \$100 showed significant choking in the baseline condition, with performance at the \$100 level worse than at its peak by 6.70% on average [9]. When instructed to reappraise the monetary gain as a potential monetary loss — converting the incentive's psychological meaning without changing its objective magnitude — choking was reduced to 1.5% [9]. Neuroimaging confirmed that reappraisal attenuated ventral striatal encoding of incentive magnitude, and individual differences in the magnitude of this attenuation predicted the behavioral effectiveness of reappraisal [9].

The psychophysiological results provided historically significant evidence for the sympathetic hyperarousal hypothesis [9]. Skin conductance was proportional to incentive magnitude across all trials [9]. On failed high-incentive trials in the baseline condition, skin conductance was significantly greater than on successful high-incentive trials ($t(37)=2.60$, $p=0.01$) and than on failed high-incentive reappraisal trials ($t(37)=3.32$, $p=0.002$) [9]. The sympathetic response to incentive was lowest when participants played for low incentive, greater when they played for high incentive and succeeded, and greatest when they played for high incentive and failed — a pattern reminiscent of the relationship between performance and arousal described by the Yerkes–Dodson law [9][1]. This exaggerated sympathetic response to high-incentive failure was abolished by reappraisal and was specific to high-incentive failures rather than representing a general association with poor performance, constituting the first experimental demonstration that choking is accompanied by measurable sympathetic hyperarousal during task execution [9].

Notably, in this study reappraisal was not associated with greater activation of the prefrontal cortex, in contrast to much of the emotion-regulation literature in which reappraisal recruits lateral prefrontal regions to exert top-down control over subcortical emotional centers [9]. The authors suggested that this divergence may reflect the specific form of reappraisal applied —

participants were instructed in how to reappraise but were unaware of the intended behavioral consequences — and that the intervention operated by altering the representation of the incentive at the moment it was presented, before the motor task was performed, rather than by inhibiting an emotional response during execution [9]. Because this intervention targets the perception of the incentive directly rather than a specific disruptive response evoked by it, it may be applicable across a broader range of domains than interventions tied to a particular task mechanism, and to real competitive contexts where incentive levels are externally determined [9][4].

13. Individual Differences and Choking Susceptibility

Substantial individual variation exists in susceptibility to choking under equivalent objective pressure, and understanding its psychological and dispositional basis is essential for targeted prevention [1]. Fear of negative evaluation specifically predicts choking in social performance contexts, with high-fear athletes showing greater decrements when pressure is increased through social evaluation while low-fear athletes maintain performance — aligning with the explicit monitoring account, in which fear of evaluation sensitizes athletes to the significance of errors [1][5]. The social element of pressure may constitute a special source of choking that operates through mechanisms partly distinct from those engaged by monetary reward: the mere or imagined presence of an evaluative audience creates an atmosphere of evaluation that heightens arousal, and according to the biopsychosocial challenge-and-threat framework, performing a complex task in the presence of others can elicit a threat-type cardiovascular response that impedes performance [1][9]. Sex and individual differences in personality and cognitive ability further shape susceptibility, such that the source of pressure, the nature of the task, and the performer's dispositional profile jointly determine whether a given high-stakes situation precipitates failure [1][13].

Reinvestment — the tendency to consciously monitor and control movement mechanics — is a dispositionally stable trait with two separable dimensions: Conscious Motor Processing (CMP) and Movement Self-Consciousness (MSC) [11]. CMP reflects deliberate attention to movement process and is associated with positive characteristics including mental toughness, intrinsic and integrated regulation, mastery-approach goal orientation, and dispositional flow, whereas MSC — concern about style of movement and making a good impression in public — is associated with stress and mastery-avoidance goal orientation [11]. The demonstration that these dimensions relate to opposite poles of psychological functioning refines reinvestment theory: not all conscious attention to movement is detrimental, and movement self-consciousness rather than conscious motor processing is the dimension most strongly implicated in breakdown under pressure [11].

In a study of 160 skilled collegiate tennis players from the United States and Japan, low self-efficacy and high movement self-consciousness predicted self-perceived choking in competitive matches, while self-efficacy emerged as a protective factor [21]. Conscious motor processing was positively related to self-regulatory capacities, challenging the view that all reinvestment is harmful [21]. The cross-cultural dimension was notable: Japanese players reported less self-regulation and higher perceived choking than American players, pointing to the influence of cultural context and indicating a need for culturally sensitive prevention approaches [21].

The two dimensions of reinvestment can be reliably distinguished psychometrically and show divergent associations with motivational and emotional constructs across cultures [11]. In comparative analyses of Japanese and Singaporean samples, conscious motor processing was positively associated with mental toughness, intrinsic and integrated regulation, mastery-approach and task goal orientations, and dispositional flow, whereas movement self-

consciousness was positively related to stress and mastery-avoidance goal orientation [11]. The finding that the two dimensions relate to opposite poles of psychological functioning argues against treating reinvestment as a unitary, uniformly harmful tendency and supports analyzing its dimensions separately when assessing an athlete's vulnerability to breakdown under pressure [11][21].

Personality research has sought to identify the trait profile of the choking-susceptible athlete [2]. In 155 experienced golfers and archers, a combination of psychological traits — fear of negative evaluation, anxiety sensitivity, self-consciousness, perfectionistic self-presentation, and perfectionism — correctly classified 71% of choking versus non-choking participants, with choking associated specifically with anxiety and perfectionism in addition to social traits [2]. Notably, the same study distinguished choking from the yips, finding that whereas the yips were predicted predominantly by social-source traits such as perfectionistic self-presentation, choking was associated with anxiety and perfectionism as well as social traits, supporting the view that the two paradoxical performances, though related, have partly distinct psychological profiles [2]. The relationship between choking susceptibility and broader dispositional resilience is not straightforward: in a heterogeneous sample of 415 athletes, self-reported mental toughness did not differ significantly between athletes categorized as choking-susceptible and non-susceptible, suggesting that choking susceptibility and mental toughness are distinct, though possibly related, constructs [22]. This dissociation cautions against assuming that simply building generic mental toughness will protect against choking, and reinforces the value of targeting the specific mechanisms — perfectionism, fear of evaluation, and movement self-consciousness — most directly implicated in performance breakdown [22][2].

14. Sex Differences in Stress Responses and Choking

Sex is an important but understudied moderating variable in the neurobiology of stress and performance under pressure [13]. Meta-analytic evidence indicates that acute stress impairs working memory to a significantly greater degree in women than in men, a difference documented in both human behavioral studies and animal models [13]. This differential vulnerability may reflect sex differences in the neurochemical milieu of the prefrontal cortex under stress: gonadal hormones, particularly estrogen, modulate dopaminergic and noradrenergic signaling in ways that influence the optimal catecholamine concentrations for prefrontal function, creating a dynamic neurochemical context that affects stress resilience [7].

For cognitive flexibility, acute stress also appears detrimental, with some evidence that impairments differ by sex and are mediated, in rodent models, by sex differences in mu-opioid receptor binding in the orbitofrontal cortex [13]. The neural circuits mediating sex-differentiated stress responses encompass the HPA axis, the sympathetic-adrenal system, and their interactions with prefrontal regulatory circuitry, all directly relevant to the mechanisms of choking [7][13]. The complexity of these relationships is underscored by experimental evidence that cortisol elevation alone — whether produced by vigorous exercise or psychological stress — does not necessarily impair working memory, implying that the catecholaminergic and hormonal interactions that differ between the sexes, rather than cortisol per se, are the proximate determinants of stress-induced cognitive impairment [15]. Understanding these sex-specific profiles is essential for developing prevention strategies tailored to the distinct vulnerabilities of male and female athletes [13].

15. Prefrontal Cortex Dysfunction Under Stress: Neuroendocrine Mechanisms

The prefrontal cortex is uniquely vulnerable to disruption by acute psychological stress [7]. Under optimal, stress-free conditions, prefrontal microcircuits sustain the persistent neural firing that underlies working memory and flexible cognitive control through precisely

balanced dopaminergic D1 receptor stimulation of pyramidal cells, adrenergic regulation of local interneurons, and glutamatergic recurrent excitation among delay-active neurons [7]. This architecture is exquisitely sensitive to perturbation: even mild stress, activating the HPA axis and sympathetic-adrenal system, releases sufficient catecholamines and glucocorticoids to shift the prefrontal cortex from its optimal operating point into a state of reduced signal-to-noise and compromised working memory [7][1].

In non-human primates, mild stressors including loud white noise and anxiogenic agents reliably impair delay-based working memory, with lesion studies confirming that the deficit is specifically attributable to prefrontal dysfunction [7]. Stress-exposed prefrontal neurons show reduced delay-period firing while demonstrating heightened responsiveness to sensory distraction, consistent with a shift from goal-directed top-down control to stimulus-driven bottom-up processing [7]. Translated to sport, the prefrontal dysfunction induced by competitive stress would be expected to impair tactical decision-making, attentional flexibility, and strategic monitoring — precisely the capacities distinguishing expert from novice athletic performance in complex, dynamic contexts [7][16].

The molecular mechanisms involve excessive noradrenaline stimulating lower-affinity alpha-1 adrenergic receptors and high dopamine stimulating D1 receptors, both of which suppress the persistent firing that maintains working memory representations, effectively taking the prefrontal cortex "offline" and shifting behavioral control toward more habitual, stimulus-driven systems [7]. From an evolutionary standpoint this shift may have been adaptive under genuine physical threat, but in modern sport requiring flexible, strategically organized performance it produces the impairments that underlie choking [7]. Prefrontal catecholamine optimization also underlies the inverted-U relationship between arousal and cognitive performance, providing a mechanistic substrate for the Yerkes–Dodson law: very high incentive levels degrade performance because catecholamine over-stimulation pushes prefrontal function off its optimal operating point [7][1]. These molecular insights carry therapeutic implications, suggesting that behavioral interventions reducing sympathetic arousal — breathing-based relaxation, cognitive reappraisal, and expressive writing — may operate in part by limiting the catecholamine surge that disrupts prefrontal function [1][9][7].

16. Cognitive Interventions: Reappraisal, Attribution Training, and Mindfulness

Recognition of the multiple neural mechanisms of choking has supported development of targeted interventions, each addressing distinct aspects of the vulnerability [1]. Expressive writing — writing about thoughts and worries concerning an upcoming high-stakes performance immediately before execution — has demonstrated significant performance-enhancing effects in high-stakes examination settings, operating through the working memory pathway by offloading the rumination that would otherwise compete for capacity during performance [1].

The mechanism of expressive writing is consistent with the distraction account of choking: anxiety and worries compete for the working memory available for performance, and by affording an opportunity to reevaluate a stressful experience in a way that reduces the necessity to worry, writing alleviates the burden that rumination places on working memory before it can degrade performance [1]. Two laboratory and two randomized field experiments demonstrated that a brief writing assignment completed immediately before an important examination significantly improved exam scores, especially among highly anxious students, and the success of this intervention corroborates the role of arousal and worry in producing choking [1]. Related metacognitive reframing techniques — for example, informing performers that physiological responses such as a rapid heartbeat are beneficial rather than harmful for performance — can likewise improve performance under stress [1][3].

Cognitive reappraisal — reconstruing the meaning of the incentive or situation to reduce its emotional salience — has been validated as an efficacious intervention with a neurobiologically characterized mechanism: reappraising a monetary gain as a potential loss reduced choking and simultaneously attenuated ventral striatal encoding of incentive magnitude and abolished the sympathetic hyperarousal accompanying failed high-incentive performance [9]. Because this strategy targets the internal representation of the incentive rather than the external incentive structure, it is applicable to real competitive contexts where the stakes are fixed and cannot be altered [9][4]. It is important to note, however, that emotion regulation during task execution may sometimes backfire, because the regulatory effort itself consumes executive resources needed for performance; this consideration favors interventions, such as pre-task reappraisal and expressive writing, that act before execution rather than during it [1][9].

Attribution training extends the cognitive approach by modifying how athletes interpret the causes of their performance outcomes [3]. In a survey study of 328 elite athletes who had experienced choking, attribution training was significantly associated with reductions in fear of failure ($\beta=-0.548$, $p<0.001$) and self-criticism ($\beta=-0.531$, $p<0.001$), with fear of failure and self-criticism in turn predicting self-efficacy [3]. By correcting maladaptive attributions of failure and encouraging adaptive interpretations of pressure as a challenge rather than a threat, attribution training reduced negative emotions and enhanced self-efficacy, expanding the cognitive-behavioral toolkit available to sport psychologists for choking prevention [3].

Mindfulness-based approaches target the anxiety that drives choking through a distinct mechanism [23]. In a study of 377 choking-susceptible athletes, mindfulness was negatively associated with perceived stress (standardized coefficient = -0.224 , $p<0.001$), and its protective effect on state-trait anxiety was mediated by both resilience and perceived stress (standardized indirect effect = 0.237 , $p<0.001$) [23]. These findings identify the psychological pathways through which mindfulness reduces anxiety in choking-susceptible athletes and provide an empirical basis for mindfulness training programs delivered by sports departments, clubs, and coaches [23]. The convergence of reappraisal, attribution training, and mindfulness on the reduction of anxiety and the enhancement of self-efficacy and resilience indicates multiple, partly complementary cognitive routes to protecting performance under pressure [3][23].

17. Behavioral and Psychological Skills Interventions

Beyond cognitive strategies, a range of behavioral and psychological skills interventions has been developed to protect performance under pressure [1]. Pre-performance routines (PPRs) — consistent sequences of cognitive and behavioral steps performed before skill execution — are widely used to minimize the effects of choking [24]. A randomized study of 54 beach volleyball players compared PPR interventions, including an embodiment technique using left-hand dynamic handgrip, against a control group on serving accuracy under pressure induced through videotaping and ego-relevant instructions [24]. This study found no significant differences in serving accuracy among the intervention groups and the control group and no additive benefit of combining PPR components, a null result the authors attributed in part to the limited efficacy of the pressure manipulation [24]. Such findings underscore that the effectiveness of pre-performance routines may be context- and task-dependent and that rigorous, ecologically valid testing is needed to establish when and for whom they protect against choking [24].

Tailoring interventions to the individual athlete's profile appears to enhance their effectiveness [25]. A mixed-methods collective case study of choking-susceptible performing artists — a population facing performance-anxiety challenges analogous to those of athletes

— found that psychological interventions tailored individually in a coaching setting produced reductions in performance anxiety and fear of negative evaluation and increases in self-efficacy and performance quality, with most participants showing lower heart rate when performing for a jury [25]. Qualitative themes included managing nervousness, increased self-confidence, and greater enjoyment of performing, emphasizing the importance of individual context in determining whether an intervention is beneficial [25]. These findings strengthen the bridge between performing arts and sport psychology and support an individualized, profile-matched approach to choking prevention [25].

External attentional focus instructions, validated in motor learning and kinematic research, also function as a practical in-the-moment behavioral strategy, promoting the automatic mode of movement control that is robust to competitive pressure [19][18]. The timing of instruction is again relevant in applied settings: because internally focused, body-mechanics instructions delivered early in learning are particularly detrimental to subsequent performance under pressure, coaches should favor external-focus cues from the outset of skill acquisition rather than introducing body-focused corrections that may aid immediate performance at the cost of long-term robustness [17][18].

Neurofeedback represents a technologically distinct behavioral intervention: by training athletes to regulate the EEG frequency bands associated with optimal performance states, neurofeedback has produced improvements in cognitive anxiety, self-regulation, attention, and reaction time across a wide range of sports, offering a direct route to modifying the brain states that underlie choking vulnerability [20][8]. The evidence indicates that sensorimotor rhythm training is especially beneficial for fine motor skills and accuracy, while protocols such as theta-beta ratio training may be better suited to complex motor tasks requiring rapid reactions, implying that neurofeedback protocols should be tailored to the specific cognitive and motor demands of the sport in question [20]. Standardized, sport-specific protocols and rigorous methodology remain to be established before neurofeedback can be confidently recommended as a routine component of choking prevention [20].

The breadth of available behavioral interventions — pre-performance routines, tailored psychological coaching, external focus strategies, mindfulness, and neurofeedback — provides practitioners with multiple, mechanism-specific options to match to the individual athlete [25][20]. Crucially, the choice among them should be guided by the athlete's dominant mechanism of failure and by ecologically valid evaluation of effectiveness, since interventions that succeed in one task or population may not transfer to another, as illustrated by the null findings for pre-performance routines in beach volleyball serving [24][25]. The integration of behavioral interventions with the cognitive strategies described above, all grounded in the neurobiological understanding of choking, offers a comprehensive and individualized approach to protecting performance under pressure [23][3].

18. Training Strategies and Prevention of Choking

The neurobiological evidence converges on a set of training strategies that may reduce vulnerability to choking through mechanisms operating at multiple levels [10]. Implicit learning approaches — analogy learning, errorless learning, and dual-task learning during skill acquisition — minimize the accumulation of verbally articulable knowledge that could be reinvested to disrupt automatized performance under pressure [10]. Practical implementation requires coaches to develop sport-specific analogies that are biomechanically accurate yet experientially intuitive, and to resist the tendency to provide detailed step-by-step movement prescriptions that may facilitate early acquisition but create long-term vulnerability [10][17].

Pre-performance anxiety management should be differentiated by mechanism [1]. Athletes who choke primarily through distraction may benefit most from expressive writing or mindfulness-based approaches that attenuate rumination before competition [1][23]. Athletes who choke through explicit monitoring may benefit from practicing under conditions of increased self-focus to habituate to self-consciousness, or from adopting an external attentional focus that naturally reduces step-by-step monitoring [18][19]. Athletes who choke through sympathetic hyperarousal under extremely high-stakes conditions may benefit from cognitive reappraisal or attribution training that modulates the subjective and neural representation of competitive incentives and the interpretation of pressure [9][3].

Working memory training may represent a more indirect but potentially durable approach, strengthening the prefrontal regulatory capacity that resists anxiety-induced distraction across contexts, although the far-transfer of such training to athletic populations requires further validation [12]. Anticipation training — providing extensive experience reading and responding to early kinematic cues — strengthens the long-term working memory representations supporting top-down attentional control during dynamic performance [16]. Notably, the adverse effect of anxiety on processing efficiency was more pronounced in skilled than in less-skilled players, because skilled athletes rely on a top-down, knowledge-based anticipatory mode that anxiety disrupts; the most expert athletes may therefore require the most targeted attentional-training interventions to protect their distinctive perceptual-cognitive advantages [16].

A comprehensive program should be individualized and multi-component, beginning with assessment of the athlete's choking profile — dispositional reinvestment, sport anxiety, working memory capacity, perfectionism, and characteristic mode of failure — and deploying the interventions best matched to that profile [21][2]. The foundation is laid during skill acquisition through implicit and externally focused learning [10][18], supplemented by pre-performance routines and cognitive strategies matched to the dominant mechanism [1][9], ongoing working memory and attentional training [12], neurofeedback where available [20], and anticipation training under graduated competitive pressure [16]. Because choking susceptibility is distinct from generic mental toughness, prevention must target the specific mechanisms implicated rather than relying on broad dispositional training [22].

The identification of an athlete's choking profile can draw on validated psychometric instruments alongside physiological and neurophysiological assessment [21]. The Movement-Specific Reinvestment Scale and its conscious motor processing and movement self-consciousness subscales offer a means of identifying athletes whose self-conscious attention to movement places them at elevated risk, while sport anxiety scales identify those most reactive to social-evaluative pressure [21][11]. The error-related negativity and EEG oscillatory markers provide objective neurophysiological complements to these self-report measures, and skin conductance and heart rate variability offer accessible indices of the autonomic arousal implicated in choking [5][19]. Combining dispositional, behavioral, and physiological assessment enables the practitioner to construct a richer profile than any single measure affords and to direct interventions toward the athlete's specific vulnerability [8][9].

Finally, prevention efforts should attend to the developmental and environmental contexts that shape choking vulnerability over time [5]. Because punitive and highly critical evaluative environments are associated with heightened error reactivity, coaching cultures that emphasize harsh criticism of mistakes may inadvertently cultivate the very neurophysiological sensitivity that predisposes athletes to choke [5]. Coaching approaches that frame errors as information for improvement rather than as occasions for punishment, that support athletes' autonomy and self-efficacy, and that foster adaptive attributions of

failure may therefore reduce choking vulnerability at its developmental source, complementing the more proximal interventions delivered to individual athletes [5][3].

19. Future Directions and Conclusions

Despite substantial progress, significant gaps remain [6]. The majority of neuroimaging studies have employed laboratory paradigms that bear limited resemblance to the complexity and ecological validity of real sport performance, capturing important aspects of choking but missing its sequential, hierarchically organized, and strategically complex nature [6][1]. Developing validated paradigms that embed sequential motor performance, tactical decision-making, and social-evaluative pressure within a unified design, while maintaining the control necessary for mechanistic inference, is a major methodological challenge [6]. Portable EEG technologies create the possibility of recording brain oscillations during actual performance, opening the prospect of identifying the real-time neural correlates of choking in authentic competitive contexts, contingent on advances in artifact-rejection methodology [8][20]. Multi-modal measurement combining fMRI with skin conductance, heart rate variability, and behavioral metrics has already revealed the sympathetic hyperarousal signature of choking and could be extended with EEG to track the full time-course of incentive encoding, error monitoring, and attentional control [9][5]. Biologically realistic computational models of the basal ganglia, cerebellum, and prefrontal cortex offer a complementary framework for formalizing how stress disrupts the cortical-subcortical circuits underlying automatic performance [14].

Choking under pressure is a neurobiologically heterogeneous phenomenon that cannot be explained by any single mechanism or brain system [1]. Convergent evidence implicates three interacting neural systems: the ventral striatum encoding excessive motivational salience and driving sympathetic hyperarousal [9]; the prefrontal-motor circuit whose top-down regulatory influence is disrupted by catecholamine excess and attentional distraction [4][7]; and the dorsal anterior cingulate cortex whose anxiety-driven engagement introduces maladaptive cognitive control into automatized sequential motor performance, demonstrated causally through rTMS inhibition [6]. The error-related negativity provides a temporally precise marker of increased error aversiveness under evaluation specifically in sport-anxious athletes [5], working memory capacity determines attentional control efficiency under state anxiety [12], and implicit learning strategies offer a structural route to building pressure resistance into skill acquisition [10].

Critically, the neural vulnerability to choking is multidimensional, and effective prevention requires characterization of the individual athlete's specific mechanisms of susceptibility [22][2]. A growing arsenal of mechanism-specific interventions — cognitive reappraisal [9], attribution training [3], mindfulness [23], tailored psychological coaching [25], external attentional focus [19], implicit and analogy learning [10], working memory and anticipation training [12][16], and neurofeedback [20] — offers the prospect of substantially reducing the vulnerability of athletes to catastrophic performance failure. As neuroimaging and electrophysiology advance toward ecological validity and real-time applicability, the prospect of individualized, neurobiologically informed performance preparation moves from speculation toward achievable practice [8][1].

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