# Integrating Emotional Stimulation into Post-Stroke Motor Rehabilitation: Evidence and Future Directions

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**Abstract:** Stroke is a leading cause of mortality and long-term disability worldwide. Among these, motor dysfunction is the most prevalent sequela and markedly reduces quality of life and independence. Although conventional motor rehabilitation strategies have demonstrated certain clinical efficacy, low motivation and inadequate adherence often limit patients' active engagement and functional gains. In recent years, a novel treatment paradigm has shifted attention toward the role of emotional processes in motor rehabilitation. This approach treats emotion as a treatment variable and seeks to enhance neuroplasticity through targeted emotional stimulation aimed at strengthening sensorimotor circuits and promoting functional reorganization of the brain. This review synthesizes recent advances in applying emotional stimulation to post-stroke motor rehabilitation, provides an integrated perspective for medical staff and researchers, and outlines the theoretical foundations and future directions that can guide the development of more efficient and engaging interventions.

**Keywords:** Affective modulation; Neuroplasticity; Motivation; Virtual reality; Brain–computer interface; Dopamine

## 1. Introduction

Stroke remains a catastrophic global health challenge, characterized by rising incidence and a profound socioeconomic burden, particularly in China and the United States [1–6]. Among its multifaceted sequelae, motor dysfunction is the most prevalent and debilitating, affecting approximately 80% of survivors by disrupting critical cortical networks (e.g., M1, PMC, SMA) and severely compromising daily independence [7–9]. Current standard of care relies fundamentally on neuroplasticity, aiming to restore function through high-dose, repetitive, and task-oriented training [10]. However, despite these established protocols, a substantial proportion of patients fail to achieve optimal recovery, suggesting that the prevailing "motor-centric" rehabilitation model may be insufficient to address the complex reality of post-stroke deficits.

The efficacy of conventional rehabilitation is frequently constrained by a critical oversight: the inextricable link between motor performance and the patient's emotional state. Beyond pathophysiological barriers like interhemispheric imbalance [9], post-stroke emotional disturbances—such as depression and anxiety—have traditionally been compartmentalized as psychiatric comorbidities requiring separate management [11]. This fragmented approach ignores a fundamental dynamic: psychological distress precipitates low motivation and poor adherence, thereby reducing the training dosage essential for neural reorganization. When rehabilitation fails to engage the patient's affective drive, the "active ingredient" of neuroplasticity—intensive, voluntary participation—is significantly diluted, stalling functional gains.

In response to these limitations, a paradigm shift toward Experiential Neurorehabilitation is emerging, which posits that emotion is not a passive bystander but an active, modifiable driver of motor recovery [12]. By leveraging targeted emotional stimulation—ranging from traditional sensory cues to immersive virtual reality (VR) and brain–computer interface (BCI) systems—interventions can enhance engagement and strengthen sensorimotor circuits [13]. This review synthesizes recent advances in integrating emotional stimulation into post-stroke motor rehabilitation. We aim to elucidate the underlying neurobiological mechanisms, evaluate current technological applications, and outline a future framework for personalized, AI-enabled interventions that restore not just physical function, but the holistic human experience.

## 2. Neurobiological Foundations Linking Emotion and Motor Control

Emotion and motor control are tightly integrated and mutually influential within the brain. Understanding the neurobiological mechanisms underlying this bidirectional coupling provides a theoretical basis for utilizing emotion-based stimulation strategies to facilitate motor recovery and neurorehabilitation through well-established anatomical pathways and functionally integrated neural networks.

### *2.1 Anatomical and Functional Connectivity Between Prefrontal and Motor Cortices*

The prefrontal cortex (PFC), including the dorsolateral PFC (DLPFC), ventromedial PFC (vmPFC), orbitofrontal cortex (OFC), and anterior cingulate cortex (ACC), is among the most evolutionarily advanced neocortical regions [14]. Functionally, it serves as a higher-order control hub that integrates cognitive, emotional, and motivational information to guide motor behavior [15]. Although the PFC does not directly generate motor commands, it exerts top-down control over the motor cortex, limbic circuitry, basal ganglia, and cerebellum through extensive projections that shape motor intention, executive control, and motor learning [16]. The DLPFC communicates with M1 through intermediary regions such as the PMC and SMA, forming cognition–motor integration loops via corticocortical projections. Connections between Broca’s area and the PMC help transform abstract plans into concrete action sequences [17,18]. Functional imaging shows co-activation of the DLPFC and M1 during complex motor tasks (e.g., dual-task walking, fine manual control), with activation scaling with task difficulty and attentional demands [19]. Moreover, the PFC influences motor control indirectly through multiple subcortical circuits. The PFC–basal ganglia–thalamus–cortex circuit govern both the initiation and suppression of intentional movements [20], while bidirectional PFC–cerebellum connections play a crucial role in fine-tuning motor learning, timing, and rhythmic control [21]. Together, these anatomical and functional pathways constitute the structural basis by which emotional and cognitive factors modulate motor performance.

### *2.2 Emotional Modulation of Motor Execution, Learning, and Coordination*

This structural connectivity also manifests in the dynamic integration of emotional and motor systems. Emotion is an embodied response that mobilizes physiological arousal, autonomic function, and goal-directed motor control [22]. Emotional stimuli exert a direct and measurable influence on motor planning, execution, and feedback processing. For example, aversive objects elicit more cautious, precise grasp strategies, indicating that an object's emotional valence shapes affordance perception and action selection during motor planning [20]. Likewise, postural threat (e.g., anxiety when elevated) induces anticipatory adjustments in postural control, alters muscle synergies, and enhances sensorimotor vigilance [23].

At higher cognitive levels, emotional states strongly affect motor learning, adaptation, and inhibitory control. Motivation, an affective drive rooted in limbic circuitry, is essential for initiating and sustaining motor-skill learning [24]. Poorly motivated patients struggle with repetitive practice. Conversely, heightened arousal, whether positive or negative, can weaken inhibitory control, which is linked to the right inferior frontal gyrus [25]. Overall, emotion should be regarded as an active regulatory component that dynamically modulates processes from motor intention and execution to fine coordination, rather than a passive background state.

### *2.3 Pathophysiology of Post-stroke Emotional Disorders*

Post-stroke emotional disorders arise from multiple mechanisms, including psychological responses to sudden disability and loss of social roles, as well as structural damage to emotion-regulating networks (e.g., limbic and prefrontal regions) [11].

At the neurochemical level, stroke disrupts key neurotransmitter systems, especially including serotonin (5-HT), and the hypothalamic–pituitary–adrenal (HPA) axis, both of which play pivotal roles in the onset and progression of post-stroke depression (PSD) [26]. In addition to neurotransmitter dysregulation, molecular alterations further contribute to emotional disturbances. Molecules implicated in emotional disorders also regulate neuroplasticity. For instance, decreased expression of brain-derived neurotrophic factor (BDNF) not only predicts PSD but also compromises neuronal survival, dendritic remodeling, and synaptogenesis—processes essential for motor recovery [27]. Consequently, stroke-induced biological alterations not only exacerbate affective symptoms but also impair neural repair mechanisms, forming a self-perpetuating vicious cycle that hinders recovery. Therefore, therapeutic interventions aimed at improving mood and restoring neurochemical homeostasis may simultaneously enhance motor recovery and neuroplasticity, supporting the rationale for emotional stimulation as a complementary rehabilitation strategy.

## 3. Core Neuroplastic Mechanisms by Which Emotional Stimulation Facilitates Motor Recovery

### *3.1 Reward and Motivation: Central Roles of Dopaminergic Circuits*

Dopamine (DA) is pivotal to reward, motivation, motor control, and learning/memory [27]. In particular, dopaminergic activity serves as a neurochemical bridge linking emotional experience with motor performance. Mesocortical dopaminergic projections from the ventral tegmental area (VTA) to M1 are essential for acquiring new motor skills [27]. Reward prediction and attainment drive DA release, strengthening active synapses via long-term potentiation (LTP) and consolidating behavior.

Stroke often dampens dopaminergic function, contributing to low motivation [27]. This motivational deficit may result in reduced engagement in rehabilitation, thereby limiting neuroplastic remodeling. Emotional stimuli that elevate DA, such as rewarding audiovisual feedback in VR games, genuine therapist encouragement, or pleasant music, can boost participation and, at the molecular level, facilitate motor-memory formation. Such interventions not only improve patient compliance but also enhance synaptic efficiency within motor circuits, thereby accelerating functional recovery.

Clinically, combining dopaminergic agents (e.g., levodopa) with therapy may improve motor outcomes, likely by enhancing motor-skill encoding [28]. Emerging neuromodulation approaches, such as vagus nerve stimulation, may also aid recovery partly by modulating DA pathways [27]. Future studies integrating pharmacological, emotional, and neuromodulatory strategies could further clarify how dopaminergic regulation supports emotional-motor coupling after stroke.

### *3.2 Stress and Arousal: The HPA Axis and Noradrenergic System*

Stressful stimuli activate a cascade of neuroendocrine responses, notably the hypothalamic–pituitary–adrenal (HPA) axis and the locus coeruleus–norepinephrine (LC-NE) system [29–31]. Under acute stress conditions, the hypothalamus releases corticotropin-releasing hormone (CRH), which stimulates pituitary secretion of adrenocorticotropic hormone (ACTH), subsequently leading to adrenal cortisol production. In parallel, LC-NE activation rapidly elevates cortical arousal and reallocates attentional resources. These are mechanisms that can transiently improve information processing and motor learning [30,31].

However, stress effects are typically biphasic. Moderate, time-limited stress can enhance both NE and cortisol signaling, thereby focusing attention, increasing motivation, and accelerating early-phase motor learning [32–37]. Mechanistically, acute stress may promote LTP-like plasticity in prefrontal circuits via glucocorticoid receptor (GR)–dependent pathways that upregulate NMDA/AMPA receptor trafficking [33,34], transiently increase brain-derived neurotrophic factor (BDNF) expression [35], and augment axonal growth [36,37]. By contrast, excessive or chronic stress impairs neurogenesis, disrupts prefrontal function, and synaptic plasticity, and worsens emotion regulation [38]. Therefore, in clinical or rehabilitation contexts, the intensity, duration, and timing of stress exposure must be precisely calibrated to optimize functional outcomes and avoid maladaptive neuroplasticity.

### *3.3 Learning and Growth: BDNF and Synaptic Plasticity*

BDNF supports neuronal survival, neurogenesis, differentiation, and synaptic plasticity (particularly LTP) [39]. Following a stroke, decreased circulating or central BDNF levels are associated with worse outcomes and a higher risk of depression [26]. Importantly, BDNF expression is not fixed but dynamically modulated by behavioral and emotional states. Aerobic exercise is among the most effective ways to increase BDNF [39]. In addition, positive social connection, stress management (e.g., mindfulness), and sufficient sunlight exposure may also help. Emotional-stimulation interventions may leverage these pathways. For example, aromatherapy or relaxation can reduce stress and indirectly increase BDNF, whereas group-based music therapy or gamified rehabilitation foster social engagement [40]. Collectively, these interventions are not merely adjuncts; they help establish a molecular environment that promotes synaptic remodeling, neural network reorganization, and ultimately functional recovery.

## 4. Applications of Emotional Stimulation in Post-stroke Motor Rehabilitation

### *4.1 Sensory-Based Emotional Stimulation*

These interventions aim to modulate emotional and physiological states through specific sensory channels (e.g., auditory, olfactory) and integrate this modulation with motor tasks, representing an emerging approach that bridges emotional regulation and motor recovery—an initial synthesis of emotion and rehabilitation.

#### *4.1.1 Music Therapy: Integrating Rhythm, Melody, and Emotion*

Music therapy exemplifies dual action on emotion and motor control. Among its modalities, rhythmic auditory stimulation (RAS), using metronomic beats or rhythmically salient music, has robust evidence, including Cochrane reviews and multiple meta-analyses, for improving gait parameters (speed, cadence, step length) and upper-limb function after stroke [41]. Specifically, RAS enhances movement timing and coordination in both lower- and upper-limb tasks [42]. Mechanistically, auditory–motor entrainment synchronizes the motor system to external rhythms, providing a temporal scaffold that aids coordination and fluency when internal timing generators are compromised [40]. Beyond timing, musical structure and affective content engage limbic circuits, evoke pleasure, reduce anxiety, and activate dopaminergic reward pathways, thereby enhancing motivation and adherence to repetitive training [13,44]. Consequently, music therapy offers not only rhythmic entrainment but also emotional reinforcement, combining temporal cues with affective incentives and highlighting the synergy between emotion and motor rehabilitation.

#### *4.1.2 Olfactory Stimulation (Aromatherapy): Exploratory Applications and Mechanisms*

Olfactory inputs have a unique neuroanatomical pathway, as they project directly to limbic structures (amygdala, hippocampus) without the thalamic relay, allowing odors to exert rapid and profound influences on emotion and memory [45]. Preclinical studies suggest that certain essential oils (e.g., lavender, rosemary) confer neuroprotection in ischemic models by reducing infarct size and oxidative stress [46]. In clinical contexts, human studies and systematic reviews indicate that aromatherapy can alleviate pain, anxiety, and depression and improve sleep quality among stroke survivors [20]. These therapeutic effects are thought to arise from the modulation of limbic function, rebalancing of neurotransmitters (dopamine and serotonin), and reduction of stress hormones such as cortisol [45]. Although direct evidence for motor improvement remains scarce, aromatherapy may serve as a beneficial adjunctive approach, either as a preconditioning intervention to optimize emotional and physiological states before rehabilitation or as a complementary therapy to enhance patient engagement and compliance during training sessions.

### *4.2 Technology-Driven Novel Emotional Interventions*

Advances in information technology, sensing, and neuroscience have enabled interventions with greater immersion, interactivity, and precision. These technological innovations not only enhance motor training but also allow for the integration of emotional and motivational components, thereby optimizing rehabilitation outcomes.

#### *4.2.1 Virtual Reality (VR): An Immersive, Task-Oriented Paradigm*

Virtual reality (VR), delivered via head-mounted displays and motion tracking, creates safe, controllable, repeatable, and ecologically valid 3-D environments [47]. Recent studies, including umbrella reviews and meta-analyses, report that both commercial VR games and customized systems improve upper-limb motor function and balance, performing as well as or better than conventional therapy [47-49]. Beyond motor outcomes, VR interventions have also been shown to enhance patient engagement and emotional well-being, which are critical determinants of rehabilitation adherence. VR aligns core neurorehabilitation principles with affective drivers: it offers engaging, task-oriented content that increases motivation and enables high-dose, high-intensity repetition; it provides immediate, quantitative multisensory feedback with rewarding audiovisual cues that likely engage dopaminergic circuits; and its immersion may reduce anxiety and fear of failure by focusing attention on in-world tasks. VR therefore exemplifies a paradigm of emotion-driven motor rehabilitation, demonstrating how emotionally enriched, technology-based interventions can promote both neural and behavioral recovery after stroke.

#### *4.2.2 Brain–Computer Interfaces (BCI): Closed-Loop Neurofeedback and Affective Modulation*

BCIs record cortical activity (e.g., EEG) to decode motor intention (e.g., imagined limb movement) and translate it into control commands for external devices (robotic orthoses, virtual avatars, or functional electrical stimulation) with real-time sensory feedback [50,51]. By establishing a bidirectional, closed-loop that bypasses damaged pathways, BCIs enable patients to "see" or "feel" intended movements, thereby delivering a strong internal reward that reinforces a sense of agency and self-efficacy. This precise pairing of motor intention and sensory outcome adheres to Hebbian principles ("neurons that fire together wire together") and efficiently strengthens motor-intention networks. Meta-analyses have demonstrated clinically significant improvements in upper-limb function, particularly in moderate-to-severe impairment [52,53]. Beyond motor restoration, emerging evidence suggests that BCI-induced feedback can also modulate emotional and motivational states, further enhancing neuroplastic adaptation [54]. Future systems are therefore expected to integrate affective computing and closed-loop neurofeedback, allowing continuous monitoring and adaptive modulation of affect-related neural signatures to achieve a comprehensive rehabilitation strategy encompassing motor, cognitive, and emotional domains.

## 5. Summary and Outlook

### *5.1 Current Limitations and Methodological Challenges*

Despite promising prospects, the overall quality of evidence remains limited. Many existing clinical trials, especially those involving novel technologies, have small sample sizes, limiting statistical power and external validity [55]. Moreover, heterogeneity in populations (stroke stage, severity), interventions (e.g., VR type, dose, frequency), and outcome measures complicates cross-study comparisons and meta-analysis [47]. Blinding is inherently difficult in behavioral and psychological interventions, which may introduce both performance and detection bias [55]. Largely, standardized randomized controlled trials are needed to facilitate translation of research evidence into clinical practice.

### *5.2 Future Directions: Toward Multimodal, Personalized, and Intelligent Rehabilitation*

Future stroke rehabilitation paradigms will be multimodal, personalized, and intelligent. Effective interventions must be grounded in a systematic and dynamic understanding of each patient's unique neurological, psychological, and functional status. Multimodal integration, combining clinical scales (e.g., FIM, FMA), cognitive-psychological assessments, neurophysiology (EEG, TMS-based cortical excitability), neuroimaging (fMRI, DTI), and molecular biomarkers (e.g., blood BDNF, miRNA), can generate a comprehensive data profile to inform prognostic models and early therapy selection [56-58]. Precision rehabilitation will tailor intervention type, dose, intensity, and timing to individual biology and behavior [7,59], leveraging the critical window in the first weeks to months after stroke to maximize gains [60]. Ultimately, an ethically grounded, human-centered, technology-enabled digital-therapeutics ecosystem should emerge, in which emotion is precisely guided and leveraged as a facilitator of neuroplasticity and motor relearning rather than a barrier to recovery.

**6. Conclusion**

Emotional stimulation represents a promising paradigm shift in post-stroke motor rehabilitation, moving beyond traditional biomechanical approaches to leverage emotion as an active driver of neuroplasticity and motor recovery. Despite the current methodological limitations, future progress lies in developing personalized, multimodal, and intelligent rehabilitation systems that dynamically integrate emotional states into therapeutic strategies. By bridging affective experience with motor relearning, this approach not only optimizes functional recovery but also restores meaning and agency to the rehabilitation process, paving the way for more holistic and human-centered care.

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