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Analysis of the Principles and Potential Effects of BCI Applications in Mental Disorders

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Abstract. Brain-computer interfaces (BCIs) are increasingly being explored in clinic settings, with a growing number of studies investigating their feasibility and underlying mechanisms. This paper reviews how BCIs translate neural activity into therapeutic feedback to restore or augment function in mental and neurological disorders. After outlining core closed-loop principles of BCIs, including signal acquisition, feature extraction, and intention-contingent feedback, as well as basic mechanisms of three situations - stroke, ADHD, and addiction - we synthesize evidence. In stroke, EEG motor-imagery (MI) BCIs that trigger robotics or functional electrical stimulation (FES) pair cortical intent with congruent proprioceptive input, yielding clinically meaningful upper-limb gains. In ADHD, neurofeedback targeting oscillations (theta/beta, SMR), slow cortical potentials, or prefrontal hemodynamics shows learnability and symptom reductions in some studies, though meta-analyses report mixed effects on blinded ratings. In addition, real-time fMRI and EEG paradigms reduce cue-reactivity and in-scanner craving by down-modulating ACC/insula activity or cue-specific EEG patterns. Across areas, effect sizes depend on contingency, dose, and protocol fidelity. Key challenges are discussed, including evidence quality and user variability. This paper proposes standardized outcomes, learning verification, and precision-medicine stratification to guide who receives which BCI and how it integrates with conventional care.

Keywords: BCIs, Stroke, ADHD, Addiction

1. Introduction

A brain-computer interface (BCI) is a system that detects and interprets activity from the central nervous system (CNS) and converts it into artificial outputs. These outputs can replace, restore, enhance, or supplement natural brain functions, allowing the brain to interact more effectively with the body or the external environment. In simpler terms, a BCI is a tool that translates brain signals into useful commands or actions [1]. Originally developed for motor restoration, BCIs have rapidly expanded toward and shown promise in neuropsychiatric care, where altered network dynamics and impaired self-regulation are core features across diagnoses.

A considerable number of studies have been conducted to explore the application of BCIs in medical treatment. There are two primaries clinical BCI applications: assistive technologies targeting the recovery of functional deficits, and rehabilitation technologies that work to promote

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motor recovery through neuroplastic changes induced by neural modulation or self-regulation. For stroke, BCIs that pair motor-imagery detection with contingent feedback or assistive actuation (e.g., functional electrical stimulation or robotics) can enhance plasticity and improve upper-limb outcomes. Cognition and emotion of stroke patients might be improved as well [2]. In patients living with attention-deficit/hyperactivity disorder (ADHD), an individualized BCI attention-training game calibrated by Stroop performance produced significant improvements in inattentive and hyperactive-impulsive symptoms, suggesting that closed-loop training can reshape attention networks [3]. In terms of mild cognitive impairment (MCI), a BCI-VR-AI diagram is proposed to deliver adaptive assessment and personalized cognitive training, allowing the synchronization of cognitive demands with neural targets in real time [4]. BCIs have been applied to down-regulate cue-reactivity and craving, causing positive outcomes in treating substance-use disorders, like smoking and drug dependence.

Taken together, these strands motivate a focused review of how BCIs can be purposefully designed for mental health care. This paper aims to provide a clear framework for the mechanisms of clinical BCI applications nowadays, limitations, and prospective directions.

2. Basic theoretical analysis

2.1. Principles of BCI-based treatment for psychiatric disorders

As mentioned in the introduction part, a BCI acquires neural activity and translates it into outputs that can replace, restore, enhance, or supplement function, thereby altering how the brain interacts with the body or external devices. Operationally, most systems follow a closed-loop pipeline: electrophysiological signals from the brain are obtained by electrodes. Then these signals are processed and extracted into specific signal features. These features represent users' objectives, and those that the user can control will be selected and translated by BCI into device commands.

Now comes the question: "Where do BCIs operate?" BCIs utilize non-invasive sensors, such as scalp EEG, capturing millisecond-scale rhythms and signals originating outside the brain. Other sensors include near-infrared systems (fNIRS) or functional magnetic resonance imaging (fMRI)/magnetoencephalography (MEG), which collect hemodynamic or magnetic signals informed by changes in intracranial blood flow, and electrocorticography (ECoG) or penetrating electrodes. Signals detected by these sensors incorporate slow cortical potentials, P300 potentials, mu/beta rhythms, and cortical neuronal activity.

2.2. Stroke

Stroke is an acute focal injury of the central nervous system (CNS) caused by abnormal vascular events, including cerebral infarction, intracerebral hemorrhage (ICH), and subarachnoid hemorrhage (SAH), producing neurological deficits that could further lead to disability or even death. Central nervous system infarction can be induced by ischemia that leads to cell death in the brain, spinal cord, or retina. While the ischemic stroke is diagnosed when both CNS infarction and overt symptoms co-occur, there are no identifiable symptoms to date secondary to silent infarction.

2.2.1. Ischemic stroke: the ischemic cascade and penumbra

An acute ischemic stroke (AIS) begins with cerebral vascular occlusion (thrombus/embolus) that sharply reduces blood flow in the downstream parenchyma, creating an irreversibly injured core surrounded by a penumbra and oligemia. The penumbra is a severely hypoperfused tissue that is

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functionally impaired yet potentially salvageable if reperfused promptly; the oligemia is mildly hypoperfused, maintaining neuronal function. When the local cerebral blood flow drops below the level needed to deliver oxygen and glucose, energy failure impairs ion pumps, promoting excess glutamate release and over-activation of N-methyl-D-aspartate (NMDA) and alpha-amino-3-hydroxy-5-methyl-4-propionate (AMPA) receptors. Resultant Na^{+}/Ca^{2+} influx depolarizes neurons and drives the glutamate–calcium cascade, where Ca^{2+}-mediated excitotoxity is a key driver of infarction, contributing to a necrotic lesion. Ca^{2+} overload and mitochondrial dysfunction amplify oxidative/nitrosative stress, while blood–brain barrier compromise, microvascular dysfunction, and inflammation extend injury into peri-infarct zones [5].

2.2.2. Hemorrhagic stroke: Intracerebral Hemorrhage (ICH) and Subarachnoid Hemorrhage (SAH)

In ICH, primary damage arises from the mass effect of the hematoma and intracranial pressure surges. Over the next hours to days, secondary injury develops as thrombin from clotting and hemoglobin that breaks down into heme and iron accumulate around the hematoma. These blood-derived products trigger oxidative stress and inflammation, disrupt the blood–brain barrier (BBB), and drive perihematomal edema and neurotoxicity in adjacent tissue. In SAH, early brain injury includes microvascular dysfunction, BBB disruption, neuroinflammatory responses, cerebral edema, oxidative cascades, and neuronal death [6]. In the subsequent days, delayed cerebral ischemia (DCI) develops, driven by a multifactorial combination of microvascular spasm, impaired autoregulation, inflammatory processes, and cortical spreading depolarizations [7].

2.3. Attention-Deficit/Hyperactivity Disorder (ADHD)

ADHD is a common and often debilitating neurodevelopmental condition defined by persistent, developmentally inappropriate levels of hyperactivity, inattention, and impulsivity. It has multifactorial causes that span genes, neurotransmitters, brain circuits, and cognitive processes.

2.3.1. Genetic and molecular mechanisms

Family and twin designs indicate high heritability of about 70–80%, and modern Genome-Wide Association Study (GWAS) show that ADHD risk is highly polygenic. 12 genome-wide significant loci were identified by GWAS, and 27 loci showed enrichment in genes associated with early neurodevelopment [8, 9].

2.3.2. Circuit and network mechanisms

At the systems level, ADHD involves alterations in fronto-striato-thalamo-cortical loops that support action selection, reinforcement learning, and cognitive control. One study found small but reliable volume reductions in the accumbens, caudate, putamen, hippocampus, and amygdala [10]. Additionally, many studies have reported reduced task-related suppression and altered coherence of the Default Mode Network (DMN), involved in internally directed thought and self-referential processing (suppressed during task demands), consistent with “DMN interference” into task-positive control networks [11, 12]. For instance, adults with ADHD exhibited higher variability in DMN activation and diminished ability to sustain DMN suppression [12].

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2.3.3. Delayed cortical maturation

According to [13], children with ADHD reach peak cortical thickness later than peers, especially in prefrontal regions, which play a vital role in the regulation of cognitive processes. Another study suggested that ADHD is associated with significant delays in connections within the DMN, as well as between the DMN and two task-positive networks (TPNs): the Frontoparietal Network (FPN) that supports executive attention and cognitive control, and the Ventral Attention Network (VAN) responsible for detecting salient external stimuli [14].

2.4. Addiction

2.4.1. Incentive sensitization

Repeated drug exposure sensitizes mesolimbic dopamine systems, resulting in excessive motivational salience of the drug cues, which lead to craving, attention attraction, and biased choice. This is caused by cue-evoked phasic dopamine release and associated plasticity within the ventral striatum (nucleus accumbens) and its connected cortico-striatal loops, contributing to stable, time-locked neural states that are relatively easy to detect [15].

2.4.2. Shift in behavioral control

Addiction is increasingly understood as a progressive transition in behavioral control. In its early stages, drug use is often goal-directed, which means that the individual consciously seeks the rewarding effects of the substance. At this point, neural activity is primarily mediated by the ventral striatum, which integrates motivational signals and reinforces behaviors that achieve desired outcomes. However, with repeated exposure, drug seeking becomes increasingly habitual, driven less by conscious goals and more by automatic patterns of behavior. This transition reflects a growing reliance on the dorsal striatum, which is critical for habit learning and procedural memory [16].

2.4.3. Loss of prefrontal cortical control

The prefrontal cortex (PFC) is responsible for executive functions, including planning and self-control. As habitual drug use develops, the PFC's control over behaviors weakens. This attenuation of top-down inhibition allows striatal systems, particularly the dorsal striatum, to dominate, promoting compulsive drug seeking even in the face of adverse outcomes [16]. Therefore, compromised PFC function is central to the shift from voluntary to compulsive use, as it reduces the brain's capacity to suppress urges or flexibly redirect behavior given negative consequences.

3. Application of BCI-based treatments

3.1. Stroke rehabilitation

In stroke treatment, motor intent is typically detected by BCIs. In a study [17], an EEG motor-imagery (MI) BCI was applied to monitor sensorimotor activity when a patient imagines or attempts arm movement. This system provides feedback that pairs cortical intent with actual movement by triggering the MIT-Manus shoulder-elbow robot to move the paretic limb when detecting the target EEG pattern. Coupling BCI with the MIT-Manus robot allows patients to achieve similar functional

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gains with fewer repetitions per session. Also, more BCI-Manus patients showed continued improvement at week 12, quantified by the Fugl-Meyer Motor Assessment (FMMA) score – a standardized stroke scale that measures motor impairment [17]. Another study further showed that motor imagery-contingent BCI training significantly improved wrist strength, range of motion, and motor-related brain connectivity in chronic stroke patients, with functional gains closely linked to neural plasticity changes [18]. Moreover, BCI can be used with functional electrical stimulation (FES) to provide long-lasting benefits (6~12 months) than sham FES and contribute to an increased ipsilesional functional connectivity [19]. These evidences all support BCIs as a promising tool for stroke rehabilitation.

While most studies concerning BCIs' implication in stroke focused on upper-limb motor recovery, lower-limb BCIs are emerging. They decode gait intent to trigger FES or exoskeleton-assisted stepping. For instance, Sebastián-Romagosa et al. reported long-lasting improvements in gait speed and the feasibility of combining EEG-BCI with robotic gait trainers [19].

3.2. DHD - closed-loop neurofeedback

BCIs for ADHD are typically delivered as closed-loop neurofeedback, where real-time brain signals are translated into immediate rewards for learners to practice stabilizing neural activity that supports attention and inhibitory control [20, 21]. Target signals could be EEG rhythms (theta/beta, SMR-sensorimotor rhythm), EEG slow cortical potentials (SCPs), or hemodynamic activity in the prefrontal cortex via real-time fMRI (often right inferior frontal cortex) or portable fNIRS (dorsolateral PFC). Early randomized trials reported that children can learn to modulate the targeted signals and, in some studies, show symptom reductions that persist at follow-up [21, 22]. SCP neurofeedback has also shown superiority to a semi-active control in a multicenter randomized controlled trial [20]. Taken together, these findings frame BCI for ADHD as a mechanistically plausible and learnable approach to self-regulation that holds promise for certain patient subgroups. At the same time, they highlight the need for standardized protocols, objective measures of learning, and precision-medicine strategies to guide participant stratification in future trials [20].

3.3. Addiction

BCIs for addiction are typically implemented as neurofeedback systems, which measure neural markers of craving or control in real time and provide feedback through visual, auditory, or game-based cues. It aims to weaken cue-reactive loops (e.g., in the insula, anterior cingulate cortex [ACC], or ventral striatum) and strengthen top-down control (dorsolateral prefrontal cortex-dlPFC/ACC). Reviews across multiple substances indicated that individuals with addictions can indeed learn to modulate these neural signals, and that reductions in cue-reactivity often coincide with acute craving reductions, although the durability of these effects varies depending on the protocol and study quality [23-25]. Mechanistically, rtfMRI-neurofeedback studies illustrated down-modulation of blood-oxygenation-level-dependent (BOLD) responses in craving circuits during cue exposure, while EEG studies demonstrated state-specific modulation of oscillations (theta/alpha) linked to craving and vigilance [24, 26, 27].

Real-time fMRI neurofeedback (rtfMRI-NF) was shown to negatively affect craving behaviors. In tobacco use disorder, several studies illustrated that smokers can volitionally decrease cue-evoked activation in ACC [28]. Follow-ups using individualized targets likewise indicated attenuation of craving-related activation with practice [5].

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EEG-based neurofeedback uses both traditional alpha–theta training and modern pattern-based protocols. Historically, Alpha-theta EEG neurofeedback has served as a complementary treatment for chronic alcohol dependence, with small trials suggesting reduced craving and improved mood [29]. More recent pattern-based EEG neurofeedback directly targets the spatiotemporal EEG signature of smoking cue reactivity; training to suppress these patterns has produced acute craving reductions during sessions [26].

4. Challenges

Across indications, many BCI trials remain small, heterogeneous, or only partially blinded, which complicates causal inference and inflates variance in effect estimates. For ADHD neurofeedback, a 38-RCT meta-analysis reported no meaningful advantage over controls on probably-blinded total-symptom ratings at the group level, even though small effects appeared when restricting to "standard" EEG protocols. This highlighted sensitivity to protocol and rater masking [30]. In substance-use disorders, mixed clinical outcomes and a shortage of large, rigorously blinded trials were noted [25]. Stroke meta-analyses similarly show modest, variable gains that depend on pairing (e.g., FES/robotics), dosing, and controls; immediate functional improvements were reported, but superiority over active comparators (e.g., robot-only therapy) was inconsistent [30].

A substantial minority of users struggle to achieve reliable BCI control with standard mental-imagery paradigms, often termed "BCI illiteracy." Predictors span cognitive and psychological domains (attention, spatial abilities, sense of agency/technology anxiety) as well as task/feedback design, suggesting that who is trained and how they are trained materially shape outcomes.

5. Conclusion

BCIs offer a mechanistically targeted route to rehabilitation and self-regulation by reinforcing neural states that support motor control and executive function. The strongest clinical signal currently arises when cortical intent is tightly coupled to immediate sensory/motor consequences (e.g., BCI-robotics/FES in stroke), with convergent neurophysiological evidence of ipsilesional recruitment and connectivity change. For ADHD and addiction, consistent target engagement (signal modulation, reduced cue-reactivity) coexists with heterogeneous clinical outcomes, highlighting the need for larger, blinded RCTs with durable, real-world endpoints. Moving from promise to practice will require: (i) standardized protocols and outcome sets, (ii) verification that patients learn the intended neural skill, (iii) tailoring based on baseline neurobiology to reduce "BCI illiteracy," and (iv) thoughtful integration with established therapies and home-based delivery to increase dose and access. With these steps, BCIs can mature from feasibility tools to scalable, evidence-based components of routine neuropsychiatric care.

References

[1] Cervera, M. A., Soekadar, S. R., Ushiba, J., et al. (2018). Brain-computer interfaces for post-stroke motor rehabilitation: A meta-analysis. Annals of Clinical and Translational Neurology, 5(5), 651.
[2] Mane, R., Chouhan, T., & Guan, C. (2020). BCI for stroke rehabilitation: Motor and beyond. Journal of Neural Engineering, 17(4), 041001.
[3] Lim, C. G., Lee, T. S., Guan, C., et al. (2012). A brain-computer interface based attention training program for treating attention deficit hyperactivity disorder. PLoS ONE, 7(10), e46692.
[4] American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). American Psychiatric Publishing.

DOI: 10.54254/2755-2721/2025.GL27988

[5] Rehman, S., Nadeem, A., Akram, U., et al. (2024). Molecular mechanisms of ischemic stroke: A review integrating clinical imaging and therapeutic perspectives. Biomedicines, 12(4), 812.
[6] Lauzier, D. C., Jayaraman, K., Yuan, J. Y., et al. (2023). Early brain injury after subarachnoid hemorrhage: Incidence and mechanisms. Stroke, 54(5), 1426-1440.
[7] Dodd, W. S., Laurent, D., Dumont, A. S., et al. (2021). Pathophysiology of delayed cerebral ischemia after subarachnoid hemorrhage: A review. Journal of the American Heart Association, 10(15), e021845.
[8] Demontis, D., Walters, G. B., et al. (2019). Discovery of the first genome-wide significant risk loci for ADHD. Nature Genetics, 51(1), 63–75.
[9] Demontis, D., Walters, G. B., et al. (2023). Genome-wide analyses of ADHD identify 27 risk loci and refine genetic architecture. Nature Genetics, 55(2), 198–208.
[10] Hoogman, M., Bralten, J., et al. (2017). Subcortical brain volume differences in ADHD across the lifespan: An ENIGMA mega-analysis. The Lancet Psychiatry, 4(4), 313–324.
[11] Raichle, M. E., MacLeod, A. M., Snyder, A. Z., et al. (2001). A default mode of brain function. Proceedings of the National Academy of Sciences of the United States of America, 98(2), 676–682.
[12] Mowinckel, A. M., Alnæs, D., Pedersen, M. L., et al. (2017). Increased default-mode variability is related to reduced task-performance and is evident in adults with ADHD. NeuroImage: Clinical, 16, 369–382.
[13] Shaw, P., Eckstrand, K., Sharp, W., et al. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. Proceedings of the National Academy of Sciences of the United States of America, 104(49), 19649-19654.
[14] Sripada, C. S., Kessler, D., & Angstadt, M. (2014). Lag in maturation of the brain's intrinsic functional architecture in attention-deficit/hyperactivity disorder. Proceedings of the National Academy of Sciences of the United States of America, 111(39), 14259–14264.
[15] McClure, S. M., Berns, G. S., & Montague, P. R. (2003). Temporal prediction errors in a passive learning task activate human striatum. Neuron, 38(2), 339–346.
[16] Everitt, B. J., & Robbins, T. W. (2016). Drug addiction: Updating actions to habits to compulsions ten years on. Annual Review of Psychology, 67(1), 23–50.
[17] Ang, K. K., Chua, K. S. G., Phua, K. S., et al. (2015). A randomized controlled trial of EEG-based motor imagery brain-computer interface robotic rehabilitation for stroke. Clinical EEG and Neuroscience, 46(4), 310-320.
[18] Kim, M. S., Park, H., Kwon, I., et al. (2025). Efficacy of brain-computer interface training with motor imagery-contingent feedback in improving upper limb function and neuroplasticity among persons with chronic stroke: A double-blinded, parallel-group, randomized controlled trial. Journal of NeuroEngineering and Rehabilitation, 22(1), Article 1.
[19] Biasiucci, A., Leeb, R., Iturrate, I., et al. (2018). Brain-actuated functional electrical stimulation elicits lasting arm motor recovery after stroke. Nature Communications, 9, Article 2421.
[20] Strehl, U., Aggensteiner, P., Wachtlin, D., et al. (2017). Neurofeedback of slow cortical potentials in children with attention-deficit/hyperactivity disorder: A multicenter randomized trial controlling for unspecific effects. Frontiers in Human Neuroscience, 11, Article 135.
[21] Gevensleben, H., Holl, B., Albrecht, B., et al. (2009). Distinct EEG effects related to neurofeedback training in children with ADHD: A randomized controlled trial. International Journal of Psychophysiology, 74(2), 149–157.
[22] Gevensleben, H., Holl, B., Albrecht, B., et al. (2010). Neurofeedback training in children with ADHD: 6-month follow-up of a randomised controlled trial. European Child & Adolescent Psychiatry, 19(9), 715-724.
[23] Karch, S., Paolini, M., Gschwendtner, S., Jeanty, H., et al. (2019). Real-time fMRI neurofeedback in patients with tobacco use disorder during smoking cessation: Functional differences and implications of the first training session in regard to future abstinence or relapse. Frontiers in Human Neuroscience, 13, Article 65.
[24] Martz, M. E., Brocke, J., Zotev, V., et al. (2020). A review of real-time fMRI neurofeedback studies: Trends and future directions. NeuroImage: Clinical, 28, Article 102301.
[25] Murphy, E., Thomson, H., Dakhili, A. H., et al. (2024). Real-time fMRI-based neurofeedback to restore brain function in substance use disorders: A systematic review of the literature. Neuroscience & Biobehavioral Reviews, 150, 107360.
[26] Bu, J., Young, K. D., Hong, W., et al. (2019). Effect of deactivation of activity patterns related to smoking cue reactivity on nicotine addiction. Brain, 142(6), 1827-1841.
[27] Hartwell, K. J., Hanlon, C. A., Li, X., et al. (2015). Individualized real-time fMRI neurofeedback to attenuate craving in nicotine-dependent smokers. Journal of Psychiatry & Neuroscience, 41(1), 48-55.
[28] Canterberry, M., Hanlon, C. A., Hartwell, et al. (2013). Sustained reduction of nicotine craving with real-time neurofeedback: Exploring the role of severity of dependence. Nicotine & Tobacco Research, 15(12), 2120-2124.

DOI: 10.54254/2755-2721/2025.GL27988

[29] Peniston, E. G., & Kulkosky, P. J. (1989). Alpha-theta brainwave training and beta-endorphin levels in alcoholics. Alcoholism: Clinical and Experimental Research, 13(2), 271-279.
[30] Westwood, S. J., Aggensteiner, P., Kaiser, A., et al. (2025). Neurofeedback for attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. JAMA Psychiatry, 82(2), 118-129.