

1 ²Using EEG-based Brain Computer Interface and Neurofeedback
2 Targeting Sensorimotor Rhythms to Improve Motor Skills:
3 Theoretical background, applications and prospects

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5 Running title: Neurofeedback targeting sensorimotor rhythms

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1 **Abstract**

2 Many Brain Computer Interface (BCI) and neurofeedback studies have investigated the impact
3 of sensorimotor rhythm (SMR) self-regulation training procedures on motor skills enhancement
4 in healthy subjects and patients with motor disabilities. This critical review aims first to
5 introduce the different definitions of SMR EEG target in BCI/Neurofeedback studies and to
6 summarize the background from neurophysiological and neuroplasticity studies that led to
7 SMR being considered as reliable and valid EEG targets to improve motor skills through
8 BCI/neurofeedback procedures. The second objective of this review is to introduce the main
9 findings regarding SMR BCI/neurofeedback in healthy subjects. Third, the main findings
10 regarding BCI/neurofeedback efficiency in patients with hypokinetic activities (in particular,
11 motor deficit following stroke) as well as in patients with hyperkinetic activities (in particular,
12 Attention Deficit Hyperactivity Disorder, ADHD) will be introduced. Due to a range of
13 limitations, a clear association between SMR BCI/neurofeedback training and enhanced motor
14 skills has yet to be established. However, SMR BCI/neurofeedback appears promising, and
15 highlights many important challenges for clinical neurophysiology with regards to therapeutic
16 approaches using BCI/neurofeedback.

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18 **Key words**

19 Neurofeedback; Motor ability; Brain Computer Interface; ADHD; Stroke; Closed loop.

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1 INTRODUCTION

2 Neurofeedback is a neurophysiological technique that aims to teach users/patients to self-
3 regulate targeted brain activity patterns in order to specifically enhance cognitive abilities or
4 reduce clinical symptoms. The choice of target brain activity patterns is thus a key issue, in
5 order for neurofeedback procedures to be efficient. From the multitude of EEG targets [61],
6 sensorimotor rhythms (SMR) appear to be a very promising and interesting neurophysiological
7 target to try to enhance motor skills. Many Brain Computer Interface (BCI) and neurofeedback
8 studies have investigated the impact of SMR self-regulation training procedures on motor skills
9 enhancement in healthy subjects and patients with motor disabilities. This critical review aims
10 first to introduce the neurophysiological framework, in line with the motor imagery literature,
11 which led to SMR being considered as a reliable and valid EEG target to improve motor skills
12 through BCI/neurofeedback procedures. The different definitions of SMR target in
13 BCI/neurofeedback studies will be introduced and the relationship between neuroplasticity,
14 motor skills and SMR BCI/neurofeedback training discussed. The second objective of this
15 review is to introduce the main findings regarding SMR BCI/neurofeedback in healthy subjects.
16 The impact of such procedures on sport, acting and surgical skills will be analyzed. Third, the
17 main findings regarding SMR BCI/neurofeedback efficiency in patients with hypokinetic
18 activities (in particular motor deficit following stroke) as well as in patients with hyperkinetic
19 activities (in particular Attention Deficit Hyperactivity Disorder, ADHD) will be introduced.
20 This review is not meant to be a systematic, exhaustive review. Rather, it aims to propose a
21 critical synthesis of the existing literature from a clinical neurophysiological point of view.
22 Indeed, the opinions within the scientific and medical community are divided regarding the
23 efficacy of BCI/neurofeedback. Most of the randomized clinical trials show significant
24 weaknesses and do not enable us to clearly conclude on the efficacy of BCI/neurofeedback
25 procedures, since the level of evidence remains too low. However, when compared to other
26 potential EEG targets, SMR present the advantage of having a relatively well-identified
27 neurophysiological relationship with motor imagery and motor skills. On the one hand, this
28 characteristic results in the fact that SMR are considered as very interesting targets to better
29 understand motor skills acquisition in various contexts. On the other hand, SMR also represent
30 promising targets for the future development of innovative neurophysiological treatments.

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1 **BCI/NEUROFEEDBACK TARGETING SENSORIMOTOR RHYTHMS FOR** 2 **ENHANCING NEUROPLASTICITY**

3 *Neuroplasticity and the acquisition of motor skills*

4 Neuroplasticity is a normal ongoing state of the human brain. It refers to the ability of the latter
5 to evolve its structure and function. This reorganization is observed from the molecular level
6 to the behavioral level [41, 45, 46, 75]. One impressive example of neuroplasticity is the
7 possibility of acquiring motor skills. This learning process involves and modifies the activity
8 of specific brain areas such as the dorsolateral prefrontal cortex (DLPFC), the primary motor
9 cortex (M1), the premotor cortex (PM), the pre-supplementary motor area (preSMA), the
10 supplementary motor area (SMA), the primary somatosensory cortex (S1) and the posterior
11 parietal cortex (PPC), striatum and cerebellum (for a review, see [25]). The progression from
12 early to late stages of motor skill learning seems to be associated with an activation from
13 anterior to more posterior regions of the brain [30], which reflects a progressive decrease in
14 reliance on attentional resources and executive functions towards more automatic processes
15 [45]. Proof of the plasticity induced by motor skill training and learning is visible both in
16 structural (anatomic) and functional changes.

17 Anatomical changes are visible using magnetic resonance imaging (MRI) or diffusion tensor
18 imaging (DTI). For instance, voxel-based morphometry (VBM) analyses suggest that musicians
19 show higher grey matter density in the sensorimotor cortex and cerebellum than control subjects
20 [39]. In addition, DTI analyses reveal higher fractional anisotropy in the internal capsule as
21 well as plastic changes in the 3D morphology of the central sulcus in response to long-term
22 motor skill training [39, 49]. Other studies investigating structural plasticity reported an
23 increased volume of grey matter density in brain areas involved in the task. These changes seem
24 to disappear when participants stop practicing, suggesting that structural plasticity is possible
25 in all directions (for a review, see [16]).

26 Functional changes are visible using transcranial magnetic stimulation (TMS), functional MRI
27 or electroencephalography (EEG). For instance, the cortical representation of the hand,
28 explored using TMS, is larger in professional racquetball players than in control participants
29 [76]. Other findings suggest that this functional reorganisation is led by an enlargement or
30 focused activation of the motor area involved in the control of the studied skill. Neuroplasticity
31 also occurs in cognitive and perceptual domains associated with improved performances. For
32 example, strong coupling of sensorimotor and auditory processing has been revealed in
33 professional musicians (for a review, see [16]).

1 However, even though neuroplasticity is probably related to cellular and molecular
2 mechanisms, one of the main challenges is to link human brain imaging findings to the
3 underlying molecular events that have been well documented in animal models. These models
4 report modulations of synaptic connections through long-term potentiation (LTP) and long-
5 term depression (LTD) in rodents [83, 84]. In humans, learning to perform a motor task also
6 seems to modulate LTP-like plasticity [87, 113], particularly in M1 via the monoaminergic
7 modulation of motor systems (for a review, see [105]. Both in humans and animal models,
8 brain derived neurotrophic factor (BDNF), a neurotrophin, also participates in this synaptic
9 plasticity [1, 51]. These mechanisms are followed by other molecular and synaptic changes in
10 order to induce automatic behaviours. For example, the involvement of neurons in the
11 sensorimotor striatum during late stages of learning has been shown in animal models, probably
12 via long lasting changes in glutamatergic neurotransmission. It might be partially part of a
13 substrate that enables the acquisition of automatic behaviours [66, 111].

14 *The acquisition of motor skills and motor imagery*

15 A very interesting method that favors neuroplasticity processes for enabling the acquisition of
16 motor skills is motor imagery. Indeed, “real” motor training, i.e., motor execution in an
17 ecological context, is not always possible. On the other hand, motor imagery triggers brain
18 structures sharing similar neural networks with motor execution, including motor-related
19 regions and the inferior and superior parietal lobules [29]. Interestingly enough, visual and
20 kinesthetic imagery seem to involve specific networks of sensory modalities, i.e.,
21 predominantly occipital regions and the superior parietal lobules for visual imagery, as well as
22 motor-associated structures and the inferior parietal lobule for kinesthetic imagery [37], see
23 **Figure 1**. The activation of these networks seems to induce changes similar to those obtained
24 by motor execution, and might be led by the same molecular mechanisms. Indeed, motor
25 imagery seems to induce LTP-like plasticity in M1, in the same way as motor execution does
26 [7].

27 Post-stroke recovery provides a well-documented model of neuroplasticity and brain
28 reorganisation following a focal lesion. Following the complex injury pathways that disrupt
29 cerebral organisation, many mechanistic pathways enable the improvement of functional
30 outcomes. Notably, both neuroplasticity and reinforcement of cortical representation areas have
31 been described in animals [72] and in humans [50]. These results suggest a very plastic
32 functional cortical representation [107] and have been hypothesized to involve various
33 mechanisms, notably including vicariance. Vicariance is the theory that the cortical functions

1 of damaged areas can be managed by other, separate brain regions. Imaging studies have
2 provided examples suggesting the occurrence of this mechanism in stroke recovery [40, 107].
3 Perilesional areas are also recruited and involved in recovery [69, 90, 106]. Underlying
4 mechanisms include the functional use of pre-existing synaptic networks as well as structural
5 changes, with the creation of new networks. All these phenomena might be enhanced by
6 specific rehabilitation training procedures. Rehabilitation training and learning of skills consist
7 of inducing recovery of impairments and disabilities in order to regain activities through specific
8 and complex interventions, while adaptive strategies compensate for impaired body functions
9 (for a review, see [48]). Thus, mental imagery –and motor imagery (MI) in particular– can be
10 considered as a rehabilitation training procedure. MI practice has been shown to enable the
11 improvement of motor skills. Nonetheless, it should be noted that MI abilities can be harmed
12 in the case of motor impairment [44]. Motor imagery produces corticospinal facilitation of the
13 specific muscles used to execute the imagined action [68] as well as increases of hand
14 representation in the motor map area over M1, as evoked by TMS, thus reducing inter-
15 hemispheric imbalance [18]. In a similar way to healthy subjects, MI training has been shown
16 to result in structural and functional modifications of the motor cortex in patients with stroke
17 [50]. Similar modifications, including synaptic and cortical map plasticity, have been shown
18 following action observation. Indeed, studies combining another rehabilitation approach to
19 motor imagery have shown re-organization of cortical activation maps for the affected hand,
20 using fMRI (for a review, see [33]). This improvement of motor function induced by MI seems
21 to be partially related to prior MI skills. In other words, recovery of motor function after
22 subcortical stroke seems to involve pre-existing cortical networks [92].

23 Another way to enhance neuroplasticity in post-stroke rehabilitation of upper-limb impairment
24 is to provide motor-impaired patients with positive sensory feedback, using mirror therapy or
25 muscle vibrations for example. The latter enables provision of haptic feedback and has been
26 suggested to improve motor function, to reduce spasticity and to induce plastic effects on M1
27 in chronic stroke patients (e.g. increasing motor map areas and reducing resting motor threshold
28 as measured using TMS) [53]. Regarding mirror therapy, patients are asked to attempt
29 synchronous bilateral upper limb movements while observing, in a mirror, the reflection of the
30 unaffected limb located in the same position as their affected limb. Mirror therapy has been
31 suggested to significantly improve motor function in stroke patients [101]. TMS studies in
32 healthy subjects revealed that a mirror visual feedback increased neuronal excitability in M1
33 [71] while fMRI studies suggested that this induced functional changes in the somatosensory
34 areas, premotor cortex or higher-order visual regions like the superior temporal gyrus and the

1 superior occipital gyrus [32, 38, 54]. In chronic stroke patients, mirror therapy seems to induce
2 cortical reorganization with a shift in activation balance within the primary motor cortex
3 towards the affected hemisphere, as observed in fMRI [55]. In a study using MEG, Rossiter et
4 al. found that mirror therapy normalized an asymmetrical pattern of movement-related beta
5 desynchronisation in M1 during bilateral movements [88].
6 The latter study leaves open the opportunity to optimize the delivery of this feedback in order
7 to enhance modifications of these cortical rhythms. The delivered visual or haptic feedback
8 might not be temporally synchronized with the efferent cortical motor activity in both mirror
9 of muscle and vibration therapies. Brain-computer interfaces (BCI) and neurofeedback, which
10 are technologies that allow for the voluntary control of an external device (BCI) or of visual
11 feedback, enable the detection of this efferent activity in order to produce the best time-matched
12 feedback, and enhance the plastic effect induced by motor imagery in patients lacking motor
13 control after a stroke or in healthy people. Hence, this tool might enable us to close the
14 sensorimotor loop, from efferent motor activity to adapted and synchronized afferent sensorial
15 feedback(s), see **Figure 2**. Indeed, recent findings suggest that training procedures combining
16 motor imagery with a somatosensory input improves motor performance through M1 plasticity
17 similarly to motor execution [11]. Hence, closing the loop with a sensory feedback might
18 enhance the effects of motor imagery and provide robust changes in the brain.

19 *Motor imagery and sensorimotor rhythms*

20 As previously described, the effect of motor imagery (MI) on motor skill learning and
21 neuroplasticity has been largely demonstrated in healthy subjects and patients with stroke. MI
22 has been shown to activate the primary motor cortex (M1) as well as brain structures involved
23 in the planning and control of voluntary movements. MI has been shown to be underlain by
24 amplitude modulations of *Sensorimotor Rhythm* (SMR) [19, 26]. While SMR have been known
25 for many decades, their functional role is still not completely understood. Nevertheless, SMR
26 exhibit a good signal-to-noise ratio that can be measured using EEG, making them a relevant
27 and reliable neurophysiological target for BCI and neurofeedback studies which aim at
28 improving MI or motor abilities. Notably, many BCI and neurofeedback studies investigate the
29 impact of SMR self-regulation training procedures on motor skills enhancement in healthy
30 subjects and patients with motor disabilities (see below).

31 It should be noted however that the characteristics of the so-called “SMR”, especially temporal
32 domain characteristics (i.e., frequency bands), vary quite extensively between studies. Broadly
33 speaking, SMR have been defined as EEG rhythms in the central region, in particular two kinds

1 of EEG band activities: mu (7-11 Hz) and beta (12-30 Hz) [43]. Mu rhythms were described by
2 Gastaut [34]. The clinical definition of mu rhythms [43] states that they are rhythms at 7–11
3 Hz, composed of arch-shaped waves occurring over the central or centro-parietal regions of the
4 scalp during wakefulness. Their amplitude varies but is mostly below 50 mV. They are blocked
5 or attenuated when a contralateral movement or a thought about movement is performed, as
6 well as during readiness to move or tactile stimulation. From a frequency band standpoint, mu
7 rhythms are part of the *Rhythmic Activity within the Alpha Band* (RAAB) [77].

8 It has been shown that it was possible to condition SMR in humans, in particular mu rhythms
9 by Gastaut in 1952 [34] and low beta SMR by Jasper and Penfield in 1949 [42]. More
10 specifically, they stated that it is possible for an individual to learn to self-regulate the amplitude
11 of their SMR during/after MI tasks. Since these pioneering works, SMR have been broadly
12 used for BCI and neurofeedback in order to improve motor skills. While SMR BCI studies
13 generally consider a broad SMR EEG band including both mu (7-11 Hz), low beta (12-15 Hz
14 or even 12-20 Hz) and high beta (20-30 Hz) frequency ranges, in most “SMR neurofeedback”
15 studies, the so-called “SMR” do not include mu rhythms and are generally focused on “low
16 beta” frequency range [56, 65].

17 SMR BCI experiments target two kinds of event-related patterns: event-related
18 desynchronization and event-related synchronization patterns. During resting conditions, SMR
19 activity in the mu frequency band is high. During motor imagery (as for voluntary movement),
20 the amplitude of the mu rhythms decreases. The decrease of the EEG signal power is referred
21 to as an event-related desynchronization. After the motor imagery stops, there is an increase in
22 the high-beta rhythm (as is also the case for movement). This increase is referred to as an event-
23 related synchronization. On the other hand, SMR neurofeedback studies dedicated to “SMR
24 self-regulation” training procedures usually consider a narrower band, usually focussing on
25 upregulation within the low-beta frequency range (12-15 Hz, although this range can vary
26 depending on the study, and may sometimes not be specified). This can also be associated with
27 training of simultaneous down-regulation of the theta band (around 4-8 Hz).

28 ***Sensorimotor rhythm self-modulation and BCI/NF***

29 To the best of our knowledge, there is no consensus with respect to the distinction between
30 brain-computer interfaces (BCI) and neurofeedback (NF). Indeed, on the one hand, the
31 functioning of both methods is in many ways comparable, and both are closed-loop systems. In
32 other words, users/patients are asked to self-regulate some specific brain activity patterns,
33 recorded using techniques such as EEG, functional near-infra red spectroscopy (fNIRS) or

1 fMRI. This brain-activity is then processed in order to provide users/patients with feedback on
2 their ability to modulate the patterns of interest. For instance, users/patients can be provided
3 with visual or haptic feedback that is temporally synchronized with the efferent cortical motor
4 activity. Haptic feedback may have the advantage to improve the neuroplasticity effect related
5 to the MI task. Users/patients should use the feedback in order to improve, i.e., to better self-
6 regulate the patterns of interest. With that said, on the other hand, BCI and neurofeedback have
7 been developed with fundamentally different objectives. Mainly, learning to self-regulate
8 specific brain patterns is an *end* in neurofeedback while it is only a *means* in BCI.

9 - The goal of neurofeedback is to teach users/patients to self-regulate target brain activity
10 patterns in order to specifically enhance cognitive abilities or reduce clinical symptoms. For
11 instance, SMR neurofeedback training procedures, in which patients had to learn to increase
12 their SMR, have proved effective in reducing ADHD symptoms [6].

13 - The goal of BCI, and more specifically of mental imagery-based BCI (MI BCI), which is the
14 most commonly used BCI paradigm based generally on SMR-EEG recording, is to control an
15 application without moving, using brain activity alone, through the completion of MI tasks such
16 as motor imagery of the left vs. right hand. Performing each of these tasks will induce specific
17 modulations of brain rhythms. For instance, a decrease in SMR amplitude over the contralateral
18 sensorimotor cortex should occur when users perform unilateral hand motor imagery [78].
19 Since each task is associated with a specific control command, such as “turn the wheelchair
20 towards the left” for left hand motor imagery, the system is able to determine which command
21 the user intended to send from the modulation of their EEG activity. Consequently, BCI users
22 should learn to self-regulate specific brain patterns in order to reliably control the application.
23 Beyond their divergent objectives, neurofeedback and BCI are also traditionally associated with
24 different methods. In order to self-regulate their SMR for instance, users are usually asked to
25 perform specific motor imagery tasks in BCI, while this is not the case in NF, in which they
26 should usually find their own strategy. Moreover, in NF, the target pattern (location, frequency)
27 is usually defined in advance. Then, users should find by themselves how to self-regulate this
28 pattern, using the feedback they are provided with. This feedback usually simply reflects the
29 amplitude of the target pattern. In BCI however, a machine learning approach is usually
30 employed. Such an approach consists of using signal processing algorithms to determine the
31 location and frequency of the target pattern that enable the best discrimination between the
32 different tasks. The EEG feedback that users are provided with reflects their ability to produce
33 EEG patterns specific to each task. In case of a BCI involving left vs. right-hand motor imagery
34 tasks, these EEG patterns would theoretically correspond to modulations of SMR. However,

1 when a machine learning approach without any a priori on the location/frequency of the pattern
2 is used, other EEG patterns could be selected.

3 **NEUROFEEDBACK TARGETING SENSORIMOTOR RHYTHMS FOR** 4 **ENHANCING MOTOR SKILLS IN HEALTHY SUBJECTS**

5 The efficacy of neurofeedback to improve healthy subjects' performances has mainly been
6 studied in three domains [104]: cognitive, sports and artistic activities. In this section, we focus
7 on neurofeedback procedures dedicated to the improvement of motor performance, for which
8 most of the studies have been carried out in the field of sport science. For more information
9 regarding neurofeedback procedures dedicated to the improvement of cognitive and affective
10 aspects of performance, please refer to [36].

11 *Sport motor skills improvement*

12 The first study evaluating neurofeedback efficiency for improving athletes' motor performance
13 was led in 1991 by Landers et al. [47]. Three groups of archers were included in the study,
14 including one passive-control group (no neurofeedback), one active-control group
15 (neurofeedback with "incorrect feedback") and one experimental group (following a
16 neurofeedback procedure based on the self-regulation of "low frequency EEG oscillations",
17 although the exact EEG band was not further described in the paper). Participants in the
18 experimental group significantly increased their performance from pre- to post-test (27 bow
19 shots) while the active-control group showed a decrement in performance and the passive-
20 control group showed no difference in performance between pre- and post-test. Nonetheless,
21 the neurofeedback procedure was not associated with any clear EEG pattern changes.
22 Following this study, several other neurofeedback experiments were conducted in the aim of
23 enhancing athletes' performance in different sports, e.g., in golf [17, 82], swimming [28], dance
24 [36, 81] or athleticism [63]. Mirifar et al. [65] proposed a systematic review of these
25 neurofeedback studies. The authors included 14 studies, of which 6 investigated self-regulation
26 of low beta SMR (12-15Hz on C3-C4 or Cz – up-regulation) and 2 investigated self-regulation
27 of alpha power over the sensorimotor cortex (mu rhythm up-regulation). They concluded that,
28 so far, a majority of published studies support the effectiveness of neurofeedback in improving
29 athletes' performance. Nonetheless, the specificity of the neurofeedback effect remains to be
30 demonstrated. Indeed, it happened that the same protocol had different effects within the same
31 or similar task, and also that different protocols resulted in similar effects within a sport. Mirifar
32 et al. stress the fact that the studies' quality was non-optimal. A similar conclusion was drawn

1 by Xiang et al. [110] following their meta-analysis of randomized controlled trials aimed at
2 assessing the efficiency of neurofeedback procedures to improve sport performance. This meta-
3 analysis, which included 10 studies of which 6 focuses on low beta SMR (12-15Hz), revealed
4 a significant effect of neurofeedback on both athletes' sport performance and EEG self-
5 regulation abilities. Nonetheless, it appeared that this effect was moderated by the control group
6 design. In other words, neurofeedback effect on sport performance was not significantly
7 different in experimental groups compared to active/placebo control groups in well-controlled
8 experiments. The authors suggest that further studies, with better designed and organized trials,
9 should be performed. Notably, as outlined by Park et al. [74] the mismatch between lab study
10 conditions (both in terms of tasks, feedback and training environment) and real sporting
11 conditions may, at least in part, explain the relatively small impact that these studies had on
12 professional athletes. It may be hypothesized that ecological training procedures would favor
13 both the acquisition and transfer of motor skills.

14 *Acting skills improvement*

15 Such an ecological neurofeedback procedure has been previously used in order to enhance
16 acting performance in one study [35]. Participants had to up-regulate their low-beta SMR
17 activity while down-regulating their theta and high-beta activities. One group performed
18 neurofeedback while being immersed in a virtual reality environment, one group was provided
19 with standard feedback on a computer screen and the last group was a passive-control group.
20 Participants of both the neurofeedback groups exhibited improved acting skills compared to the
21 passive-control group. Moreover, the virtual reality group reached the *neurofeedback*
22 *performance asymptote* earlier than the standard computer screen group, reinforcing the
23 hypothesis that ecological settings favor learning. In the same vein of artistic performance
24 enhancement, Eegner and Gruzelier [27] carried out two experiments that aimed to investigate
25 neurofeedback effects on musical skills. The first experiment revealed that participants who
26 followed neurofeedback training exhibited an improvement in musical performance. More
27 specifically, participants who carried out not an SMR neurofeedback protocol, but an
28 alpha/theta training procedure (which consisted of “increasing theta (5–8 Hz) over alpha (8–
29 11 Hz) activity levels during a wakeful eyes-closed condition for the purpose of relaxation
30 training” [27]) combined with weekly physical exercises and mental skills training exhibited a
31 positive correlation between neurofeedback performance (ability to self-regulate alpha/beta
32 ratio) and musical performance. These results suggest that slow wave neurofeedback training
33 could be used to enhance artistic performance. Nonetheless, as stressed by Vernon [104] a

1 number of limitations related to the experimental protocol prevent firm conclusions with
2 regards to the specificity of this improvement, i.e., the extent to which this improvement was
3 due to the neurofeedback procedure itself or to confounding factors.

4 *Surgical skills improvement*

5 Furthermore, Ros et al. [85] proposed an SMR neurofeedback procedure in which participants
6 had to “*elevate the low-beta SMR (12-15 Hz) while concurrently suppressing theta activity (4-*
7 *7 Hz)*”. This training procedure enabled a significant improvement both in surgical technique
8 and task completion time in microsurgeons in comparison to a passive-control group. In a more
9 general/abstract context, Ros et al. [86] investigated the effects of a neurofeedback procedure
10 of mu suppression over the right motor cortex on motor performance in a within-subject
11 experimental design. Participants had to perform two serial reaction time tasks (SRTT), one
12 immediately after the neurofeedback procedure, the other one either 7 days before or after the
13 neurofeedback procedure. Performances at the SRTT improved significantly quicker during the
14 task when the latter was performed just after a neurofeedback procedure than when it was
15 performed 7 days before or after. This result suggests that a single neurofeedback session may
16 be directly used to facilitate the early acquisition of a procedural motor task and that
17 neurofeedback effects could be exploited immediately after this neurofeedback session.

18 **BCI/NEUROFEEDBACK TARGETING SENSORIMOTOR RHYTHMS FOR** 19 **ENHANCING MOTOR SKILLS IN PATIENTS WITH BRAIN AND MENTAL** 20 **DISORDERS**

21 Motor abilities can be affected in case of brain or mental disorders. In this section, we focus on
22 SMR BCI/neurofeedback procedures dedicated to the improvement of motor skills in brain and
23 mental disorders for which most of the studies have been conducted. Concerning brain
24 disorders, we focus on stroke. In this disorder, SMR BCI/neurofeedback training procedures
25 aim to reduce hypokinetic activity by training to down-regulate mu rhythm or low beta SMR
26 activity (enhance SMR event-related desynchronization) or to up-regulate high beta SMR
27 activity (enhance SMR event-related synchronization) by performing MI. Such an application
28 of BCIs based on MI results from the literature on the benefits of MI for post-stroke
29 rehabilitation. Concerning mental disorders, we focus on ADHD. In this disorder,
30 neurofeedback aims to reduce hyperkinetic activity by training to up-regulate low beta SMR
31 activity during an attentional resting condition (no MI / motor inhibition). For a general review
32 of neurofeedback and mental and brain disorder, please refer to [61].

1 *Reducing hypokinetic activity: the case of stroke*

2 BCI methods have been used according to two strategies for post-stroke neurorehabilitation
3 [24, 94, 102]. The first strategy consists of using BCI approaches as “assistive technologies”,
4 i.e., as substitution for a lost motor ability [20]. The second strategy consists of using BCI as
5 “rehabilitative/restorative technologies”, by using neurofeedback procedures, in order to
6 facilitate brain plasticity and motor recovery [26]. Both these techniques have been largely
7 developed by Birbaumer’s and Pfurtscheller’s teams.

8 Assistive BCI were initially used for patients with tetraplegia in order to translate some specific
9 EEG brain patterns into a selection of letters or words on a computer screen [9, 10] or to control
10 a prosthetic limb [20]. However, the reliability and performance of such non-invasive EEG BCI
11 are not high enough for these technologies to be used in daily life as assistive technology.
12 Further researches are currently being conducted with invasive BCI or hybrid BCI (EEG with
13 other physiological signals such as electrooculograms (EOG) or electromyograms (EMG) [64].
14 Rehabilitative BCI training procedures are very close to neurofeedback procedures.
15 Nonetheless, in the field of stroke rehabilitation, the term BCI is more often used than the term
16 neurofeedback. Both these procedures rely on the fact that SMR modulations in the hemisphere
17 ipsilateral to the stroke lesion can benefit the motor recovery [94]. In order to maximize the
18 brain plasticity effect, the feedback is integrated into an orthotic device that assists the desired
19 motor movement of the paralyzed hand [9, 14]. The hypothesis behind such a closed loop neural
20 interface is that re-establishing the contingency loop between the ipsilateral cortical activity
21 related to motor imagery (motor planning or attempted execution in relation with the measured
22 SMR activity) and proprioceptive feedback (induced by the haptic effect of the orthotic device)
23 would provide a somatosensory feedback to the primary motor cortex (M1), via the
24 somatosensory cortex S1 and direct thalamic input. This would therefore induce simultaneous
25 activation of presynaptic inputs to M1 with postsynaptic M1 activation that could result in
26 Hebbian potentiation, improve motor learning and brain plasticity [26], and thus maximize the
27 effect of the physical therapy applied after the stimulation with SMR training [19], see **Figure**
28 **2**.

29 The first case study investigating the use of SMR neurofeedback (reward production of low
30 beta activity (15-21 Hz) and down-regulation of production of theta activity (4-8 Hz) in a patient
31 with a stroke was published in 1995, and suggested a possible beneficial effect of
32 neurofeedback on motor recovery [89]. However, the protocol was not specifically designed in
33 line with the literature on MI in post-stroke. In 2007, Birbaumer et al. [9] developed a specific
34 SMR BCI/neurofeedback training procedure that enabled a stroke patient suffering from a hand

1 motor deficit to control a hand orthotic device (open and close commands, based on MI). In
2 2008, it was shown that a majority of patients with stroke could use such a technology by
3 learning to down-regulate their mu rhythm (i.e., 9-12 Hz oscillations in central areas), thus
4 enhancing SMR event-related desynchronization, in the hemisphere ipsilateral to the stroke
5 lesion [14]. This study thus suggests that patients with a lesion (due to stroke) in the motor
6 cortex may still be able to perform motor imagery and motor attempts of the paretic hand.
7 Nevertheless, this study did not reveal any functional improvement related to the
8 BCI/neurofeedback training procedure. This may be due to the fact that the training was not
9 associated with a goal-directed physical therapy that could enable transfer and generalization
10 of the skills into daily life activities. In 2009-2010, two case studies (down-regulation of mu
11 rhythm, here defined as 8-15 Hz frequency band, occurring during MI [13], and high beta SMR
12 (21-24 Hz) up-regulation occurring after MI [23]) suggested a possible beneficial effect on
13 motor recovery of the association of ipsilateral SMR BCI/neurofeedback training procedure
14 and goal-directed physical therapy [13, 23]. Moreover, fMRI analysis showed that clinical
15 improvement was associated with increased activation of the hemisphere ipsilateral to the
16 stroke lesion [15], which suggests a specific effect of the SMR training procedure on brain
17 plasticity and recovery after stroke.

18 A 2013 randomized double blind controlled study included 32 patients with stroke without
19 residual finger movements [80]. The 16 patients of the active group performed 20 sessions of
20 SMR BCI/neurofeedback training (the exact beta EEG band in central areas was not described
21 in the paper) associated with a goal-directed physical therapy. The other 16 patients of the sham
22 group performed 20 sessions of random SMR BCI/neurofeedback training, also associated with
23 a goal-directed physical therapy. The active group exhibited a higher clinical hand motor
24 recovery. This clinical improvement correlated with fMRI results showing increased activation
25 of the hemisphere ipsilateral to the stroke lesion [80], which was confirmed in other
26 neuroimaging and electrophysiological studies [79, 103, 112]. Interestingly, the efficacy of the
27 SMR EEG BCI/neurofeedback training was related to the integrity of descending and ascending
28 cortico-spinal pathways [12]. This result strengthens the importance of the motor-
29 somatosensory loop to induce Hebbian potentiation and thus brain plasticity [94]. Lastly, it has
30 been shown that stroke recovery was higher with haptic than with visual feedback [73].

31 The efficacy of SMR BCI/neurofeedback training has been consistently suggested in less well-
32 controlled studies. For a recent systematic review, see [67]. Interestingly, Thibault et al. [100]
33 highlighted the fact that only one neurofeedback randomized double blind controlled study
34 demonstrated a clinical superiority of neurofeedback over sham neurofeedback [80]. This study

1 is therefore important in the current debate regarding the efficacy of neurofeedback [57].
2 Nonetheless, the characteristics of the population were not detailed in this study, which may
3 reduce its legitimacy to generalize the results to the entire stroke population. Further studies are
4 thus needed to establish well-defined stroke criteria (severity, localization: cortical vs
5 subcortical, type: ischemic vs hemorrhagic, temporal evolution, etc.) that could be associated
6 with predictive factors of response to such neurofeedback therapies.

7 *Reducing hyperkinetic activity: the case of ADHD*

8 In order to reduce pathological excessive motor activity, it has been proposed that patients could
9 be trained to voluntarily self-modulate their SMR brain oscillations through neurofeedback
10 procedures. The neurophysiological rationale for such protocol did not come from MI literature,
11 but largely from the literature by Sterman's team. In the 1960s, Sterman showed that operant
12 conditioning of SMR through neurofeedback procedures could be applied on cats, which were
13 trained to modulate their SMR brain oscillation [109]. Their results suggested that the cats
14 trained to enhance their SMR activity were more resistant to a drug that induced epileptic
15 seizures [96]. In 1972, the same team published the first case study showing a possible
16 beneficial effect of low-beta SMR up-regulation neurofeedback (i.e., 11-13 Hz oscillations in
17 central areas) on the frequency of generalized motor epileptic seizure [98]. Many open-label
18 studies and a few more controlled trials on the topic have been published since; for a review
19 see [60]. Their hypothesis according to which it would be useful to use SMR training
20 procedures to control seizure activity was based on intracerebral recordings in animals [97,
21 108]. During neurofeedback training, the subject is trained to increase their SMR rhythm and
22 to decrease motor activities in the sensorimotor cortex (S1) in a vigilant state of alertness and
23 motor relaxation [61]. It would decrease the activity of motor efferences in the posterior
24 ventrolateral nucleus (VLP) of the ventrobasal (VB) thalamic complex as well as in the red
25 nucleus (NR) of the brainstem, leading to a reduction in muscle tension and myotatic reflex
26 [97]. The reduction of muscular activity would in turn decrease the activity of somatic afferent
27 paths, which would favour a transition from rapid non-rhythmic activity to rhythmic oscillatory
28 activity between the VLP and the reticular nucleus (NRT) of the thalamus [97]. These
29 oscillatory activities would spread through the thalamo-cortical loops at S1 and give rise to the
30 SMR rhythm synchronization recorded in EEG. The mechanisms of action of neurofeedback
31 on SMR rhythms would thus consist in reducing the activity of efferences and afferences in the
32 sensorimotor system, leading to a decrease in cortical neuronal excitability and an increase in
33 the epileptogenic threshold [97]. It should be noted that SMR share similarities with sleep

1 spindle activities and that training to increase SMR in waking states increases the number of
2 spindles during stage 2 slow sleep states [99]. Therefore, SMR neurofeedback training
3 procedures have been applied to patients with insomnia [31, 91]. Moreover, as it has been
4 clinically shown that SMR neurofeedback induces a decrease of motor activity as well as an
5 improvement of the vigilant state of alertness related to attention capacities, it has been
6 suggested to use such a training for Attentional Deficit Hyperactivity Disorder (ADHD)
7 (previously called hyperkinetic disorder) therapies [58]. In 1976 [52], Lubar's team published
8 the first case study showing a possible beneficial effect of neurofeedback on hyperkinetic
9 activity in a child with ADHD treated with low-beta SMR neurofeedback ("*production of 12-*
10 *14-Hz activity in the absence of 4-7 Hz slow-wave activity*"). This study is very interesting
11 because: i) training (during a single session) and learning (across different sessions) effects
12 during the neurofeedback procedure were controlled, and ii) training procedure and feedback
13 presentation were reversed (trained to decrease the amplitude of SMR) in order to investigate
14 the specificity of the effect on motor inhibition [52].

15 Since then, many studies investigating the efficacy of neurofeedback as a potential therapy for
16 ADHD have been published. In most studies, neurofeedback training protocols correspond to
17 low beta SMR neurofeedback, which in the field of ADHD is conventionally called a
18 Theta/Beta Ratio (TBR) training (on EEG recorded from Cz or C3-C4). Indeed, TBR training
19 procedures consist of rewarding low beta activity (around 16-20 Hz) while down-regulating the
20 theta activity (around 4-8 Hz), in order to decrease the Theta/Beta Ratio. ADHD clinical
21 applications have received a lot of attention both from non-specialist, general public sources
22 [2] and from research groups [3]. Nevertheless, the methodological quality of these studies is
23 very variable, and the results of the different meta-analyses tend to swing back and forth [62].
24 Four meta-analyses investigated the therapeutic usefulness of EEG neurofeedback in ADHD
25 [5, 21, 56, 95]. All these meta-analyses concluded that: i) EEG neurofeedback could be more
26 effective on the inattention dimension than in the hyperactivity dimension of ADHD, and ii)
27 neurofeedback is more effective in randomized open trials than in randomized blinded trials,
28 for which the size effect remains non-significant in two meta-analyses [21, 95]. The discrepancy
29 between these meta-analyses can be explained by the selection of the studies included and the
30 difference regarding the training procedure [62]. Moreover, from a neurophysiological
31 standpoint, it can be questioned whether the chosen EEG targets are valid targets for ADHD
32 children. Indeed, the level of evidence regarding the relationship between SMR activity and the
33 hyperactivity and inattention dimensions of ADHD remains poor [4]. Therefore, an important
34 and timely field of research consists of determining better, i.e. more reliable and specific, EEG

1 targets related to hyperkinetic activity [22], but also to the inattention dimension in ADHD.
2 This will enable more relevant and efficient training to be provided to patients [59], which
3 should ensure that the EEG target is modified during the neurofeedback procedure.

4 **CONCLUSION**

5 In this critical review, we first described and discussed the relationship between neuroplasticity,
6 motor skills and SMR BCI/neurofeedback training in line with the MI literature. More
7 precisely, we argued that BCI/neurofeedback could be used to train healthy subjects and
8 patients to voluntarily self-regulate their SMR in order to trigger neuroplasticity phenomena
9 and enable the acquisition of motor skills. Based on this theoretical background, we introduce
10 the literature in which such procedures have been tested and evaluated. Mainly, we describe
11 studies in which SMR BCI/neurofeedback demonstrated efficiency in improving sport
12 performance as well as acting and surgical skills. BCI/Neurofeedback training seems to be very
13 promising for improving healthy subjects' motor skills. Nonetheless, due to a range of
14 limitations (e.g., selection and design for the control group, varied number of sessions, non-
15 ecological training procedures, non-optimal feedback, absence of recordings of changes in
16 baseline EEG activity) we agree with previous authors that “a clear association between
17 BCI/neurofeedback training and enhanced performance [both in terms of EEG self-regulation
18 and motor skills] has yet to be established” [104]. We also present the literature in which the
19 efficiency of these BCI/neurofeedback techniques to improve the clinical symptoms of patients
20 showing hyperkinetic (ADHD) or hypokinetic (stroke) activities was assessed. While these first
21 results are promising, further investigations are also required, especially in order to determine
22 the specificity of the effects observed: is the clinical improvement due to neuroplasticity
23 phenomena? To which extent? What is the influence of the placebo effect and the mechanisms
24 underlying the latter? What is the impact of the training procedure and which variables should
25 be controlled for? In the context of stroke studies, Ramos-Murguialday et al. [80] highlight the
26 challenges to be addressed in order to ensure the efficiency of BCI/neurofeedback techniques:

- 27 i. It is possible to assess the clinical efficacy of BCI/neurofeedback through a well-
28 controlled study (with adequate size of study population, randomized and blinded
29 protocol, adequate control group, and high quality of EEG BCI/neurofeedback
30 sessions),
- 31 ii. It is of the utmost importance to determine reliable, valid and well specified (particularly
32 in terms of spatial and temporal characteristics) EEG targets, related to the clinical

1 parameter of efficacy, to underlying pathophysiological mechanisms of the disorder and
2 also mechanisms of neuroplastic modulation,

- 3 iii. It is also necessary to control for learning, training and plasticity effects by designing
4 and implementing an appropriate BCI/neurofeedback protocol design (with optimized
5 quality of signal recordings, signal processing, threshold, feedback reinforcements,
6 duration and number of sessions, transfer/generalization methods) [3].

7 These challenges appear relevant and could be applied to other pathologies. Regarding clinical
8 aspects, this review has mainly focused on SMR BCI/neurofeedback procedures in stroke and
9 ADHD. Nevertheless, motor skills are also altered in various other neurological and psychiatric
10 disorders. In particular, motor disabilities are core manifestations in depression and
11 schizophrenia; some studies suggest a relationship with SMR alterations [70, 93]. Here as well,
12 as highlighted by Ramos-Murguialday et al. [80], further neurophysiological studies are
13 required to better investigate the relationship between specific motor manifestations and SMR
14 activities in these disorders. If such relationships were demonstrated, SMR BCI/neurofeedback
15 could be of great interest to reduce the symptoms of motor disabilities, which are currently
16 under-investigated and under-treated in psychiatry [8].

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1 **Disclosure of potential conflicts of interest:**

2 The authors declare that they have no conflict of interest.

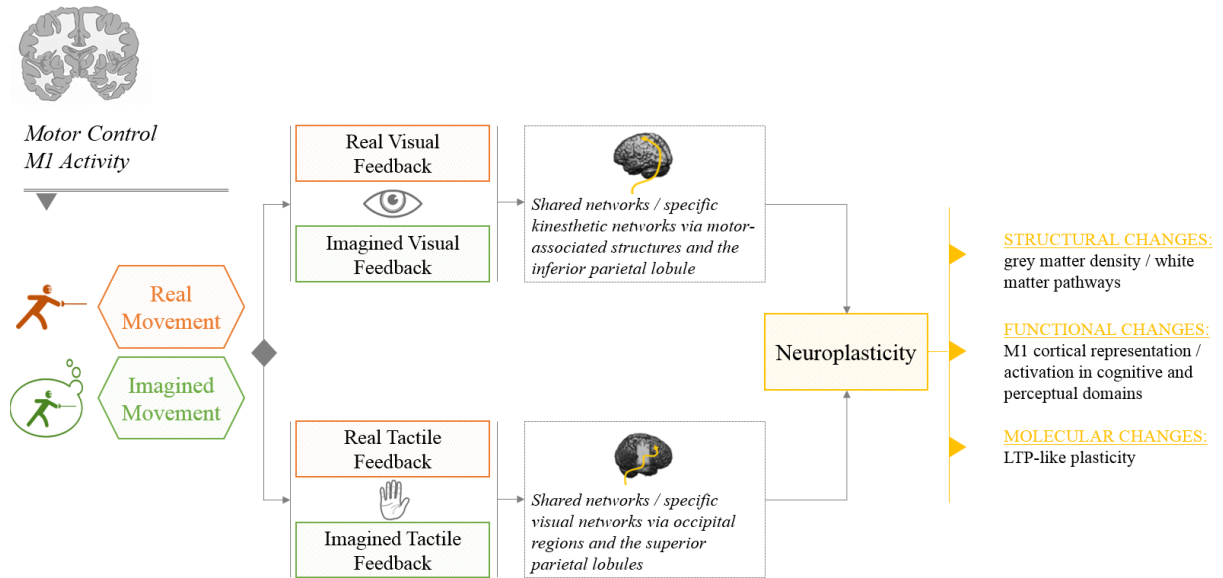
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1 **Figure 1:** Neural processes shared between real and imagined movements, which impact the
 2 brain through neuroplasticity. These plastic effects occur at different levels: molecular,
 3 functional and structural changes induced by visual and/or haptic feedback. *LTP = Long-Term*
 4 *Potentiation*

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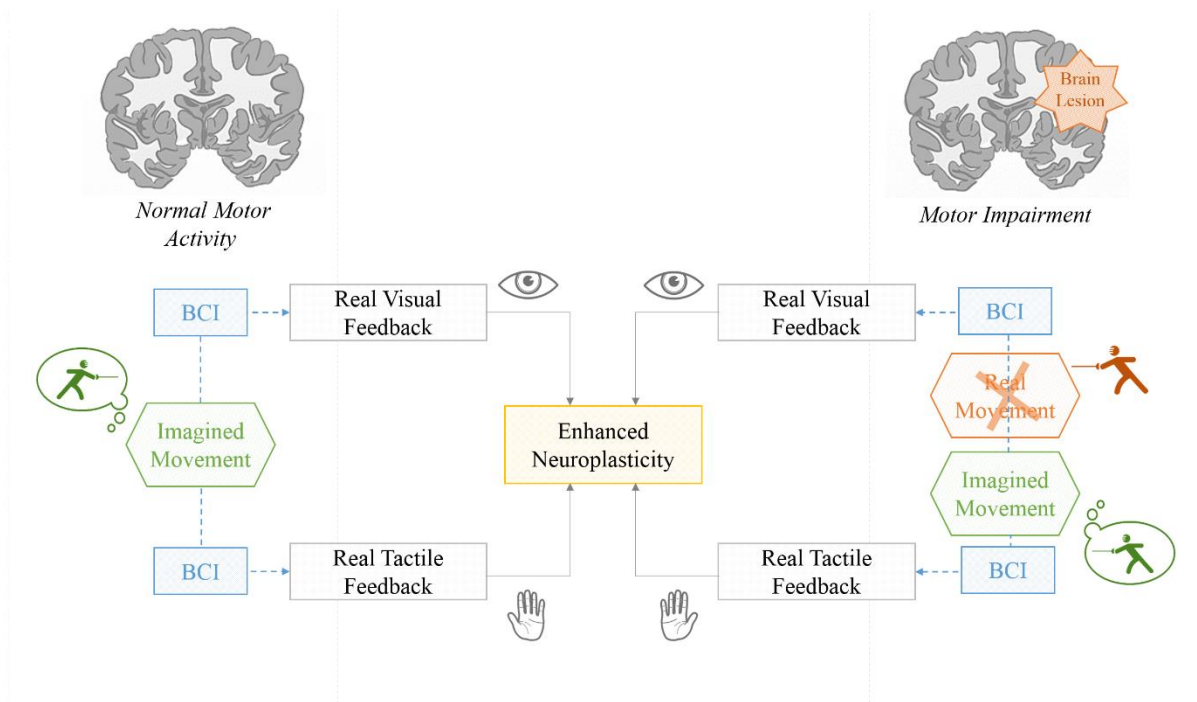


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1 **Figure 2:** Impact of the brain lesion. Due to the lesion and motor impairment, only imagined
 2 movement might have an impact on plasticity, involving networks and mechanisms which
 3 enhance neuroplasticity, particularly using brain computer interfaces (BCI) that enable their
 4 users to be provided with visual and/or haptic feedback
 5



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