Chapter 7
Neuromodulation Techniques in the Treatment of Addictions

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7.1 Introduction

Firmly grounded in a vision that addiction is a brain disease (Leshner, 1997), neurostimulation emerged as an encouraging set of techniques aimed at restoring brain functions and improving clinical trajectories (Ekhtiari et al., 2019). It capitalised on influential theories that considered abnormal neurocognitive functioning as a key dimension of addiction (Goldstein & Volkow, 2002, 2011; Koob & Volkow, 2016; Noël et al., 2013; Robbins & Everitt, 1996; Robinson & Berridge, 2008, 2016).

Indeed, the progress made in brain imaging over the last decades represents a marked advancement in our understanding of substance use disorder (SUD) (American Psychiatric Association, 2013), as it offers concrete and effective modelling of addictive states' neurobiological underpinnings (Parvaz et al., 2011). While the initial investigations mainly focused on limbic-dysregulated activity and the reward system, the research emphasises a wider disrupted neuronal circuitry of addiction (Goldstein & Volkow, 2011; Noël et al., 2013; Koob & Volkow, 2016). Indeed, SUD could arise from an imbalance between three separate but interacting neural systems: a reflective, principally prefrontal cortex (PFC)-dependent system involving inhibitory control and decision-making, predicting the future consequences of behaviour; an impulsive system, mostly on the amygdala-striatum, promoting automatic, salient and habitual behaviours; and the insula that integrates

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interoception, which further integrates conscious feelings and decision-making processes involved in short risky profit (Noël et al., 2013).

In addition to pharmacological intervention (Mann et al., 2014), a growing amount of data has promoted the efficacy of non-pharmacological neurocognitive interventions for SUD (Coles et al., 2018; Noël et al., 2019). To date, the dorsolateral prefrontal cortex (DLPFC) has been the most targeted brain area for improving cognitive control, decision-making and reducing craving intensity (Bechara, 2005; Lüscher et al., 2020; Zilverstand et al., 2018). Insula has recently become a highly relevant subject for reducing SUD symptoms (Ibrahim et al., 2019).

In this chapter, we briefly summarise key findings on the following: (1) deep brain stimulation (DBS), an invasive and focal electrical therapy using electrodes implanted deep into the brain; (2) repetitive transcranial magnetic stimulation (rTMS), a non-invasive technique using a stimulation coil over the scalp delivering a magnetic pulse through the skull over a period; (3) transcranial direct current stimulation (tDCS), a painless non-invasive device delivering low direct current across the scalp with a positive (anodal) and a negative (cathodal) electrode; and (4) neurofeedback (NF), a brain–computer interface using a real-time display of brain activity fed back to the patient so they can learn to implement strategies to regulate their brain activity.

7.2 Invasive Brain Stimulation

DBS is an invasive technique that continuously stimulates brain areas in the long term (Herrington et al., 2016; Montgomery & Gale, 2008). It consists of a pulse generator implanted with brain surgery, with four electrodes placed in deep brain areas. It is possible to turn the system off/on or modify its frequency and intensity. In the 1980s, DBS was applied as an intervention for movement disorders and treating tremors in patients with Parkinson's disease (Benabid et al., 1987). During the 2000s, it was used in psychiatric disorders for patients with treatment-refractory disorders, first in obsessive-compulsive disorder (Nuttin et al., 1999), followed by major depression (Mayberg et al., 2005). Case studies observing the effect of DBS on the nucleus accumbens (NAc) in patients with Parkinson's disease and psychiatric patients with concomitant alcohol and nicotine use disorders show an unexpected reduction in consumption (Ardouin et al., 2006; Kuhn et al., 2007, 2009). Following these observations, Luigjes et al. (2012) suggested stimulating the NAc, involved in motivation and inhibitory control.

Eight case studies have explored the effect of DBS on addiction among a total of 11 patients (meta-analysis Luigjes et al., 2019). Four focused on alcohol use disorder (Voges et al., 2006; Müller et al., 2009, 2016; Kuhn et al., 2011), three on opioid use disorder (Zhou et al., 2011; Valencia-Alfonso et al., 2012; Kuhn et al., 2014) targeting the bilateral NAc and one on cocaine use disorder focusing on the bilateral anterior cingulate cortex (Gonçalves-Ferreira et al., 2016). Most patients were still abstainers after at least 12 months. All opioid use disorder patients and half alcohol

disorder patients were abstainers, while half alcohol use disorder patients and all cocaine use disorder patients were non-abstainers with reduced consumption. A study comparing methadone maintenance and DBS as a treatment for opioid use disorder showed that over 6 months, 47% of patients under methadone had opiate-free urine against 49% on DBS (Stephen et al., 2012).

The results were positive for addiction disorders, but the problem is that DBS is extremely invasive, with 0.4% surgeries leading to death and 2% leading to adverse events (e.g., haemorrhage problems) (Voges et al., 2006). Although DBS appears to be well tolerated after recovery from brain surgery, the risk seems too high and knowledge too unclear to suggest it as common clinical practice for addiction treatment (Carter & Hall, 2011). Indeed, the mechanisms underlying the beneficial effects of DBS have been investigated (Luigjes et al., 2012, 2019; Pierce & Vassoler, 2013; Herrington et al., 2016). The identified mechanisms include neuroplasticity and possibly neuroprotection and neurogenesis (Jakobs et al., 2019) for exhaustive cellular mechanisms.

In conclusion, studies on DBS have yielded positive clinical outcomes, but the cost–benefit ratio is questionable and ethically disputable. DBS should be reserved for patients refractory to any other less invasive treatment as a last resort.

7.3 Non-invasive Brain Stimulation

Strengthening the brain area for clinical improvement without brain surgery and fatal risk was the main aim of non-invasive brain stimulation (NIBS). Although NIBS are less powerful, they allow interventions with fewer risks and adverse events (Rossi et al., 2009, 2020). The first recommendations for using NIBS for clinical purposes date back to 1994 (Rossini et al., 1994). Since then, numerous clinical trials have been conducted in psychiatry (Tortella, 2015; Kekic et al., 2016; Lefaucheur et al., 2017; Fregni et al., 2020), including SUD (Jansen et al., 2013; Grall-Bronnec et al., 2014; Hone-Blanchet et al., 2015; Schluter et al., 2018; Ekhtiari et al., 2019; Luigjes et al., 2019; Stein et al., 2019; Song et al., 2019; Bollen et al., 2021). The most studied NIBS are repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS). rTMS is less invasive than DBS but more invasive than tDCS. rTMS can induce action potential by magnetic stimulation. tDCS, using electric stimulation, is easier and cheaper than rTMS. Multiple sessions of NIBS are safe even for children and adolescents with a similar rate of adverse events as adults, which are mainly headache (11.5%) for rTMS and redness (4.7%), itching (5.8%) and tingling (11.5%) for tDCS (Krishnan et al., 2015).

Both techniques are recommended to be repeated for at least five 5- to 30-min sessions over the DLPFC to be effective in SUD (Luigjes et al., 2019; Song et al., 2019). In SUD patients, DLPFC activation is disrupted, reflecting poor memory, attention and inhibitory capacity in the context of substance-related stimuli (Goldstein & Volkow, 2011). Indeed, the DLPFC is involved in inhibitory control

and decision-making, resisting the urge (Bechara, 2005; Koechlin & Hyafil, 2007; Badre & Nee, 2018; Zilverstand et al., 2018). Induced craving is linked to DLPFC in eight functional magnetic resonance imaging (fMRI) studies (Wilson et al., 2004). It correlates with glutamatergic dysfunction in the NAc and the anterior cingulate cortex, which are two important areas of the reward system (Bauer et al., 2013). An imbalance between the hyperactive emotional system and the hypoactive executive function system is hypothesised to reflect the chronicity of addiction disorders (McClure & Bickel, 2014; Zilverstand et al., 2018; Lindgren et al., 2019). The DLPFC is the most relevant area to be strengthened via the NIBS (Lapenta et al., 2014; Sauvaget et al., 2015; Baeken et al., 2016; Lefaucheur et al., 2017; Luigjes et al., 2019; Song et al., 2019; Fregni et al., 2020; Bollen et al., 2021).

A meta-analysis of 48 NIBS studies concluded that DLPFC neuromodulation has a small effect on craving and a moderate effect on consumption, with no significant difference between the type of substance (alcohol, illicit drugs, nicotine or eating disorders), neuromodulation (rTMS or tDCS) or DLPFC stimulation laterality (left or right DLPFC). Several repeated sessions were more effective than a single session. Moreover, craving and the total sessions showed a positive linear association (Song et al., 2019).

7.3.1 Repetitive Transcranial Magnetic Stimulation

The rTMS uses a coil placed on the scalp stimulating magnetic pulses through the skull in intervals. The magnetic field involves a focal electrical current that depolarises underlying cortical neurons. The frequency, intensity and duration of current, with the properties and area, influenced the effect. The rTMS can be either low frequency (<2 Hz) to inhibit the target area and decrease its activity or high frequency (>5 Hz), also called deep TMS, to increase the regulating activity and excite the target area (Chen et al., 1997; Siebner et al., 2000; Luigjes et al., 2019).

Typically, for addictive disorders' studies, rTMS targets the right DLPFC with a high frequency to excite the area (Amiaz et al., 2009) or the left DLPFC at a low frequency to inhibit it (Trojak et al., 2015). A recent meta-analysis based on 26 studies found that rTMS over the left DLPFC reduces craving and the bilateral DLPFC reduces consumption, compared to sham stimulation (medium and robust effect) (Zhang et al., 2019a). The DLPFC seems to be the most accurate area to target, but laterality remains a controversial issue. Song et al. (2019) did not find a difference between the right and left hemisphere, while Maiti et al. (2017) found an effect on nicotine craving with rTMS on the bilateral DLPFC. Enokibara et al. (2016) also found a craving reduction with rTMS on the right DLPFC. This controversy could be due to the small number of clinical trials, small sample sizes and high heterogeneity (e.g., type of SUD, baseline characteristics of the sample, rTMS method, number of sessions and context such as psychiatric and pharmacologic interventions). Although the rTMS effect did not significantly differ by the SUD type, the craving effect size was small for alcohol use disorder, medium for nicotine

use disorder and large for illicit drugs. Additionally, more pulses during stimulation were associated with a greater craving effect size (Zhang et al., 2019a).

Five 8-min sessions of 10 Hz rTMS over the left DLPFC reduced the craving of methamphetamine use disorder patients compared to sham (Su et al., 2017). The blinding TMS procedure commonly uses a sham coil without an electromagnetic pulse or a considerably low current to imitate the cutaneous sensation on the scalp muscles (Ekhtiari et al., 2019). Many clinical trials do not include sham rTMS conditions for comparison but use controlled conditions such as waiting lists or habitual rehabilitation (e.g., Liu et al., 2020). A recent clinical trial showed that 20 sessions of 10 Hz rTMS with rehabilitating female methamphetamine use disorder patients reduced their craving for at least 30 days after discharge compared with the control group (Liu et al., 2019). Young female patients showed a greater craving reduction, possibly due to greater cortical plasticity. Additionally, rTMS was more effective in the high-craving subgroup. Indeed, induced craving to activate its neuronal network could make the intervention more effective. A recent randomised, double-blinded, sham-controlled trial showed that rTMS (10 sessions over 2 weeks at 10 Hz, 300 pulses per session) over the left DLPFC along with a smoking video allowed smokers to reduce not only craving cues but also cigarette consumption during the 2-week treatment and 1 month after the treatment (Li et al., 2020).

In addition, other potential target areas could be the insula and medial PFC, which are involved in the maladaptive response in a stressful or rewarding context, influencing decision-making (Euston et al., 2012). After brain damage in the insula, patients are likely to stop smoking easily and not feel a craving after quitting (Naqvi et al., 2007). It corroborates the involvement of the insula in craving (Bonson, 2002) and decision-making (Naqvi & Bechara, 2010). These findings suggest that the insula is a potential target for neuromodulation (Ibrahim et al., 2019). To date, the first and only SUD clinical trial targeting this area was a double-blinded, randomised study using bilateral stimulation over the DLPFC and insula (Dinur-Klein et al., 2014). In total, 115 smokers intending to stop smoking received 13 sessions of either high-frequency rTMS (10 Hz), low-frequency rTMS (1 Hz) or sham rTMS, with or without presentation of smoking cues (six subgroups in total; condition 3 × 2). High-frequency rTMS significantly reduced the number of cigarettes consumed, the Fargerström Test for Nicotine Dependence (evaluating the dependence), and increased the abstinence rate compared to low-frequency rTMS and sham rTMS, especially when applied with smoking cue exposure. High-frequency rTMS showed a reduction in cigarette consumption at 6-month follow-up compared to sham rTMS but not compared to low-frequency rTMS or the condition without exposure. Insula stimulation with deep rTMS is promising for reducing cigarette consumption in the long term.

The newer forms of rTMS have also yielded encouraging results, such as intermittent theta-burst stimulation (iTBS), which is a shorter intervention than typical rTMS (approximately 5 min against 8–10 min) (Chen et al., 2020b; Su et al., 2020). A clinical trial on methamphetamine use disorder patients showed that 20 daily sessions of iTBS over the DLPFC (900 pulses per day) reduced craving and improved cognition and sleep quality compared to sham conditions (Su et al., 2020).

7.3.2 Transcranial Direct Current Stimulation

The tDCS is the most investigated neuromodulation among transcranial electrical stimulation (tES) classification. The tES is a classification of non-invasive stimulation intended to change brain activity by passing an electrical current. Depending on the technique, there are several tESs (Bikson et al., 2019): transcranial random noise stimulation (tRNS) uses a random stimulus to desynchronise pathological rhythms (Terney et al., 2008); transcranial pulsed current stimulation (tPCS) uses either monophasic or biphasic pulsed waveforms (Jaberzadeh et al., 2014) and transcranial alternating current stimulation (tACS) uses a sinusoidal current waveform (Antal et al., 2008). For blinding, studies used sham tES, a short duration stimulation (10–30 s) to give the cutaneous sensation of being stimulated (Ekhtiari et al., 2019).

tDCS is electric neuromodulation that acts with a low intensity of constant current applied by two electrodes on the scalp. The current may vary between 0.5 and 2 mA and last between 10 and 40 min with fade in and fade out (10–30 s) of the current (Higgins & George, 2019). tDCS can be used in three montages (Zhao et al., 2017). The bi-cephalic montage indicates that the anode and cathode are placed on the scalp, the anode delivers current to the brain and increases cortical excitability, and the cathode inhibits brain excitability and current escapes from it (Nitsche & Paulus, 2000, 2001). The distance between the two electrodes on the scalp can influence the strength of neurostimulation (Bikson et al., 2010). In the mono-cephalic montage, the anode is placed on the scalp and the cathode on the body as the reference electrode (e.g., arm or neck). It makes possible to focus on the observation of the anode effect and limit confusion. The non-cephalic montage suggests non-cortical stimulation, such as the cerebellum (Zhao et al., 2017).

Similar to other NIBS, the mechanisms of tDCS are still unclear (Nitsche et al., 2003; Arul-Anandam & Loo, 2009; Stagg & Nitsche, 2011; Brunoni et al., 2012; Philip et al., 2017; Zhao et al., 2017). The literature suggests that tDCS mechanisms act like long-term potentiation and long-term depression (Nitsche et al., 2003). The action potentials are modulated by tDCS even after the stimulation period (Nitsche & Paulus, 2000, 2001), and several neuromodulation sessions could increase the duration of the effects (Boggio et al., 2007). The mechanisms of tDCS during and after stimulation are different. During stimulation, anodal and cathodal tDCS modulates neuronal excitability by altering the resting membrane potential of neurons. The modulation persists after stimulation and produces glutamatergic and GABAergic synaptic plasticity as an aftereffect (Stagg & Nitsche, 2011). Contrary to TMS, tDCS itself is not sufficiently high to directly cause neuronal firing. If the intrinsic fluctuation of the neuron voltage is close to the threshold, the tDCS excitation can make it a potential action (Philip et al., 2017). Therefore, tDCS responsivity depends on cortical excitability, influenced by age, gender, anxiety level, lack of sleep, hormonal status and medication (Sauvaget et al., 2015). There are significant inter-individual differences in response to tDCS (Strube et al., 2016), contextual or inherent (Fertonani & Miniussi, 2017; Li et al., 2015).

Although this neuromodulation has been investigated as an intervention in SUDs (Hone-Blanchet et al., 2015; Schluter et al., 2018; Luigjes et al., 2019; Chen et al., 2020a), not so much for behavioural addiction, the data have been encouraging (Sauvaget et al., 2015). The new guidelines of Fregni et al. (2020) categorised tDCS as an effective treatment for reducing craving and relapse in addictive disorders, especially in alcohol use disorder. The recommendation is bilateral stimulation with the right DLFC anodal and the left DLPFC cathodal tDCS (F4 and F3 positions, respectively, according to the 10–20 international system for electroencephalography [EEG] electrode placement). More tDCS studies are required to conclude regarding crack-cocaine or methamphetamine use disorders. A longer duration of stimulation is related to a larger effect size in reducing craving (Chen et al., 2020a). Multiple sessions improve craving reduction compared to a single tDCS session (Song et al., 2019; Chen et al., 2020a).

A recent clinical trial showed that 10 sessions of DLPFC tDCS over 5 weeks on methamphetamine use disorder patients reduced craving and improved executive functions immediately after and 1 month after the intervention. Interestingly, there is a significant correlation between the reduction in craving and cognitive control improvement (Alizadehgoradel et al., 2020). Most clinical trials have targeted craving. Numerous meta-analyses have reported a reduction in craving (Jansen et al., 2013; Sauvaget et al., 2015; Lupi et al., 2017; Spagnolo & Goldman, 2017; Chen et al., 2020a, b; Kang et al., 2019; Luigjes et al., 2019; Song et al., 2019; Bollen et al., 2021). A recent meta-analysis of 32 tDCS studies found a medium effect in reducing craving, indicating more effect with longer stimulation sessions and a higher number of sessions (Chen et al., 2020a).

Few clinical trials have investigated the effect of tDCS on the relapse rate. At the 6-month follow-up after 10 sessions of tDCS, eight patients suffering from alcohol use disorder were still abstainers in the active condition against two in the sham condition (50% vs. 11.8%) (Klauss et al., 2014). The DLPFC stimulation can increase the quality of life and can decrease the relapse rate in patients suffering from alcohol use disorder, sometimes without reduced craving, depressive and anxiety symptoms, or improved cognitive function (13 min × 10 sessions twice a day of 2 mA stimulation, anode-right and cathode-left DLPFC, and 6-month follow-up) (Klauss et al., 2014) and sometimes with reduced craving (20 min × 10 daily sessions of 2 mA stimulation, anode-right and cathode-left DLPFC, and 3-month follow-up) (Klauss et al., 2018b). In the same design as in Klauss et al. (2018b) but on patients with crack-cocaine use disorder (Klauss et al., 2018a), the relapse rate was not affected. Another clinical trial with guided tDCS (10 sessions of 2 mA, anodal-right DLPFC and cathodal-left DLPFC over 5 consecutive days) did not reduce the craving or relapse rate or improve cognitive function in patients with cocaine use disorder (Verveer et al., 2020). Nevertheless, active tDCS reduced the relapse rate in the crack-cocaine subgroup users compared to sham tDCS.

Despite the positive impact of tDCS on craving and relapse rate, results remain inconsistent across clinical trials. This is possibly due to the large heterogeneity of the experimental setting (e.g., type of substances, targeted area, localisation of anode/cathode, ampere of tDCS, duration of stimulation, the time between sessions,

number of sessions) (Luigjes et al., 2019; Chen et al., 2020a) and variations between the studied samples (e.g., characteristics of the sample at baseline, sample size, study design) (Bollen et al., 2021). Although most studies use a between-subjects design comparing multiple conditions (e.g., active tDCS vs. sham), a within-subjects design (e.g., a sample performing both conditions in a counterbalanced order) could limit baseline bias. Further, cognitive remediation during tDCS could improve cognitive functions and reduce clinical symptoms (Elmasry et al., 2015; Dedoncker et al., 2016; Noël et al., 2019; Zhang et al., 2019b; Bollen et al., 2021).

7.3.3 Combined Non-invasive Brain Stimulation

NIBS techniques have the advantage of being combined with other interventions. In most cases, the participant is passively stimulated (i.e., in the absence of any effort), while in other cases, another intervention may be offered simultaneously (e.g., cognitive remediation, rehabilitation of cognitive biases, mindfulness or psychotherapy, simultaneously with tDCS). Combining complementary interventions could (1) combine the effects of the two interventions and (2) potentiate the effects through synergy (Dedoncker et al., 2021).

tDCS can be especially suitable for combined intervention because the sensation is minor and should not distract the patient during a task. According to the activity-selectivity assumption, tDCS preferentially induces modulation in already activated neuronal networks compared to inactive neuronal networks (Bikson et al., 2013). Therefore, it seems possible to target specific neuronal networks by inducing neuronal pre-activation with cognitive training or substance-related stimuli. For the synergistic effect on neuronal plasticity change, tDCS and cognitive training or psychotherapy should activate the same neuronal pathway (Dedoncker et al., 2021).

The combination of these therapeutic interventions, such as exogenous neuromodulation (e.g., tDCS) and endogenous activation (e.g., cognitive training or psychotherapy), has been investigated in a few studies with non-clinical participants and participants with cognitive impairments (Elmasry et al., 2015; Zhang et al., 2019b). To date, no clinical trial in SUD has combined tDCS with psychotherapy. However, eight have combined tDCS with cognitive training related to SUD. Two randomised controlled clinical trials in alcohol use disorder combined tDCS with cognitive bias modification (CBM) (den Uyl et al., 2017, 2018).

CBM is a broad classification of cognitive training focused on cognitive bias retraining. It uses images related to a specific SUD substance that needs treatment. The alcohol attentional bias modification (ABM) uses a dot-probe task to exercise visual disengagement from alcohol images by associating alcohol-related images on the opposite side of the dot to be viewed. It may also use the alcohol approach-avoidance task (AAT) to train the subject to push away the alcohol-related images with a joystick to create an avoidance tendency towards alcohol (developed by Wiers et al., 2009). Four alcohol AAT sessions during 1 week of hospitalisation for alcohol use disorder reduced the abstinence rate by 17% 2 weeks after discharge

(Manning et al., 2021). Meta-analyses of CBM show a reduced relapse rate (Allom et al., 2016; Jones et al., 2016), cognitive biases and cue reactivity (Boffo et al., 2019; Loijen et al., 2020). However, their effects are still limited. Cognitive training could be an interesting add-on treatment for addictive disorders, at least in the short term (Manning et al., 2021).

Two studies combined tDCS with CBM during hospitalisation for inpatients with alcohol use disorder inpatients (den Uyl et al., 2017, 2018). They did not follow the recent guidelines (Fregni et al., 2020) but proposed 20-min neuromodulation at 2 mA with anodal tDCS over the left DLPFC (35 cm^2) and cathodal tDCS over the right DLPFC (100 cm^2) (den Uyl et al., 2017). The two studies differed in the type of CMB. The first retrained the AAT with a joystick (push 90% of alcohol images and 10% of soft drink images; pull 90% of soft drink images and 10% of alcohol images in the active training task; 50% of pull and push in the inactive training task) (den Uyl et al., 2017). The second was the alcohol ABM via a dot-probe task with two stimuli: either alcohol or soft drink images or, in some cases, two soft drink images (absent target or two objects as a surprise trial). In the active training, the contingency probe after alcohol was 90% and 10% after alcohol stimuli (50/50 in the inactive version) (den Uyl et al., 2018). The first study investigated the potential effects of four stimulation sessions on the DLPFC simultaneously with alcohol approach tendency retraining (4× tDCS + AAT, three groups in parallel design) on alcohol bias, craving and relapse after 3 months and 1 year post-discharge (den Uyl et al., 2017). The second study investigated the effects of four sessions of simultaneous simulation on DLPFC combined with ABM (4× tDCS + ABM, 2 × 2 factor parallel design) on craving, alcohol bias and relapse after 1 year (den Uyl et al., 2018). One year after hospitalisation with the combined intervention, den Uyl et al. (2018) showed non-significant results for alcohol relapse rate. Nonetheless, the relapse rate trend was in the expected direction: the combined condition (21%), followed by active tDCS with inactive ABM condition (31%), sham tDCS with active ABM condition (38%) and a worse relapse rate in sham tDCS with inactive ABM condition (45%). A trend-level effect appeared for the first study showing that tDCS concurrent with active training reduces the relapse rate at 1 year only compared to sham tDCS (no difference compared to tDCS separated to the CBM) (den Uyl et al., 2017). Notably, the trend effect appears only when they consider other predictors (gender, duration of alcohol problem, number of detoxifications, alcohol problems, duration of treatment, depression symptoms and scored craving) in the logistic regression. Moreover, there was no effect at the 3-month follow-up.

There are no significant results showing the interest in combining tDCS with CBM. Nonetheless, the two studies measured only long-term relapse (3-month and 1-year follow-up), and results went in the expected direction, showing an average lower relapse rate in the active tDCS condition simultaneous with CBM at 1-year follow-up (den Uyl et al., 2017, 2018). The combined intervention did not influence alcohol-scored craving. However, it was extremely low in patients. Perhaps induced craving is a more sensible measure, as den Uyl et al. (2016) found that active combined tDCS reduces the induced craving (by alcohol images) in EEG tasks in heavy drinkers. More combined clinical trials following the tDCS guidelines

(anode-right DLPFC and cathode-left DLPFC and >5 sessions) (Noël et al., 2019) and measuring early relapse should be investigated (Manning et al., 2021). In addition, reducing the electrode surface could increase the effect (Bollen et al., 2021).

The learning effect of tDCS can be observed 'online' (i.e., during the training with the tDCS) or 'offline' (i.e., the same task after the intervention). With online learning data, it is possible to see if the stimulation increases the learning effect of the cognitive task by comparing the improvement in training in the active tDCS condition compared to the sham tDCS condition. The two studies on AUD patients by den Uyl et al. (2017, 2018) revealed in exploratory analyses that the learning process has been improved by active tDCS on the DLPFC. The first study showed a learning effect on the approach alcohol bias enhanced by tDCS between sessions 1 and 2; however, it disappeared in the last two sessions (mini-assessment before each of the four interventions) (den Uyl et al., 2017). The second study also found an enhancer effect of tDCS on the ABM. The combined intervention with active tDCS and active ABM had a stronger avoidance bias (only with the analysis of the mean of the four sessions) (den Uyl et al., 2018). In conclusion, although the results are fragile, they suggest that tDCS could accelerate the learning process of CBM with ABM and AAT. However, they failed to maintain the effect on offline measures with a similar task after treatment.

To our knowledge, only one clinical trial combining rTMS with another intervention has been reported (Trojak et al., 2015). It combined 10 sessions of low-frequency rTMS with nicotine replacement therapy (nicotine in the form of gum). It showed an improvement in cigarette abstinence directly after the 2-week intervention, but the effect did not last. As there are only two groups comparing sham and verum rTMS, it is impossible to determine if the effect is from this combination. To date, concurrently combining rTMS with CBM or cognitive training has not yet been studied in SUD. Muscular and cutaneous sensations induced by rTMS could not allow the patient to focus correctly on the other intervention. Thus, sequential complementary interventions would be more appropriate.

In conclusion, few studies have combined NIBS concurrently with a complementary intervention, and they did not follow the new recommendations. Further studies combining more than four sessions of bilateral tDCS with anode-right DLPFC and cathode-left DLPFC simultaneously with CBM, cognitive revalidation or psychotherapy are encouraged. Studies combining insula high-frequency rTMS or DLPFC high/low-frequency rTMS during sequential CBM, cognitive revalidation or psychotherapy will advance clinical research.

7.4 Neurofeedback

From Richard Caton's first description of brain electrical activity (1875) to our actual knowledge of EEG, a history of neurophysiology has led to breakthrough advances in technology, allowing an in-depth assessment of brain functioning and

neuromodulatory interventions. In 1935, after Berger (1929) discovered the now vastly recognised synchronised 'alpha EEG rhythm', Alfred Lee Loomis demonstrated that conditioning could be applied to EEG activity by bringing alpha rhythm under voluntary control. This period marked the first demonstration of EEG biofeedback. Subsequently, during the 1960s, Maurice (Barry) Sterman became a pioneer of EEG-biofeedback clinical application through his work on internal inhibition and basal forebrain modulation together with Carmine Clemente at UCLA (USA) (Arns & Sterman, 2019). Currently, there is a definite and growing interest, in both clinical and research domains, towards this neuromodulation technique evidenced by its application to a large sample of psychiatric ailments such as addiction (Cox et al., 2016; Dousset et al., 2020; Horrell et al., 2010), posttraumatic stress disorder (Reiter et al., 2016) and schizophrenia (Balconi & Vanutelli, 2019; Rieger et al., 2018). It has even extended to enhancing healthy subjects' abilities, such as improving performance (Arns et al., 2008; Crivelli et al., 2019).

Essentially, biofeedback is the use of instrumentation to mirror psychophysiological processes of which the individual is not normally aware, and which may be brought under voluntary control (Thompson & Thompson, 2015). In that respect, neurofeedback (NF) stands for a specific kind of biofeedback, with reflected information being cerebral activity measurements. Interestingly, biofeedback is a natural and universally shared regulatory mechanism as our biological system evolves by constantly adapting itself according to the information sent by the peripheral nervous system, just as NF displays are sent by the optic or auditory pathways (Thompson & Thompson, 2015). In this subsection, we will expose the theoretical and methodological aspects of NF, its application through fMRI and EEG interfaces, and the future perspectives towards a better practice.

7.4.1 Theoretical and Methodological Aspects

From a methodological perspective, brain activity measures are converted into visual or auditory signals fed back in real-time to the patient. The patient is asked to work on this fed-back display via mental strategies such as imagining particular events happening (e.g., moving a limb of the body, thinking about negative consequences of drug consumption, etc.), and expected changes are positively reinforced (Cox et al., 2016). Consequently, patients control their responses, see their progress in real-time and achieve optimum performance to control their symptoms or an unwished behaviour (Cox et al., 2016). Thus, NF requires patients to take on an active role in their care—finding personal mental strategies impacting brain activity by themselves and actively implementing them repetitively. Therefore, contrary to medication or compared to the aforementioned neuromodulation techniques (TMS and tDCS) that imply passive involvement, as learning is an active process requiring repetition of training sessions, NF entails implication, motivation and dynamic engagement from the patient.

Theoretically, by rewarding successive approximations, we can shape a behaviour: The patient learns to switch on or switch off a specific network in the brain, and if it is often enough, neuroplastic changes will occur, based on learning (Thompson & Thompson, 2015). Indeed, '… brain plasticity can be induced by demands associated with training, practice or learning and is defined as the brain capacity to continuously remodeling the neuronal synaptic organization in order to optimize the brain's networks functioning …' (Kubben, 2012). To a large degree, this learning process relies on operant conditioning and the fundamental principle of Thorndike's law of effect, whereby rewarding behaviour increases the likelihood of its recurrence (Sterman, 1996; Thompson & Thompson, 2015). In operant conditioning of brain waves, the patient receives a reward (e.g., a smiley or a sun, indicating the level of the current performance) when they successfully put themselves in the targeted mental state—a process that will become almost automatic after several practice sessions. Subsequently, as patients face salient stimuli leading to an intense and irrepressible desire of the substance and ultimately result in consumption, the last step is to apply the learned skill in ecological situations, a transfer process hypothesised to involve classical conditioning (Thompson & Thompson, 2015).

Thus, NF offers the possibility to modify cortical activity, a phenomenon that cannot be achieved without objective brain measurements (Micoulaud-Franchi et al., 2013; Thibault et al., 2016). There are several interfaces for the application of NF, including EEG, fMRI, functional near-infrared spectroscopy (fNIRS) and magnetoencephalography (MEG), many of which involve different technologies and, thus, different procedures (Alkoby et al., 2018; Orndorff-Plunkett et al., 2017; Thibault et al., 2016). Currently, the widespread forms mostly involve fMRI-NF and EEG-NF (Dickerson, 2018). Applied to NF, each method presents its advantages and drawbacks (Thibault et al., 2016; Orndorff-Plunkett et al., 2017). However, a common interesting feature is that it allows targeting neural networks and is not limited to the intervention on just one brain region. Addictive disorders are characterised by abnormal behaviours generated by dysfunctional neurocognitive networks (Kalivas & Volkow, 2005; Noël et al., 2013). By modulating these networks, NF investigation seems to be attractive and promising for reducing symptoms and promoting resilience in favour of an optimal intercession (Sitaram et al., 2017).

7.4.2 Functional Magnetic Resonance Imaging Neurofeedback

As the fMRI-NF offers a good spatial resolution, it has the advantage of localising brain signals to specific areas (Cox et al., 2016). Once the regions of interest (ROIs) have been identified through the peak of the BOLD signal, an NF-training protocol can be implemented to modulate (increase or reduce) neural activity in these particular ROIs (Bracht et al., 2021; Hanlon et al., 2013). Many studies have

demonstrated the effectiveness of fMRI-NF training protocols in patients suffering from addiction by manipulating relevant brain regions related to the abnormal bottom-up system that generates a 'wanting' (craving) behaviour (Luigjes et al., 2013). In fact, it appears that NF training impacts abstinence by reducing the activity of craving-related regions—the anterior cingulate cortex (ACC) (Hartwell et al., 2016; Karch et al., 2019), PFC (Hartwell et al., 2016; Karch et al., 2019), insula and ventral striatum (Kirsch et al., 2016)—and the feedback on the connectivity between the anterior (frontal) and posterior regions (temporal and parietal) (Karch et al., 2019; Luigjes et al., 2019). Conjointly, ACC activity correlates with craving ratings, and patients might be more able to exert voluntary control over the ACC than the PFC (Fovet et al., 2015; Hanlon et al., 2013; Hartwell et al., 2016; Li et al., 2013). Notably, these areas are mostly included in the reward system, which plays a major role in adaptive behaviour, control of behaviours and learning processes (Bari et al., 2018; Karch et al., 2019). Some studies have already attempted to identify the network involved in the brain self-regulation process during real-time fMRI-NF training. This network mostly recruits the anterior insular cortex, basal ganglia and ACC. These regions are recurring targets of fMRI-NF protocols. Therefore, NF studies face a new challenge by considering the potential overlap between the activated regions in response to the NF-induced regulatory phenomenon and the regions whose activity constitutes the target of the experimental protocol for symptom reduction (Emmert et al., 2016).

7.4.3 Electroencephalography Neurofeedback

As NF relies on real-time processes, EEG-NF presents a clear advantage for optimal learning owing to its high temporal resolution (Dousset et al., 2020). EEG-NF is used as a neuromodulation technique to identify target brain frequencies, to increase or reduce specific forms of EEG activity (Gunkelman & Johnstone, 2005). Thus, EEG-NF protocols rely on electrical activity recorded from the scalp and mainly focus on alpha (8–12 Hz), beta (13–30 Hz), delta (0–4 Hz), theta (4–8 Hz) and gamma (30–50 Hz) frequencies or their combination such as alpha/theta ratio and beta/theta ratio (Marzbani et al., 2016; Orndorff-Plunkett et al., 2017; Pandey et al., 2012).

As mentioned, addictive behaviours result partly from an altered top-down process on craving, setting up a reduced cognitive control with impaired inhibition of the dominant response. This impairment has been attributed to abnormal neuroelectrical characteristics: a discrepancy in the N2/P3 complex component with either increased or decreased amplitudes and prolonged latencies (Campanella et al., 2014; Luijten et al., 2014; Petit et al., 2014). Given that identifying the relevant frequency patterns underlying this impairment remains difficult, researchers set out to pinpoint the most suitable NF protocol for treating SUD (Dousset et al., 2020). Since the 1980s, the most popular protocol has been Peniston and Kulkosky's protocol (modulation of alpha/theta frequencies) (Peniston & Kulkosky, 1989),

which targets a state of relaxation. By increasing alpha/theta activity while reducing β-endorphin levels, this protocol counterbalances anxiety-eliciting situations. Thus, patients are relieved from the tension linked with withdrawal in the early stages of abstinence (Peniston & Kulkosky, 1989; Saxby & Peniston, 1995). Scott and Kaiser's protocol, connecting alpha/theta regulation with SMR-beta modulation, extends Peniston's protocol to a larger panel of substances of abuse (Luigjes et al., 2019). On the one hand, alpha/theta modulation intends to soothe conditions of stress and anxiety. On the other hand, SMR-beta modulation aims to alleviate impulsivity by remediating cognitive deficits. Together, these protocols seem to have a pronounced impact on maintaining abstinence (Scott et al., 2005; Sokhadze et al., 2008; Dalkner et al., 2017). Although they have ample merit, the evolution of our knowledge regarding the underlying mechanisms of SUD provides us with the opportunity to investigate the modulation of other frequency bands (Dousset et al., 2020). For instance, as the N2/P3 complex may be viewed as an overlay of brain oscillatory components with the theta band shaping the N200 and the early part of the P300 wave, and the delta band shaping the main part of the P300 (Jones et al., 2006), a delta/theta protocol could be a promising perspective for the care of addicted patients (Kamarajan et al., 2004).

7.4.4 Neurofeedback: Future Perspectives and New Insights

Despite the conventional treatments devised for SUD, the relapse rate remains astonishingly high and outlines the limitations of the conventional systematic approach that offers medication and psychotherapy (Andersson et al., 2019). In fact, SUD induces long-lasting changes in brain functioning resulting from the interaction between chronic substance use, genetic disposition and environment. Hence, a psychiatric diagnosis must consider heterogeneous entities characterised by extremely complex changes in the brain (Perna et al., 2018). Assuming the idea of neurobiological heterogeneity within SUD, identifying biomarkers should allow us to move towards stratified psychiatry, meaning stratifying subgroups of patients' profiles paving the way to personalised medicine to provide reliable and customised assistance (Arns, 2020; Perna et al., 2018). To quote Arns et al. (2011), '… in this area the goal is to prescribe the right treatment, for the right person at the right time as opposed to the current one-size-fits-all treatments' (Arns et al., 2011). The underlying idea behind personalised medicine is that brain imaging data illustrate stable phenotypes incorporating both the effects of nature and nurture. It allows the identification of neurological biomarkers and leads to predictions regarding treatment outcomes (Perna et al., 2018). Ultimately, such insights could allow the implementation of tailor-made NF protocols related to precise alterations and should lead to a more targeted intervention, thereby fostering specific needs to be breached. For example, according to theoretical concepts, both the *incentive-sensitization theory* of Robinson and Berridge (1993) and the *dual-process model* introduced by Wiers et al. (2007) are linked to the *I-RISA (impaired response*

inhibition and salience attribution) syndrome conceptualised by Goldstein and Volkow (2002), putting forward an increased salience of drug-related cues paired with disabled inhibition of the dominant response. In that frame, and as already discussed in our review published in 2020, '… the challenge of maintaining abstinence more directly, i.e., through tailor-made experimental NF protocols targeting inhibitory control and/or attentional bias, warrants increased attention to patient particularities: some benefit more from decreasing/suppressing attentional bias, others more from increasing inhibitory control, and others instead make the most of both' (Dousset et al., 2020).

In the same vein, from a perspective relying upon this dualistic vision of inhibitory control, implementing NF protocols exclusively related to either proactive or reactive processes would lead to more targeted care, meeting a more specific need. In fact, a reactive course is involved in conflict resolution and interference resistance, while a proactive course operates as an anticipatory mechanism and avoids interference by actively maintaining the goal (Braver, 2012). Recent evidence suggests that these distinct modes of control call for both common and specific network activation patterns. More precisely, addictive-inhibited behaviours involve activating an anterior–posterior theta oscillatory network (Cooper et al., 2015). Nevertheless, imaging data put forward different modulations of this network depending on whether the task recruits a proactive or reactive state. On the one hand, a proactive neurobehavioural state is associated with a centro-parietal network involving delta–theta–beta oscillations and mostly recruiting the left putamen, bilateral parietal lobe and premotor cortex. On the other hand, reactive control seems to be strongly involved in right-lateralised frontal, parietal and temporal networks, along with alpha–theta band activity (Cooper et al., 2015; Garcia et al., 2017; van Belle et al., 2014).

Overall, from what we know, and we are still learning about NF, this non-invasive method seems attractive and promising for modulating dysfunctional brain networks associated with SUD to reduce symptoms and promote resilience. As NF is a new approach in managing psychiatric ailments, the challenge remains for establishing standardised procedures for mapping brain networks targeted with NF (Bracht et al., 2021). In this respect, all the investigations will refine our knowledge of how NF works through identifying variables and characteristics that make the NF training effective in favour of an optimal intercession.

7.5 General Conclusion

The primary aim of this chapter was to summarise the main neurocognitive interventions aimed at addressing the clinical aspects of SUDs. To the best of our knowledge, the neurobiology of addiction, that is, an overwhelming motivational drug-seeking and a low capacity to control the desire to consume, is indexed by long-lasting changes in brain function. To remodel these dysfunctional neural circuits, the development of neuromodulation techniques has evolved in response to

the enduring vulnerability to relapse even after years of abstinence. We have explicitly focused on the therapeutic potential of DBS, rTMS, tES and NF approaches in this chapter. Experimental evidence for these neuromodulation techniques demonstrated encouraging results in consolidating abstinence, highlighting the critical role of cognitive functioning in regaining control over problematic behaviours when facing stimuli predicting the availability of the substance and its use. Importantly, despite compelling arguments favouring the previously stated neuromodulation procedures, more standardised and rigorous experimental designs and objective reports are needed to consolidate efficacy (Ekhtiari et al., 2019; Fried et al., 2021; Ros et al., 2020).

In line with the recommendation of several recent reviews (Bollen et al., 2021; Dedoncker et al., 2021), to increase their efficacy, tDCS and rTMS should be combined with psychological interventions (e.g., mindfulness, cognitive training), ideally, tailored to fit distinct endophenotypes (e.g., impaired inhibitory control, low working memory) and learning. Indeed, the state of the brain at the time of stimulation can be critical for optimal clinical outcomes (Dinur-Klein et al., 2014). The 'activity-selectivity' hypothesis stresses that tDCS preferentially modulates populations of active and inactive neurons (Bikson et al., 2013). Finally, the recent recognition that the insula, a region of the cerebral cortex, is involved in various critical aspects underlying SUDs (interoception, decision-making, etc.) led us to recommend targeting this region with brain stimulation in the future (Ibrahim et al., 2019), particularly with deep TMS (Dinur-Klein et al., 2014).

Regarding neurofeedback intervention, considering the progress on our fundamental understanding of the neurobiological underpinnings of SUD, the currently enforced protocols should be kept up to date to specifically target the needs of patients presenting distinguished profiles and move towards a stratified medicine. Further, according to the current fostering discussions in this area, an important aspect of neurofeedback practice is that future studies are required to provide (1) well-controlled experimental designs, (2) objective measures of brain changes and (3) links between neurological biomarkers, cognition and clinical improvements to reliably authenticate the specific impact of neurofeedback and demonstrate robust evidence of its efficiency (Thibault et al., 2017; Micoulaud-Franchi et al., 2018; Thibault & Raz, 2018).

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