Neurophysiologie Clinique/Clinical Neurophysiology (2015) xxx, xxx—xxx

Disponible en ligne sur ScienceDirect www.sciencedirect.com

Elsevier Masson France EM|consulte www.em-consulte.com/en

REVIEW/MISE AU POINT

Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice

Neurofeedback électroencéphalographique : niveaux de preuve clinique pour les troubles mentaux et du cerveau et propositions de bonnes pratiques cliniques

J.-A. Micoulaud-Franchi[a,*,b], **A. McGonigal**[c,d], **R. Lopez**[e], **C. Daudet**[f,g], **I. Kotwas**[h], **F. Bartolomei**[c,d,i]

[a] *Service d'explorations fonctionnelles du système nerveux, clinique du sommeil, CHU de Bordeaux, place Amélie-Raba-Léon, 33076 Bordeaux, France*
[b] *USR CNRS 3413 SANPSY, CHU Pellegrin, université de Bordeaux, 33076 Bordeaux, France*
[c] *Service de neurophysiologie clinique, centre hospitalo universitaire de la Timone, 264, rue Saint-Pierre, 13005 Marseille, France*
[d] *Unité mixte Inserm épilepsie et cognition UMR 751, 27, boulevard Jean-Moulin, 13385 Marseille cedex 05, France*
[e] *Unité des troubles du sommeil, consultation TDA/H adulte, hôpital Gui-de-Chauliac, Montpellier, France*
[f] *Cabinet Saint-Augustin, psychiatrie, psychothérapie, Neurofeedback, 63, rue Pelouse-de-Douet, 33000 Bordeaux, France*
[g] *Université de Bordeaux, 33000 Bordeaux, France*
[h] *Laboratoire parole et langage, UMR 7309, Aix-Marseille université, Marseille, France*
[i] *Hôpital Henri-Gastaut, établissement hospitalier spécialisé dans le traitement des épilepsies, 300, boulevard de Sainte-Marguerite, 13009 Marseille, France*

Received 17 May 2015; accepted 6 October 2015

KEYWORDS
Neurofeedback;
Evidence-based medicine;

Summary The technique of electroencephalographic neurofeedback (EEG NF) emerged in the 1970s and is a technique that measures a subject's EEG signal, processes it in real time, extracts a parameter of interest and presents this information in visual or auditory form. The goal is to effectuate a behavioural modification by modulating brain activity. The EEG NF opens new

* Corresponding author.
E-mail address: jarthur.micoulaud@gmail.com (J.-A. Micoulaud-Franchi).

http://dx.doi.org/10.1016/j.neucli.2015.10.077

Model;
Learning curves;
Cognitive remediation

therapeutic possibilities in the fields of psychiatry and neurology. However, the development of EEG NF in clinical practice requires (i) a good level of evidence of therapeutic efficacy of this technique, (ii) a good practice guide for this technique. Firstly, this article investigates selected trials with the following criteria: study design with controlled, randomized, and open or blind protocol, primary endpoint related to the mental and brain disorders treated and assessed with standardized measurement tools, identifiable EEG neurophysiological targets, underpinned by pathophysiological relevance. Trials were found for: epilepsies, migraine, stroke, chronic insomnia, attentional-deficit/hyperactivity disorder (ADHD), autism spectrum disorder, major depressive disorder, anxiety disorders, addictive disorders, psychotic disorders. Secondly, this article investigates the principles of neurofeedback therapy in line with learning theory. Different underlying therapeutic models are presented didactically between two continua: a continuum between implicit and explicit learning and a continuum between the biomedical model (centred on ''the disease'') and integrative biopsychosocial model of health (centred on ''the illness''). The main relevant learning model is to link neurofeedback therapy with the field of cognitive remediation techniques. The methodological specificity of neurofeedback is to be guided by biologically relevant neurophysiological parameters. Guidelines for good clinical practice of EEG NF concerning technical issues of electrophysiology and of learning are suggested. These require validation by institutional structures for the clinical practice of EEG NF.

MOTS CLÉS
Neurofeedback ;
Médecine fondée sur des preuves ;
Modèle ;
Courbes d'apprentissage ;
Remédiation cognitive

Résumé La technique du neurofeedback électroencéphalographique (NF EEG) a émergé dans les années 1970. Il s'agit d'une technique qui mesure le signal EEG d'un sujet, le traite en temps réel, extrait un paramètre d'intérêt et présente cette information sous forme visuelle ou auditive au sujet. L'objectif est de permettre une modification du comportement du sujet en apprenant à moduler l'activité de son cerveau. Le NF EEG ouvre de nouvelles possibilités thérapeutiques dans les domaines de la psychiatrie et de la neurologie. Cependant, le développement du NF EEG dans la pratique clinique exige : (i) un niveau satisfaisant de preuves de l'efficacité thérapeutique de cette technique, (ii) un guide de bonnes pratiques pour cette technique. Premièrement, cet article a analysé les essais cliniques sélectionnés avec les critères suivant : protocole contrôlé, randomisé, en ouvert ou en aveugle, critère principal de jugement lié aux troubles mentaux ou du cerveau évalués avec des outils de mesure validés, paramètres neurophysiologiques EEG identifiables dans la méthodologie et soutenues par une physiopathologie pertinente. Des essais cliniques ont été trouvés pour : l'épilepsies, la migraine, les AVC, l'insomnie chronique, le trouble déficit de l'attention/hyperactivité (TDA/H), les troubles du spectre autistique, le trouble dépressif caractérisé, les troubles anxieux, les troubles addictifs, et les troubles psychotiques. Deuxièmement, cet article a examiné les modèles thérapeutiques en neurofeedback en lien avec les théories de l'apprentissage. Différents modèles thérapeutiques sont présentés de façon didactique entre deux continuums : un continuum situé entre l'apprentissage implicite et explicite et un autre continuum situé entre un modèle biomédical (centrée sur « la maladie ») et un modèle biopsychosocial intégratif de la santé (centrée sur « le malade »). Le modèle thérapeutique le plus pertinent semble de lier les thérapies par NF EEG avec le domaine des techniques de remédiation cognitive. La spécificité méthodologique du NF EEG est d'être guidé par des paramètres neurophysiologiques biologiquement pertinents. Des guides de bonnes pratiques cliniques pour le NF EEG concernant les questions techniques de l'électrophysiologie et de l'apprentissage sont proposés. La pratique clinique du NF EEG nécessite la validation de ces bonnes pratiques dans le cadre de structures institutionnelles reconnues.

Introduction

It has long been recognised that brain activity may be modulated by conditioning responses. This was first demonstrated in electroencephalographic (EEG) studies from the 1930s and 1940s with regards to the alpha-blocking response [32,45,60], for example by associating an auditory stimulus with a light stimulus, and progressively ''training'' alpha rhythms to eventually attenuate to the auditory stimulation alone (see [7,6,86] for review). Subsequent studies in the 1960s confirmed that alpha-blocking could indeed be conditioned [47,68] and that it was also possible to modulate both EEG synchronization and behavior using operant conditioning; that is, increasing preferred effects and reducing unwanted effects by using a system of reward and punishment [22].

While some inconsistencies exist across studies, it is nevertheless recognized that brain activity as measured by EEG may thus be modified according to specific principles of learning theory [91]. Utilising this concept of ''voluntary control'' of the EEG through learning, the technique of electroencephalographic neurofeedback (EEG NF) emerged in the 1970s. EEG NF is a technique that measures a subject's EEG signal, processes it in real time, extracts a parameter of interest and presents this information in visual or auditory form [23]. The goal is to effectuate a behavioural modification by modulating brain activity [23].

The likely mechanism of this has been considered operant learning; that is, through conditioning, the subject becomes able to increase a preferred state or behavior and decrease undesired ones [86]. However, another viewpoint is that the therapeutic effect of EEG NF occurs through cognitive remediation techniques, guided by specific and biologically relevant neurophysiological parameters [86]. As such, it can be considered within the larger spectrum of new generation neurobehavioral therapies that have been developed according to principles of cognitive science, and that are increasingly employed across a range of neurological, psychiatric and psychological conditions [87].

Following the digitization of EEG in the 1990s, there was renewed interest in EEG NF in the context of easier access to electrophysiological techniques and the possibility of more precise electrophysiological information. Increasing use of clinical EEG NF in psychiatry and neurology since the 2000s [11] has coincided with the development of NF using functional magnetic resonance imaging in real time (fMRI NF), which is currently used only in research, but which offers innovative therapeutic perspectives [48,66,98,103]. Another pertinent technical advance concomitant with the resurgence of EEG NF is the development of the brain-computer interface (BCI), which offers original rehabilitation and therapeutic solutions [27,111]. Despite great interest in fMRI NF and the BCI field of research, significant controversy exists concerning the development of EEG NF in the fields of psychiatry and neurology. With regards to the efficacy of neurofeedback in mental and brain disorders, opinion within the scientific community appears to be rather sharply divided: one school of thought is positive and considers neurofeedback to be effective, whereas the other is very sceptical and does not consider neurofeedback training to have any value, even as an adjunctive treatment. Thus, this article aims to review the evidence for (i) therapeutic efficacy of this technique according to the principle of evidence-based medicine; (ii) good clinical practice for this technique in line with a defined underlying therapeutic model and an institutional structure for regulated practice.

Level of evidence of therapeutic efficacy

There are many trials of efficacy of EEG NF in mental and brain disorders, but most have significant methodological weaknesses [83], which could explain the scepticism of many researchers and clinicians concerning the effectiveness of NF to treat mental and brain disorders. In order to rigorously evaluate the efficacy of EEG NF, in the present article only trials with the following criteria will be presented:

- a study design with controlled, randomized, and open or blind protocol;
- a primary endpoint related to the disorder treated and assessed with standardized measurement tools;
- an identifiable EEG neurophysiological target, underpinned by pathophysiological relevance (Table 1).

EEG NF neurophysiological targets can be classified into three categories according to three general neurophysiological mechanisms:

- arousal [41];
- emotional valence [28];
- sleep.

From a simplified point of view, arousal level can be related to the EEG power in certain spectral bands [41,75]. An increase in central frontal beta band (13—30 Hz) can be related to an increase in arousal [39] and an increase in central frontal theta (4—8 Hz) band is related to a decrease in arousal with subjective sleepiness [102]. Moreover, an increase in occipital alpha (8—12 Hz) can be related to a relaxed state [73].

Emotional valence has been related to EEG power asymmetry in frontal lobes [28]. Despite some inconsistencies in the literature, an increase in alpha power in the left compared to the right frontal cortex has been related to susceptibility toward negative emotions [97].

Sleep stages 2 and 3 (non-REM) are characterized by the presence of sleep spindles. In animal models, it has been found that an increase of sensory motor rhythm (SMR) (Rolandic *mu*) can be related to an increase of spindles and to increased sleep quality [96].

Arousal is a relevant potential EEG NF target for various clinical conditions including attention-deficit/hyperactivity disorder (ADHD), epilepsy, anxiety disorder and addiction. Indeed, ADHD symptoms can be related to a decrease in arousal [10,41]. Epilepsy can be worsened by sleep deprivation and it has already been shown that some patients with epilepsy can control their seizures by enhancing their arousal level [69]. On the other hand, anxiety disorder is related to an increase of arousal [2]. Emotional valence targets are relevant for major depressive disorder, which is characterized by emotional negative bias [29]. The SMR target may be relevant for paretic after stroke, as it was found that SMR is related to motor imagery [72] and that motor imagery could have a positive effect on stroke rehabilitation [13]. Sleep targets are relevant for insomnia that is linked to poor sleep quality [81]. (For summary see Table 1.) Thus, the particular clinical context can be used to determine the neurophysiological target selected for use with EEG NF.

Epilepsies

The neurological disease that has benefited from the most study with EEG NF is pharmacoresistant epilepsy in adults [95,105]. Two meta-analyses reinforced the therapeutic effectiveness of EEG NF in pharmacoresistant epilepsy

Table 1 EEG NF main protocols, EEG targets and supposed psychophysiological effects.

Disorder	Neurophysiological target[a]	EEG target	Aims of EEG NF training	Psychophysiological effect of the training
ADHD and epilepsy[c]	Decrease of the level of reticulo thalamo cortical activation (hypoarousal)[b]	↘ β on fronto central cortex ↗ θ on fronto central cortex	↗ β on fronto central cortex ↘ θ on fronto central cortex	Increase of arousal and of attentional resource
MDD	Dysregulation of the balance between positive and negative emotional valence (negative bias)	↗ α on left frontal cortex ↘ α on right frontal cortex	↘ α on left frontal cortex ↗ α in right frontal cortex	Inversion of the balance between positive and negative emotional valence
GAD[d]	Cortical hyperarousal and neurovegetative hyper reactivity	↘ α ↗ β	↗ α on occipital cortex	Relaxation by decrease of cortical hyperarousal
Insomnia	Decrease of sleep spindle	↘ Rolandic *mu* (or Sensory Motor Rhythm)	↗ Rolandic *mu*	Reduction of sleep latency and increase of sleep spindle

ADHD: attentional-deficit/hyperactivity disorder [65]; MDD: major depressive disorder [21]; GAD: generalized anxiety disorder [79,1]; β: EEG spectral power in beta band > 14 Hz (associated with cortical arousal); θ: EEG spectral power in theta band 3,5—7 Hz (associated with sleepiness); α: EEG spectral power in alpha band 8—13 Hz (associated with cortical hypometabolism).

[a] The identified pathophysiological target cannot be present in some patients. Its absence would be a poor prognostic factor for the EEG NF protocol.

[b] In ADHD and epilepsy, additional protocols were validated on slow cortical potentials (slow cortical potential, [PCS]) and would focus instead on fronto-striatal networks and regulation of neuronal excitability [100]. Such a protocol was also used in migraine [90].

[c] The protocol used in OCD is mostly related to an EEG spectral power reduction protocol in the theta band [51].

[d] The protocol used in addictions is close to that used in GAD but is called ''deep relaxation protocol'' because it involves increasing the spectral power in the theta rather than the alpha band but [77,84].

[95,105]. However, these meta-analyses included some studies with poor methodological quality. Only two studies have satisfactory methodological criteria [52,55]. A randomized and blinded trial was performed in 8 pharmacoresistant subjects with epilepsy compared to a group of 8 matched subjects receiving biofeedback ''placebo'' sessions. Only the group with EEG NF showed a significant reduction in their frequency of seizures (about 61%) [55]. A controlled but unrandomized trial was conducted in 41 patients with pharmacoresistant epilepsy divided into 3 groups: 34 patients with EEG NF training, 7 patients with change of pharmacological treatment (associated with occupational workshops), and 11 with a placebo biofeedback training based on breathing techniques. Only the first two groups showed a decline in the number of seizures (50%), which was significantly superior to the placebo biofeedback [52]. This study was not randomized, but placebo scales were measured for each group based on questionnaires of patient satisfaction with their treatment [52]; no difference was seen between groups and thus a significant placebo effect in explaining the results was effectively eliminated.

In line with EEG NF, a biofeedback technique focusing on skin conductance was recently studied in a randomized blind trial, confirming the effectiveness of this technique, with approximately 50% reduction in seizure frequency [69]. This method was also effective in treating a subgroup of patients with pharmacoresistant temporal lobe epilepsy whose seizures were triggered by stress [67]. This last study is indeed of interest in its study of a specific subgroup of epilepsy patients, since very heterogeneous populations in terms of epilepsy type and severity tend to characterize previous EEG NF studies.

Migraine

Treatment of migraine using biofeedback has been studied in a single randomized open label study in 10 children (versus 10 children on a waiting list) and was in favour of a reduction in number of days with migraine per month in the group treated with EEG NF [90]. Several other studies have been conducted, and although in favour of efficacy, EEG NF does not have satisfactory methodological criteria [63,99]. The efficacy of EEG NF has yet to be confirmed, especially in adults. However, it should be noted that: (i) biofeedback on peripheral parameters (skin temperature, skin conductance, electromyography) presents a high level of evidence to support its use in migraine [71,88], especially confirmed in a meta-analysis [70]; and (ii) that neurofeedback by hemoencephalography (measuring changes in blood translucence that reflect oxygenation and local blood flow) represents an interesting avenue for the treatment of migraines [20].

Stroke

The use of EEG in patients with stroke was initially in rehabilitation in the field of EEG BCI. The aim was to teach the patient to generate SMR modulation in order to control an external mechanical orthosis [3]. Recently, it was proposed that SMR modulation could be also be used through EEG NF for re-education in order to improve motor function recovery after stroke [78]. One randomized blinded trial with 10 patients per group was conducted. Three intervention groups were performed: occupational therapy (OT) alone; OT with EEG NF; and OT with electromyographic (EMG) biofeedback. EEG NF and EMG biofeedback showed similar motor function recovery; further studies with EEG NF are needed. It should be noted that the fields of EEG BCI and EEG NF in the context of stroke patients are closely related because of the possible recovery effect of EEG BCI through a neuroplasticity mechanism [31].

Chronic insomnia

Chronic insomnia was studied with two randomized blinded trials with respectively 16 and 12 patients treated with EEG NF (versus 11 and 12 subjects treated with ''placebo neurofeedback''). These studies were in favour of superior efficacy for EEG NF [42,81]. The effect of EEG NF on sleep could also improve memory function [81].

Attentional-deficit/hyperactivity disorder

Attention-deficit/hyperactivity disorder (ADHD) in children is the psychiatric disorder for which EEG NF has received the most attention from society [11] and from research groups [11,35]. For example, EEG NF in ADHD received a grade 1 (''best support'') rating from the American Academy of Pediatrics in 2013.

Three meta-analyses studies reinforced the therapeutic usefulness of EEG NF in ADHD [9,65,92]. The first meta-analysis of Arns et al. (2009) found an effect size (ES) that was more significant for the inattention dimension than for the hyperactivity dimension of ADHD [9]. However, this meta-analysis is now slightly outdated and included some studies with poor methodological quality. The second meta-analysis of Sonuga-Barke et al. (2013) found an ES significantly higher than in the control group in randomized and open trials, but this effect remained non-significant in randomized and blinded trials [92]. The third meta-analysis of Micoulaud-Franchi et al. (2014) found an ES that was significantly higher than in the control group in randomized and blinded trials for the inattention dimension of ADHD [65]. In these two last meta-analyses, teacher assessment was chosen if the control group performed a cognitive remediation [34,94,104], and assessment by parents and teachers was chosen if the control group performed a placebo NF protocol [12,54]. It should be noted that efficacy with regards to the inattention dimension was proportional to the number of neurofeedback sessions [9] and seemed to be maintained over time [57].

Despite, these meta-analyses, the effectiveness of EEG NF in ADHD remain debated [8,19,26,93,107,108]. In particular, the meta-analysis of Micoulaud-Franchi et al. (2014) did not include the Arnold et al. study (2013) [4] and the studies that compare EEG NF to medication [16,15,74]. These choices warrant some explanations. The study by Arnold et al. used a well-controlled, randomized and blinded protocol [4]. However, the EEG NF protocol was not based on basic learning theory (in particular by the type of reinforcement) used in standard EEG NF protocols [86] and the EEG recording was carried out using an unconventional set-up with electrodes placed on the forehead, a region known to be problematic for recording because of muscle artefacts. The Arnold et al. study thus highlights the need for good clinical practice recommendations for EEG NF, in line with neurophysiological good practice for EEG recording [30] and learning theory [86,109]. The reason for excluding studies comparing EEG NF to medication is because the medication comparison group is not the most appropriate comparison method to EEG NF. Indeed, semi-active control (such as cognitive remediation techniques) and sham-NF groups to compare the EEG NF intervention are more appropriate to control for the non-specific effects of EEG NF [11]. However, comparison with medication found that EEG NF was not as effective as medication in improving clinical symptoms [16,74] or cognitive function [15], but could be more effective on academic performance in the longer term [64].

Autism spectrum disorder

Autism spectrum disorder (ASD) in children is the second condition to have benefited from a relatively large number of efficacy studies with EEG NF [43]. However, it should be noted that, rather than demonstrating specific effectiveness on the signs of ASD, EEG NF may rather be effective on the comorbid ADHD that is present in approximately 40—50% of subjects with ASD [43].

Major depressive disorder

Major depressive disorder (MDD) was studied using a single randomized and open trial in 12 subjects treated with EEG NF (versus 11 subjects treated with ''placebo psychotherapy''). This study was in favour of a superior efficacy of EEG NF [21]. A recent pilot study in 9 subjects reported results for a specific neurophysiological effect of this therapy [76]. The effectiveness of EEG NF has yet to be confirmed in MDD. It should be noted that fMRI NF could offer innovative therapeutic perspectives in the field of mood disorders [59].

Anxiety disorders

Within anxiety disorders, generalized anxiety disorder (GAD) benefited from two randomized and open trials with respectively nine [79] and fifteen [1] subjects in comparison to relaxation techniques with electromyographic (EMG) biofeedback in equal size groups. Obsessive-compulsive disorder (OCD) benefited from a randomized and blinded trial in 10 subjects (versus 10 subjects performing a biofeedback ''placebo'') [51]. All of these studies showed results in favour of a superior efficacy of EEG NF. However, this efficacy should be compared to the well-established benefits of cognitive behavioral therapy (CBT). It should be noted that the relaxation sought by EEG NF protocols used in the GAD studies is probably different from the relaxation obtained by other techniques. EEG NF could thus be proposed as an alternative therapy for subjects who fail to achieve relaxation with other methods [85] and further research is needed to investigate the neuroplasticity effect of EEG NF in anxiety disorder, in particular in post-traumatic stress disorder (PTSD) [49].

Addictive disorders

The first randomized and open study with EEG NF was performed on 15 subjects (versus 14 subjects receiving treatment as usual). All subjects were veterans of the Vietnam War, with a diagnosis of PTSD and addictive disorder. The study found a superior efficacy of EEG NF on maintaining abstinence from alcohol [77]. This effect was confirmed in a separate randomized and open trial in 61 subjects (versus 60 subjects undergoing occupational support). Subjects in this second study did not suffer from PTSD but from various addictions (alcohol, heroin, cocaine, methamphetamine) [84]. It should be noted, as for the ASD, that the effect of EEG NF in addictive disorders might be non-specific, through an effect on anxiety disorder or ADHD comorbidities.

Psychotic disorders

Psychotic disorders have not so far benefited from randomized controlled trials of EEG NF. Interestingly, it was found that patients with schizophrenia were capable of learning self-control of EEG despite their cognitive and motivational deficits [37]. An uncontrolled clinical case study suggested efficacy of personalized EEG NF on psychotic symptoms [104], and the potential therapeutic avenues for EEG NF in schizophrenia seem to be more specifically the treatment of hallucinations using EEG or fMRI NF [62].

Good clinical practice

Any treatment must account for its model according to scientific criteria of evidence and relevance and build the best practice guide for the therapeutic technique and the institutional structure to support it [40]. This is particularly important for EEG NF. Indeed, not only the methodological quality of the trials is discussed in recent studies on evidence of EEG NF efficacy, but also the quality of the conduct of the EEG NF sessions themselves within the ''active'' group [8,112].

Therapeutic model

The practice of EEG NF can be examined in the light of two therapeutic models that can be described didactically, in terms of a spectrum between two paradigmatic poles [36,101].

The first continuum is between implicit and explicit learning. Implicit learning (or ''automatic'' learning) refers to the strict application of the operating conditioning paradigm that was the foundation of the first neurofeedback protocols. Following this therapeutic approach, EEG NF sessions would target the brain in order to automatically correct certain neurophysiological abnormalities. Such an approach shares a therapeutic model close to the effect of electrical brain stimulation [89].

Explicit (or ''voluntary control'') learning refers to cognitive remediation techniques and the development of new skills in the subject suffering from a mental and brain disorders [87]. Following this therapeutic approach, EEG NF sessions would aim to teach the subject by successive ''trial and error'', then to repeat and reinforce new psychophysiological or neurocognitive skills (e.g. attentional and arousal, emotional regulation, or relaxation skills). The subject would require a certain awareness of the skills being learned and would then have to try to transfer these new skills into everyday life. In this model, the role of the therapist is essential to explain the technique, enhance learning, maintain motivation during the sessions and allow tracking of the performance in a given session and in successive sessions [112]. Compared to CBT techniques or other forms of cognitive remediation [87], the originality of EEG NF is not in prescribing specific cognitive tasks but in allowing the subject to find his or her own cognitive task associated with positive reinforcement and thus to maximize the effect on the neurophysiological characteristics targeted by the neurofeedback protocol [86]. This model, which defined neurofeedback as a neurophysiological remediation, implies specific training of the practitioner, who must continually ask the question: ''is the patient learning something?'' [38,112].

The second continuum is between the biomedical model (centred on ''the disease'') and integrative biopsychosocial model of health (centred on ''the illness''). The biomedical model focuses on the brain and postulates a linear causality between a pathophysiological abnormality seen as a trait and a mental and brain disorder. This approach would be to ''correct'' these abnormalities in order to achieve a therapeutic effect [36]. In this approach, a way to improve EEG NF protocols would be to target ''personalized'' EEG abnormalities in a given subject, previously measured by quantitative EEG [46,106], perhaps even topographically identified by EEG source modelling [14,25]. Despite its theoretical appeal, this type of EEG NF protocol remains to be validated. Moreover, the quantitative EEG technique, which involves comparing the EEG data of a subject to a normative EEG basis (including *Z*-score), raises questions about the relevance of EEG trait markers in mental and brain disorders [5,24], as well as questions about technical limitations [18,44,46,106].

Integrative biopsychosocial models of health focus on the patient and suggest links between pathophysiological abnormalities seen as a neurocognitive state and psychosocial dimensions. Firstly, this model posits that: (i) the abnormal neurocognitive state can be compensated by cognitive flexibility in order to ''compensate'' cognitive impairment by ''reinforcing'' other skills [36] and (ii) the flexibility can be enhanced by neurofeedback in order to balance between neuronal synchronization and desynchronization, respectively required for integration/disintegration of neuronal assemblies during flexible cognition [80]. Secondly, this integrative model posits that this neurocognitive flexibility and the reinforcing of new skills would be facilitated by the inclusion of psychosocial variables such as ''locus of control'' or ''self-efficacy'' [35,86,110]. The modulating effect of these psychosocial variables on learning performance during EEG NF sessions [110], as well as on the therapeutic efficacy of the treatment, remains to be studied [35].

Currently, there are important discussions on the choice of therapeutic models for defining the best practice of EEG NF. The quality of EEG NF practice does not fit into the extremes of the spectrum. Early explicit learning (with effort) can become implicit (''automatic'') thereafter

Please cite this article in press as: Micoulaud-Franchi J-A, et al. Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice. Neurophysiologie Clinique/Clinical Neurophysiology (2015), http://dx.doi.org/10.1016/j.neucli.2015.10.077

Table 2 Signal processing software control parameters required for recording EEG in the context of EEG NF. This suggests minimal specifications that should be available when choosing neurofeedback software.

Parameters	Definitions
Filters	Decomposition of the EEG signal by Fourier analysis (or other) for measuring the amplitude of the EEG spectral power in a selected frequency band
Epoch	EEG epoch time in which the spectral analysis will be applied
Sliding averaging	Length of sliding averaging for rendering smoother amplitude fluctuations of EEG spectral powers
Feedback	Feedback on the targeted EEG activity (often continuous) and positive reinforcement (depending on the threshold and holding time) Perceptual characteristics for neurophysiological target (visual or auditory continuous or punctual, score or level, etc.)
Threshold	Amplitude of the EEG spectral power above (or below) which positive reinforcement can be made
Occupation time	Time after which the amplitude value above (or below) the threshold will lead to positive reinforcement
Inhibition	Inhibition of positive reinforcement in case of change of the amplitude of the power spectral EEG related to a biological or technical artifact, not related to a neurocognitive activity of the subject
Annotation	Annotation system for a EEG-neurofeedback session
Duration and sequence of a session	Succession of training periods and rest periods and determination of their durations and numbers
Session report	Evaluation of the training parameter during the session (reflection of the performance)
Storage	Neurofeedback sessions with the EEG signal Records of meetings of accounts (Note: the format of the stored files determines their exportability)
End of treatment report	Evaluation of the learning curve during the sessions (evolution of the training parameter)
Clinical data management	Clinical data management system coupled with neurofeedback sessions

[36,53,101] and an approach targeting a specific quantitative EEG brain abnormality can complete an approach taking into account the self-efficacy of the subject. A degree of lively scientific debate provoked by different therapeutic models of EEG NF is in any case necessary for conscientious and ethical research, and practice of this therapeutic approach [36,86,101].

Practice guideline

The rules of good practice guidelines of EEG NF concern:

- technical issues of electrophysiology [30];
- technical issues of learning [86].

These aspects are rarely discussed in the EEG NF efficacy trials and there is no current consensus [11,38,61,112]. They will be essential to consider in order to gradually improve the practice of EEG NF in neurology and psychiatry [40]. The number of sessions of neurofeedback is usually 20—30 sessions, one to three times per week, but the ideal number and the optimum spacing have not yet been defined [101].

From the electrophysiological point of view, the practice of neurofeedback requires high quality recording of EEG signal [30]. The practitioner must:

- be familiar with the recording equipment and the entire acquisition chain (amplifier, filters, high pass and low pass digital analogue converter);
- correctly set up the recording environment, especially concerning placement of EEG electrodes, to obtain correct impedances, and EEG montages to obtain valid deviation;
- be able to read and continuously monitor the EEG to identify artifacts and adjust the set-up of the material.

As part of the explicit model of learning, the practice of EEG NF requires the measurement of a parameter derived from the EEG and its representation through understandable information for the subject, in order to promote the development of new neurocognitive skills transferable to everyday life. Each software package marketed for the practice of EEG NF has different advantages and disadvantages regarding the handling of the control parameters. The therapist needs to manipulate these parameters to enhance learning and motivation of the subject during the sessions (Table 2).

Good knowledge of electrophysiological and learning techniques will be particularly useful with regards to four key elements of neurofeedback sessions:

- information: this is necessary before (explanation), during (motivation) and after (explanation) each session in order

Please cite this article in press as: Micoulaud-Franchi J-A, et al. Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice. Neurophysiologie Clinique/Clinical Neurophysiology (2015), http://dx.doi.org/10.1016/j.neucli.2015.10.077

for the subject to understand the principle of electrophysiological measurement and to adhere to the therapeutic model involved in the course. However, it seems advisable to minimize the information about the type of neurocognitive strategies prior to a session [50,101];

- the choice of threshold: this is an essential step. Adjusting a threshold (and a given occupation time) determines the number of positive reinforcements required to strengthen the subject in a type of neurocognitive strategy. The threshold may be set automatically or manually. The manual threshold seems to lead to better learning [86,101] but, unlike the automatic threshold, requires performing a baseline measurement before each session;
- the type of positive reinforcement: this can be visual or auditory, proportioned (graduated) or binary (present or absent), immediate or delayed, simple or complex, frequent or rare. Visual feedback, which is proportionate, immediate and simple, seems to allow better learning [101]. The number of reinforcements must be sufficient to maintain the motivation of the subject but not so great that they alter learning [101,38]. Note that positive reinforcement embedded in an entertaining interface (such as video games) may increase the motivation of the subject but impair learning [35,86];
- skill transfer sessions in daily life: these allow the generalization of learned skills [11,35,61]. They may be more effective when neurofeedback sessions are conducted with reference cues to the context of generalization: for example neurofeedback sessions for ADHD in an environment reminiscent of a classroom (now possible with portable neurofeedback equipment) [101]; future developments might even combine neurofeedback in virtual environments [17].

Institutional structure

The place of EEG NF within the current therapeutic approach remains uncertain for the moment. EEG NF does not properly belong to any single discipline. Neurofeedback is indeed at the interface between several domains: biomedical engineering, neurophysiology and psychophysiology, learning science, cognitive behavioral therapy, cognitive remediation and health psychology.

EEG NF in neurology and psychiatry is certainly not a therapeutic measure to consider as sole treatment in subjects presenting a mental and brain disorder. This is a complementary non-pharmacological therapy that could be used in conjunction with other treatment modalities. The institutional structure of EEG NF would enable:

- promotion of the development of research protocols [36,101,109], which would strengthen the level of therapeutic evidence of EEG NF; improve understanding of neurobiological mechanisms of learning and efficacy of EEG NF; and identify variables (neurophysiological, cognitive, affective and psychosocial) that modulate learning and the efficacy of EEG NF;
- quality control of the clinical practice of EEG NF [40]. This last point requires appropriate training of therapists using EEG NF. The Biofeedback Certification International Alliance (BCIA) model is an example that would allow controlled and rigorous development of this therapeutic technique [40].

Conclusion

In the fields of neurology and psychiatry, the therapeutic role of EEG NF currently remains controversial. Further studies are needed to confirm the effectiveness of EEG NF to treat mental and brain disorders. However, promising results have so far been found for therapeutic efficacy in children with ADHD and in patients with pharmacoresistant epilepsy. In parallel to the need for controlled, randomized, blinded studies, recommendations for good practice of this technique and its institutional structure remain to be established, as has been done for example for external brain stimulation [56].

Despite various obstacles to the implementation of EEG NF in clinical practice, the questions raised by this therapy seem to go beyond the strict field of neurofeedback and beyond the controversy between positive and sceptical opinions on the efficacy of EEG NF. Indeed, reflections on neurofeedback are paradigmatic of current thinking in the neurophysiological therapeutic field as applied to psychiatry and neurology. Use of electrophysiological and neuroimaging methods are changing approaches to performing CBT, cognitive remediation [58,82], external brain stimulation treatments [89] and pharmacotherapy [33]. These techniques deal with psychophysiological interactions between the subject and new therapeutic neurophysiological tools available to psychiatrist and neurologist; and raise important questions about how best to evaluate of their efficacy, their best practice rules and their place within medical institutions, which merit further exploration. Despite its current controversy, EEG NF appears thus to be an experimental field that is emblematic of the ongoing evolution of neurophysiology in the therapeutic field of neurology and psychiatry.

Disclosure of interest

The authors declare that they have no competing interest.

References

[1] Agnihotri H, Paul M, Singh Sandhu J. Biofeedback approach in the treatment of generalized anxiety disorder. Iran J Psychiatry 2007;2:90—5.
[2] American Psychiatryic Association. Diagnostic and statistical manual of mental disorder, 5th ed, Text Revision (DSM-5). Washington, DC: American Psychiatryic Association; 2013.
[3] Ang KK, Guan C, Chua KS, Ang BT, Kuah CW, Wang C, et al. A large clinical study on the ability of stroke patients to use an EEG-based motor imagery brain-computer interface. Clin EEG Neurosci 2012;42:253—8.
[4] Arnold LE, Lofthouse N, Hersch S, Pan X, Hurt E, Bates B, et al. EEG-neurofeedback for ADHD: double-blind sham-controlled randomized pilot feasibility trial. J Atten Disord 2013;17:410—9.
[5] Arns M, Gordon E. Quantitative EEG (QEEG) in psychiatry: diagnostic or prognostic use? Clin Neurophysiol 2014.
[6] Arns M. Historical Archives: the beginning.... J Neurother 2010;14:291—2.

[7] Arns M, Kenemans JL. Neurofeedback in ADHD and insomnia: vigilance stabilization through sleep spindles and circadian networks. Neurosci Biobehav Rev 2014;44:183—94.
[8] Arns M, Strehl U. Evidence for efficacy of neurofeedback in ADHD? Am J Psychiatry 2013;170:799—800.
[9] Arns M, de Ridder S, Strehl U, Breteler M, Coenen A. Efficacy of neurofeedback treatment in ADHD: the effects on inattention, impulsivity and hyperactivity: a meta-analysis. Clin EEG Neurosci 2009;40:180—9.
[10] Arns M, Conners CK, Kraemer HC. A decade of EEG Theta/Beta Ratio Research in ADHD: a meta-analysis. J Atten Disord 2013;17:374—83.
[11] Arns M, Heinrich H, Strehl U. Evaluation of neurofeedback in ADHD: the long and winding road. Biol Psychol 2014;95:108—15.
[12] Bakhshayesh AR, Hansch S, Wyschkon A, Rezai MJ, Esser G. Neurofeedback in ADHD: a single-blind randomized controlled trial. Eur Child Adolesc Psychiatry 2011;20:481—91.
[13] Barclay-Goddard RE, Stevenson TJ, Poluha W, Thalman L. Mental practice for treating upper extremity deficits in individuals with hemiparesis after stroke. Cochrane Database Syst Rev 2011:CD005950.
[14] Bauer H, Pllana A. EEG-based local brain activity feedback training-tomographic neurofeedback. Front Hum Neurosci 2014;8:1005.
[15] Bink M, van Nieuwenhuizen C, Popma A, Bongers IL, van Boxtel GJ. Neurocognitive effects of neurofeedback in adolescents with ADHD: a randomized controlled trial. J Clin Psychiatry 2014;75:535—42.
[16] Bink M, van Nieuwenhuizen C, Popma A, Bongers IL, van Boxtel GJ. Behavioral effects of neurofeedback in adolescents with ADHD: a randomized controlled trial. Eur Child Adolesc Psychiatry 2015;24:1035—48.
[17] Bioulac S, Lallemand S, Rizzo A, Philip P, Fabrigoule C, Bouvard MP. Impact of time on task on ADHD patient's performances in a virtual classroom. Eur J Paediatr Neurol 2012;16:514—21.
[18] Budzynski TH, Budzynski HK, Evans JR, Abarbanel A. Introduction to quantitative EEG and neurofeedback. London: Elsevier; 2009.
[19] Cannon RL, Pigott HE, Surmeli T, Simkin DR, Thatcher RW, Van den Bergh W, et al. The problem of patient heterogeneity and lack of proper training in a study of EEG-neurofeedback in children. J Clin Psychiatry 2014;75:289—90.
[20] Carmen J. Passive infrared hemoencephalography: four years and 100 migraines. J Neurotherapy 2004;8:23—51.
[21] Choi SW, Chi SE, Chung SY, Kim JW, Ahn CY, Kim HT. Is alpha wave neurofeedback effective with randomized clinical trials in depression? A pilot study. Neuropsychobiology 2011;63:43—51.
[22] Clemente CD, Sterman MB, Wyrwicka W. Post-reinforcement EEG synchronization during alimentary behavior. Electroencephalogr Clin Neurophysiol 1964;16:355—65.
[23] Coben R, Evans JR. Neurofeedback and neuromodulation techniques and applications. London: Elsevier; 2011.
[24] Coburn KL, Lauterbach EC, Boutros NN, Black KJ, Arciniegas DB, Coffey CE. The value of quantitative electroencephalography in clinical psychiatry: a report by the Committee on Research of the American Neuropsychiatric Association. J Neuropsychiatry Clin Neurosci 2006;18:460—500.
[25] Congedo M, Lubar JF, Joffe D. Low-resolution electromagnetic tomography neurofeedback. IEEE Trans Neural Syst Rehabil Eng 2004;12:387—97.
[26] Dagenais E, Leroux-Boudreault A, El-Baalbaki G, Begin J. Doubting the efficacy/effectiveness of electroencephalographic neurofeedback in treating children with attention-deficit/hyperactivity disorder is as yet unjustified. J Clin Psychiatry 2014;75:778—9.
[27] Daly JJ, Wolpaw JR. Brain-computer interfaces in neurological rehabilitation. Lancet Neurol 2008;7:1032—43.
[28] Davidson RJ. Anterior electrophysiological asymmetries, emotion, and depression: conceptual and methodological conundrums. Psychophysiology 1998;35:607—14.
[29] Davidson RJ, Pizzagalli D, Nitschke JB, Putnam K. Depression: perspectives from affective neuroscience. Annu Rev Psychol 2002;53:545—74.
[30] Deuschl G, Eisen A. Recommendations for the practice of clinical neurophysiology: guidelines of the international federation of clinical physiology. Amsterdam: Elsevier; 1999.
[31] Dobkin BH. Brain-computer interface technology as a tool to augment plasticity and outcomes for neurological rehabilitation. J Physiol 2007;579:637—42.
[32] Durup G, Fessard A. L'électroencéphalogramme de l'homme. Observations psycho-physiologiques relatives à l'action des stimuli visuels et auditifs. L'année Psychologique 1935;36:1—32.
[33] Fink M. Remembering the lost neuroscience of pharmaco-EEG. Acta Psychiatry Scand 2010;121:161—73.
[34] Gevensleben H, Holl B, Albrecht B, Vogel C, Schlamp D, Kratz O, et al. Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. J Child Psychol Psychiatry 2009;50:780—9.
[35] Gevensleben H, Rothenberger A, Moll GH, Heinrich H. Neurofeedback in children with ADHD: validation and challenges. Expert Rev Neurother 2012;12:447—60.
[36] Gevensleben H, Moll GH, Rothenberger A, Heinrich H. Neurofeedback in attention-deficit/hyperactivity disorder — different models, different ways of application. Front Hum Neurosci 2014;8:846.
[37] Gruzelier J. Self-regulation of electrocortical activity in schizophrenia and schizotypy: a review. Clin Electroencephalogr 2000;31:23—9.
[38] Gruzelier JH. EEG-neurofeedback for optimising performance. III: a review of methodological and theoretical considerations. Neurosci Biobehav Rev 2013.
[39] Haenschel C, Baldeweg T, Croft RJ, Whittington M, Gruzelier J. Gamma and beta frequency oscillations in response to novel auditory stimuli: a comparison of human electroencephalogram (EEG) data with in vitro models. Proc Natl Acad Sci USA 2000;97:7645—50.
[40] Hammond D, Bodenhamer-Davis G, CGluck G, Stokes D, Harper S, Trudeau D, et al. Standards of practice for neurofeedback and neurotherapy: a position paper of the International Society for Neurofeedback & Research. J Neurother 2011;15:54—64.
[41] Hegerl U, Hensch T. The vigilance regulation model of affective disorders and ADHD. Neurosci Biobehav Rev 2012;44:45—57.
[42] Hoedlmoser K, Pecherstorfer T, Gruber G, Anderer P, Doppelmayr M, Klimesch W, et al. Instrumental conditioning of human sensorimotor rhythm (12—15 Hz) and its impact on sleep as well as declarative learning. Sleep 2008;31:1401—8.
[43] Holtmann M, Steiner S, Hohmann S, Poustka L, Banaschewski T, Bolte S. Neurofeedback in autism spectrum disorders. Dev Med Child Neurol 2011;53:986—93.
[44] Hughes JR, John ER. Conventional and quantitative electroencephalography in psychiatry. J Neuropsychiatry Clin Neurosci 1999;11:190—208.
[45] Jasper H, Shagass C. Conditionning of the occipital alpha rythm in man. J Exp Psychol 1941;28:373—88.
[46] John ER, Karmel BZ, Corning WC, Easton P, Brown D, Ahn H, et al. Neurometrics. Science 1977;196:1393—410.
[47] Kamiya J. Biofeedback training in voluntary control of EEG alpha rhythms. Calif Med 1971;115:44.
[48] Kim S, Birbaumer N. Real-time functional MRI neurofeedback: a tool for psychiatry. Curr Opin Psychiatry 2014;27:332—6.

Please cite this article in press as: Micoulaud-Franchi J-A, et al. Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice. Neurophysiologie Clinique/Clinical Neurophysiology (2015), http://dx.doi.org/10.1016/j.neucli.2015.10.077

[49] Kluetsch RC, Ros T, Theberge J, Frewen PA, Calhoun VD, Schmahl C, et al. Plastic modulation of PTSD resting-state networks and subjective wellbeing by EEG-neurofeedback. Acta Psychiatry Scand 2014;130:123—36.
[50] Kober SE, Witte M, Ninaus M, Neuper C, Wood G. Learning to modulate one's own brain activity: the effect of spontaneous mental strategies. Front Hum Neurosci 2013;7:695.
[51] Koprivova J, Congedo M, Raszka M, Prasko J, Brunovsky M, Horacek J. Prediction of treatment response and the effect of independent component neurofeedback in obsessive-compulsive disorder: a randomized, sham-controlled, double-blind study. Neuropsychobiology 2013;67:210—23.
[52] Kotchoubey B, Strehl U, Uhlmann C, Holzapfel S, Konig M, Froscher W, et al. Modification of slow cortical potentials in patients with refractory epilepsy: a controlled outcome study. Epilepsia 2001;42:406—16.
[53] Lacroix JM. The acquisition of autonomic control through biofeedback: the case against an afferent process and a two-process alternative. Psychophysiology 1981;18:573—87.
[54] Lansbergen MM, van Dongen-Boomsma M, Buitelaar JK, Slaats-Willemse D. ADHD and EEG-neurofeedback: a double-blind randomized placebo-controlled feasibility study. J Neural Transm 2011;118:275—84.
[55] Lantz DL, Sterman MB. Neuropsychological assessment of subjects with uncontrolled epilepsy: effects of EEG feedback training. Epilepsia 1988;29:163—71.
[56] Lefaucheur JP, Andre-Obadia N, Poulet E, Devanne H, Haffen E, Londero A, et al. [French guidelines on the use of repetitive transcranial magnetic stimulation (rTMS): safety and therapeutic indications]. Neurophysiol Clin 2011;41:221—95.
[57] Leins U, Goth G, Hinterberger T, Klinger C, Rumpf N, Strehl U. Neurofeedback for children with ADHD: a comparison of SCP and Theta/Beta protocols. Appl Psychophysiol Biofeedback 2007;32:73—88.
[58] Linden DE. How psychotherapy changes the brain — the contribution of functional neuroimaging. Mol Psychiatry 2006;11:528—38.
[59] Linden DE. Neurofeedback and networks of depression. Dialogues Clin Neurosci 2014;16:103—12.
[60] Loomis A, Harvey E, Hobart G. Electrical potentials of the human brain. J Exp Psychol 1936;19:249—79.
[61] Mayer K, Wyckoff SN, Strehl U. One size fits all? Slow cortical potentials neurofeedback: a review. J Atten Disord 2012;17:393—409.
[62] McCarthy-Jones S. Taking back the brain: could neurofeedback training be effective for relieving distressing auditory verbal hallucinations in patients with schizophrenia? Schizophr Bull 2012.
[63] McKenzie RF, Ehrisman WJ, Montgomery PS, Barnes RH. The treatment of headache by means of electroencephalographic biofeedback. Headache 1974;13:164—72.
[64] Meisel V, Servera M, Garcia-Banda G, Cardo E, Moreno I. Reprint of ''Neurofeedback and standard pharmacological intervention in ADHD: a randomized controlled trial with six-month follow-up''. Biol Psychol 2014;95:116—25.
[65] Micoulaud Franchi J, Geoffroy P, Fond G, Lopez R, Bioulac S, Philip P. EEG-neurofeedback treatments in children with ADHD: an updated meta-analysis of randomized controlled trials. Front Hum Neurosc 2014, http://dx.doi.org/10.3389/fnhum.2014.00906.
[66] Micoulaud-Franchi JA, Fakra E, Cermolacce M, Vion-Dury J. [Towards a new approach of neurophysiology in clinical psychiatry: functional magnetic resonance imaging neurofeedback applied to emotional dysfunctions]. Neurophysiol Clin 2012;42:79—94.
[67] Micoulaud-Franchi JA, Kotwas I, Lanteaume L, Berthet C, Bastien M, Vion-Dury J, et al. Skin conductance biofeedback training in adults with drug-resistant temporal lobe epilepsy and stress-triggered seizures: a proof-of-concept study. Epilepsy Behav 2014;41:244—50.
[68] Milstein V. Contingent alpha blocking: conditioning or sensitization? Electroencephalogr Clin Neurophysiol 1965;18:272—7.
[69] Nagai Y, Goldstein LH, Fenwick PB, Trimble MR. Clinical efficacy of galvanic skin response biofeedback training in reducing seizures in adult epilepsy: a preliminary randomized controlled study. Epilepsy Behav 2004;5:216—23.
[70] Nestoriuc Y, Martin A. Efficacy of biofeedback for migraine: a meta-analysis. Pain 2007;128:111—27.
[71] Nestoriuc Y, Martin A, Rief W, Andrasik F. Biofeedback treatment for headache disorders: a comprehensive efficacy review. Appl Psychophysiol Biofeedback 2008;33:125—40.
[72] Neuper C, Schlogl A, Pfurtscheller G. Enhancement of left-right sensorimotor EEG differences during feedback-regulated motor imagery. J Clin Neurophysiol 1999;16:373—82.
[73] Niedermeyer E. Alpha rhythms as physiological and abnormal phenomena. Int J Psychophysiol 1997;26:31—49.
[74] Ogrim G, Hestad KA. Effects of neurofeedback versus stimulant medication in attention-deficit/hyperactivity disorder: a randomized pilot study. J Child Adolesc Psychopharmacol 2013;23:448—57.
[75] Oken BS, Salinsky MC, Elsas SM. Vigilance, alertness, or sustained attention: physiological basis and measurement. Clin Neurophysiol 2006;117:1885—901.
[76] Peeters F, Oehlen M, Ronner J, van Os J, Lousberg R. Neurofeedback as a treatment for major depressive disorder — a pilot study. PLoS One 2014;9:e91837.
[77] Peniston E, Kulkosky P. Alpha-theta brainwave neurofeedback therapy for Vietnam veterans with combat-related post-traumatic stress disorder. Med Psychother 1991;4:47—60.
[78] Rayegani SM, Raeissadat SA, Sedighipour L, Rezazadeh IM, Bahrami MH, Eliaspour D, et al. Effect of neurofeedback and electromyographic-biofeedback therapy on improving hand function in stroke patients. Top Stroke Rehabil 2014;21:137—51.
[79] Rice KM, Blanchard EB, Purcell M. Biofeedback treatments of generalized anxiety disorder: preliminary results. Biofeedback Self Regul 1993;18:93—105.
[80] Ros TBJB, Lanius RA, Vuilleumier P. Tuning pathological brain oscillations with neurofeedback: a systems neuroscience framework. Front Hum Neurosci 2014;8:1008.
[81] Schabus M, Heib DP, Lechinger J, Griessenberger H, Klimesch W, Pawlizki A, et al. Enhancing sleep quality and memory in insomnia using instrumental sensorimotor rhythm conditioning. Biol Psychol 2014;95:126—34.
[82] Schneider F, Backes V, Mathiak K. Brain imaging: on the way toward a therapeutic discipline. Eur Arch Psychiatry Clin Neurosci 2009;259(Suppl. 2):S143—7.
[83] Schoenberg PL, David AS. Biofeedback for psychiatryic disorders: a systematic review. Appl Psychophysiol Biofeedback 2014;39:109—35.
[84] Scott WC, Kaiser D, Othmer S, Sideroff SI. Effects of an EEG biofeedback protocol on a mixed substance abusing population. Am J Drug Alcohol Abuse 2005;31:455—69.
[85] Servant D. La relaxation : nouvelles approches, nouvelles pratiques. Paris; 2009.
[86] Sherlin LH, Arns M, Lubar J, Heinrich H, Kerson C, Strehl U, et al. Neurofeedback and basic lerning therory: implications for research and practice. J Neurother 2011;15:292—304.
[87] Siegle G, Ghinassi F, Thase M. Neurobehavioral therapies in the 21st century: summary of an emerging field and an extended example of cognitive control training for depression. Cogn Ther Res 2007;31:235—62.
[88] Silberstein SD. Practice parameter: evidence-based guidelines for migraine headache (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2000;55:754—62.

[89] Silvanto J, Pascual-Leone A. State-dependency of transcranial magnetic stimulation. Brain Topogr 2008;21:1—10.
[90] Siniatchkin M, Hierundar A, Kropp P, Kuhnert R, Gerber WD, Stephani U. Self-regulation of slow cortical potentials in children with migraine: an exploratory study. Appl Psychophysiol Biofeedback 2000;25:13—32.
[91] Skinner BF. Are theories of learning necessary? Psychol Rev 1950;57:193—216.
[92] Sonuga-Barke EJ, Brandeis D, Cortese S, Daley D, Ferrin M, Holtmann M, et al. Nonpharmacological interventions for ADHD: systematic review and meta-analyses of randomized controlled trials of dietary and psychological treatments. Am J Psychiatry 2013;170:275—89.
[93] Sonuga-Barke E, Brandeis D, Cortese S, Daley D, Danckaerts M, Dopfner M, et al. Response to Chronis-Tuscano et al. and Arns and Strehl. Am J Psychiatry 2013;170:800—2.
[94] Steiner NJ, Sheldrick RC, Gotthelf D, Perrin EC. Computer-based attention training in the schools for children with attention-deficit/hyperactivity disorder: a preliminary trial. Clin Pediatr (Phila) 2011;50:615—22.
[95] Sterman MB. Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. Clin Electroencephalogr 2000;31:45—55.
[96] Sterman MB, Howe RC, Macdonald LR. Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. Science 1970;167:1146—8.
[97] Stewart JL, Bismark AW, Towers DN, Coan JA, Allen JJ. Resting frontal EEG asymmetry as an endophenotype for depression risk: sex-specific patterns of frontal brain asymmetry. J Abnorm Psychol 2010;119:502—12.
[98] Stoeckel LE, Garrison KA, Ghosh S, Wighton P, Hanlon CA, Gilman JM, et al. Optimizing real time fMRI neurofeedback for therapeutic discovery and development. Neuroimage Clin 2014;5:245—55.
[99] Stokes DA, Lappin MS. Neurofeedback and biofeedback with 37 migraineurs: a clinical outcome study. Behav Brain Funct 2010;6:9.
[100] Strehl U. Slow cortical potentials neurofeedback. J Neurother 2009;13:117—26.
[101] Strehl U. What learning theories can teach us in designing neurofeedback treatments. Front Hum Neurosci 2014;8:894.
[102] Strijkstra AM, Beersma DG, Drayer B, Halbesma N, Daan S. Subjective sleepiness correlates negatively with global alpha (8—12 Hz) and positively with central frontal theta (4—8 Hz) frequencies in the human resting awake electroencephalogram. Neurosci Lett 2003;340:17—20.
[103] Sulzer J, Haller S, Scharnowski F, Weiskopf N, Birbaumer N, Blefari ML, et al. Real-time fMRI neurofeedback: progress and challenges. Neuroimage 2013;76:386—99.
[104] Surmeli T, Ertem A, Eralp E, Kos IH. Schizophrenia and the efficacy of qEEG-guided neurofeedback treatment: a clinical case series. Clin EEG Neurosci 2012;43:133—44.
[105] Tan G, Thornby J, Hammond DC, Strehl U, Canady B, Arnemann K, et al. Meta-analysis of EEG biofeedback in treating epilepsy. Clin EEG Neurosci 2009;40:173—9.
[106] Thatcher LG, Barlow JF. Thirteen-year-old girl with one month history of back pain and limp. S D J Med 1976;29:31—5.
[107] van Dongen-Boomsma M. Dr van Dongen-Boomsma replies. J Clin Psychiatry 2014;75:779.
[108] van Dongen-Boomsma M, Vollebregt MA, Slaats-Willemse D, Buitelaar JK. Dr van Dongen-Boomsma and colleagues reply. J Clin Psychiatry 2014;75:290.
[109] Vollebregt MA, van Dongen-Boomsma M, Slaats-Willemse D, Buitelaar JK. What future research should bring to help resolving the debate about the efficacy of EEG-neurofeedback in children with ADHD. Front Hum Neurosci 2014;8:321.
[110] Witte M, Kober SE, Ninaus M, Neuper C, Wood G. Control beliefs can predict the ability to up-regulate sensorimotor rhythm during neurofeedback training. Front Hum Neurosci 2013;7:478.
[111] Wolpaw JR, Birbaumer N, McFarland DJ, Pfurtscheller G, Vaughan TM. Brain-computer interfaces for communication and control. Clin Neurophysiol 2002;113:767—91.
[112] Zuberer A, Drandeis D, Drechsler R. Are treatment effects of neurofeedback training in children with ADHD related to the successful regulation of brain activity? A review on the learning of regulation of brain activity and a contribution to the discussion on specificity. Front Hum Neurosc 2015, http://dx.doi.org/10.3389/fnhum.2015.00135.

Please cite this article in press as: Micoulaud-Franchi J-A, et al. Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice. Neurophysiologie Clinique/Clinical Neurophysiology (2015), http://dx.doi.org/10.1016/j.neucli.2015.10.077