NEUROFEEDBACK/BIOFEEDBACK FOR BEHAVIORAL AND SUBSTANCE USE DISORDERS

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BENEFIT CONSIDERATIONS

Before using this policy, please check the member-specific benefit plan document and any federal or state mandates, if applicable.

COVERAGE RATIONALE

Neurofeedback/biofeedback (with or without EEG guidance) is unproven for the treatment of individuals with behavioral and substance use disorders, including attention-deficit/hyperactivity disorder (ADHD), depression, anxiety, obsessive-compulsive disorder, posttraumatic stress disorder, alcohol/drug abuse, and autism spectrum disorder.

The reviewed evidence, including randomized controlled trials and systematic reviews, does not clearly demonstrate a treatment effect of neurofeedback/biofeedback on symptoms of ADHD. Many of these reviewed studies contained a number of significant limitations. Additionally, there is a lack of well-designed clinical trials with sufficient sample sizes demonstrating the effectiveness of neurofeedback/biofeedback in the treatment of other behavioral and substance use disorders.

DESCRIPTION OF SERVICES

Neurofeedback/biofeedback therapy teaches individuals to alter EEG patterns or other physiological processes through real-time visual, auditory or other feedback and learning. As the individual’s EEG pattern or other physiological process improves or is learned through the feedback, symptoms of ADHD or other behavioral disorders are expected to improve.

In some instances, neurofeedback and quantitative electroencephalography (qEEG) are used in combination. When this occurs, the individual’s EEG pattern is analyzed by qEEG, and an individualized feedback protocol is defined for the individual based on the reported findings.
Summary of Clinical Evidence

The reviewed evidence does not clearly demonstrate a treatment effect of neurofeedback/biofeedback on symptoms of attention-deficit/hyperactivity disorder (ADHD). Some of the reviewed studies compared the effectiveness of neurofeedback/biofeedback to stimulant medications, such as methylphenidate. In these studies, neurofeedback/biofeedback and medications were typically found to have similar efficacy, with no significant differences found between groups in terms of outcome.

A number of limitations among the reviewed studies limit conclusions regarding the effectiveness of neurofeedback/biofeedback for the treatment of ADHD. Many of the earlier trials that were reviewed permitted open treatment choice and had small sample sizes. Other studies reported difficulties isolating the effects of neurofeedback in participants who were also taking stimulant medications at the time of study.

A number of professional societies have issued statements and proposed treatment guidelines for ADHD. Many of them agree that a current lack of efficacy in larger, well-controlled and methodologically sound trials prevent neurofeedback/biofeedback from being considered a front-line treatment for ADHD.

The reviewed evidence indicates that there is a lack of well-designed clinical trials with sufficient sample sizes demonstrating the effectiveness of neurofeedback/biofeedback in the treatment of other behavioral and substance use disorders, such as depression, anxiety, obsessive compulsive disorder, posttraumatic stress disorder, alcohol/drug abuse, and autism spectrum disorder.

Clinical Trials: ADHD

A meta-analysis conducted by Cortese and colleagues (2016) examined the effects of neurofeedback on ADHD symptoms and neuropsychological deficits in randomized controlled trials (RCTs) made up of children and adolescents with ADHD. The authors found and included a total of 13 RCTs, totaling 520 participants with ADHD in all. Trials where medication was part of normal clinical provision in either the control or active arm were permitted. Outcome measures included ADHD symptoms and neuropsychological laboratory-based measures. There was a small-to-moderate effect found on inattention, impulsivity/hyperactivity and total ADHD symptoms when proximal assessments were the outcome. However, the effects dropped to non-statistically significant levels for total ADHD and inattention symptoms in sensitivity analyses considering only trials with active/sham controls. When “probably blinded” outcomes were included in the analysis, effect size for outcomes dropped further, and none were significant. The authors concluded that the evidence from RCTs currently fails to support neurofeedback as an effective treatment for ADHD.

Janssen and colleagues (2016) conducted a randomized controlled trial to compare the effects of neurofeedback (NF), methylphenidate (MPH), and physical activity (PA) in children with DSM-IV-diagnosed ADHD. The study used a multicenter three way parallel group RCT design, enrolling a total of 112 children, all between the ages of 7 and 13. Results found that the MPH group showed a specific increase in P3 amplitude (demonstrating improved response inhibition) when compared to the NF and PA groups. The authors note that their finding of stimulants being the lone treatment to demonstrate significant improvement in response inhibition was in line with recent doubts on efficacy and specificity of NF as a treatment for ADHD.

Bink and colleagues (2016) conducted a one-year follow-up of neurofeedback (NFB) treatment in adolescents with ADHD, compared with treatment as usual (TAU). Using the study population and parameters described in Bink, et al (2014), a total of 60 adolescents (41 receiving NFB+TAU and 19 receiving TAU only) were followed-up with after one year. Attrition analysis showed that participants who dropped out from the study did not differ from the completers in terms of characteristics or behavioral measures. The authors found that after the one year period, self-reported inattention levels had decreased, and neurocognitive task performance became faster, regardless of treatment group. The authors caution their use of post hoc analyses and that by splitting up the treatment groups, the relatively small subgroups led to a subsequent decrease in power. They note that further research, comparing neurofeedback and stimulant medication is essential to determine whether neurofeedback can be a long-term alternative for stimulant medication.

Using the same study population and parameters as Bink, et al (2014), Bink and colleagues (2015) investigated the additional value of neurofeedback on behavior over treatment as usual (TAU) among adolescents with ADHD and comorbid disorders. For this study, primary behavioral outcome measures, assessed both before and after the intervention, included the ADHD-rating scale, the Youth Self Report, and the Child Behavior Checklist. For all of the outcome measures, behavioral problems, mainly inattention, were found to decrease equally for both groups with medium to large effect sizes. The authors concluded that the combination of neurofeedback combined with treatment...
42% in the placebo-neurofeedback group rated as “minimally improved”; differences between the groups were not significant. Total adverse events decreased significantly over time, and similarly in the two groups. Post-hoc analyses of a randomized, controlled trial with two- and six-month follow-up conducted by Meisel and colleagues (2012) evaluated the efficacy of neurofeedback compared to standard pharmacological intervention in the treatment of 27 children (aged 7-14) with ADHD. In the post-assessment, the pharmacological group had significantly lower levels of symptoms compared to the neurofeedback group. Results found that some neurofeedback protocol-specific effects (increased response speed, reduced attentional resource allocation during target processing) were obtained; however, due to the limited sample size, medium effects did not reach a level of significance. Self-regulation skills were not sufficiently learned during theta/beta and SCP training, which the authors considered a limitation. The authors note that future studies including larger sample sizes are needed to evaluate the protocol-specific effects on attention and motor system excitability.

As a follow-up to Steiner, et al (2014a), Steiner and colleagues (2014b) next evaluated sustained improvement from the intervention at 6-month follow-up. Parent response rates were 90%. At six months post-intervention, the neurofeedback participants maintained significant gains on Conners 3-P (Inattention; Executive Functioning; Hyperactivity/Impulsivity), and BRIEF subscales, including the Global Executive Composite. These remained significantly greater than gains found among children in the cognitive training and control conditions. At the 6-month follow-up, neurofeedback participants also maintained the same stimulant medication dosage, while the cognitive training and control conditions showed statistically and clinically significant increases.

Steiner and colleagues (2014a) conducted a randomized control trial to evaluate the efficacy of 2 computer attention training systems administered in school for children with ADHD. The trial took place in 19 public elementary schools. All children were in either second or fourth grade and were randomized to 40 sessions of neurofeedback (n = 34) or cognitive training (n = 34), or to a control (n = 36) condition that received computer attention training treatment the following school year. There were no statistical differences between randomization conditions at baseline or between participants who completed or did not complete the intervention. Results found that children in the neurofeedback group showed significant improvement compared with those in the control condition on many indices. Children who received cognitive training showed no improvement compared to the baseline condition. Children in the neurofeedback condition showed significant improvements compared to those in the cognitive training condition on Conners 3-P Executive Functioning, all BRIEF summary indices, SKAMP Attention, and Conners 3-T Inattention subscales. Stimulant medication dosage in methylphenidate equivalencies significantly increased for children in the cognitive training and control conditions, but not for those in the neurofeedback condition. Limitations noted by the authors include that both children and parents were aware of the child’s intervention condition, and that projected sample size based on the power analysis was not achieved.

A randomized, placebo-controlled trial conducted by van Dongen-Boomsma and colleagues (2012) assessed the efficacy and safety of EEG neurofeedback in children with ADHD. This was a follow-up to the pilot study conducted by Lansbergen, et al (2011). Results found that ADHD symptoms decreased over time in both groups. Similar results were observed when inattentive and hyperactive/impulsive scores were analyzed separately. Teacher-rated ADHD symptoms decreased significantly over time, without a difference between groups. On the CGI-I scale, 18% of children in the EEG-neurofeedback group were rated as “much improved”, with 41% in the EEG-neurofeedback and 42% in the placebo-neurofeedback group rated as “minimally improved”; differences between the groups were not significant. Total adverse events decreased significantly over time, and similarly in the two groups. Post-hoc analyses did not reveal any significant treatment effect for any outcome. Based on the results, the authors concluded that EEG-neurofeedback was not superior to placebo-neurofeedback in improving symptoms in children with ADHD.

A randomized, controlled trial with two- and six-month follow-up conducted by Meisel and colleagues (2012) evaluated the efficacy of neurofeedback compared to standard pharmacological intervention in the treatment of 27 children (aged 7-14) with ADHD. In the post-assessment, the pharmacological group had significantly lower levels of...
inattention according to teachers, and better scores in math. At two-month follow-up, ADHD-RS inattention and math scores were significantly better for the pharmacological group. At six-month follow-up, no significant differences were found between the two groups in inattention, hyperactivity, or functional impairment. The authors conclude that, taking into consideration large and medium effects sizes throughout the different behavioral evaluations, neurofeedback and medication effects were comparable. However, the authors caution that the sample was limited, and not all neurofeedback participants remained medication-free at six-month follow-up, making it unfeasible to conclude that neurofeedback and medication are equivalent treatments for ADHD.

A controlled, randomized clinical study of children and adolescents with ADHD conducted by Duric and colleagues (2012) evaluated the effects of neurofeedback on the core symptoms of ADHD. Of an initial subject pool of 275 children and adolescents ranging in age from 6-18 years, 91 participated in 30 sessions of intensive neurofeedback. No significant differences in demographic factors or ADHD core symptoms were noted between groups at baseline. Results found that parents reported significant effects for all three treatment groups, while no significant differences between the treatment groups were observed.

A double-blind, randomized, placebo-controlled feasibility (pilot) study conducted by Lansbergen and colleagues (2011) explored the initial efficacy of individualized EEG-neurofeedback training in children with ADHD. A total of 14 children (aged 8-15 years) with ADHD were randomly allocated to either 30 sessions of EEG-neurofeedback (n = 8) or placebo feedback (n = 6). Blinded analysis demonstrated clinical improvement over time (reduced ADHD DSM-IV symptoms rated by investigator), but did not reveal significant differences between the groups. The authors suggest that the findings indicate that behavioral improvements after individualized EEG-neurofeedback training may not be caused by the ability to self-regulate brain activity, but rather by unspecific effects such as invested time and attention, therapist interaction, or expectancy. Given the small sample size, the authors note that it is preliminary to conclude that individualized EEG-neurofeedback training is not effective in improving ADHD symptoms.

In a randomized, double-blind, crossover, placebo-controlled study, deBeus and Kaiser (2011) studied whether participants in a neurofeedback (NF) intervention who learned to change their EEG would improve in functioning compared to a placebo condition. Children with primary ADHD and co-occurring diagnoses were permitted, as well as those using only stimulant medication. Because there were distinct differences in results between those who responded to neurofeedback training and those who did not, only those who showed response were included in the authors’ analysis. The findings demonstrated significant positive treatment effects for teacher rating scales and IVA quotients for successful neurofeedback learners. Parental ratings improved regardless of whether a child was a learner or a placebo participant. The authors point out several limitations: the use of a crossover design; allowing children to be medicated during neurofeedback training; not using EEG profiling to guide neurofeedback protocol choices; and not enough neurofeedback sessions.

In a single-blind, randomized controlled trial, Bakhshayesh and colleagues (2011) compared the effects of two matched biofeedback training variants on the primary symptoms of ADHD. The first variant was EEG neurofeedback (NF) aimed at theta/beta ratio reduction. The second variant was EMG biofeedback (BF) aimed at forehead muscle relaxation. A total of 35 children with ADHD, aged 6-14 years old, were randomly assigned to either the NF group (n = 18) or the BF group (n = 17). Results found that training reduced theta/beta ratios and EMG levels in both groups. Parents reported significant reductions in primary ADHD symptoms, and inattention improvements in the NF group were higher compared to the BF group. NF training also improved attention and reaction times on the psychometric measures. Regarding hyperactivity and impulsivity symptoms, the results implied that non-specific factors, such as behavioral contingencies, self-efficacy, structured learning environment, and feed-forward processes, may also contribute to the positive behavioral effects induced by neurofeedback training.

Gevensleben and colleagues (2010) conducted a six-month follow-up of treatment effects from Gevensleben, et al 2009. Of the children with complete data from the previous study, follow-up information was analyzed in 61 children on a per-protocol basis. Reductions of inattention and hyperactivity/impulsivity at follow-up (compared to pre-training) were 25-30% in the neurofeedback group compared to 10-15% in the control group. The authors note the large number of dropouts, and also the non-blind design, which could not rule out the contribution of unspecific effects to the behavioral effects, and that the low responder rate and the portion of children starting a medication argue against neurofeedback as a stand-alone intervention for children with ADHD. They emphasize a need for further research to determine optimal neurofeedback protocol and adequate number of treatment sessions.

In a multisite, randomized controlled study, Gevensleben and colleagues (2009) evaluated the clinical efficacy of neurofeedback in children with ADHD. A total of 102 children with ADHD, aged 8 to 12 years, participated. Subjects were randomly assigned to either neurofeedback or computerized attention skills training, with no significant pre-training differences in demographic, psychological, or clinical variables. For parent and teacher ratings, improvements
in the neurofeedback group were superior to those of the control group. For the primary outcome measure (parent-rated FBB-HKS total score), the effect size was .60. The authors note that owing to various restrictions of the training setting, it may not be appropriate to indirectly compare the efficacy of neurofeedback based on the results of this RCT with other treatment approaches, such as long-acting medications with similar effect sizes. The authors also point to a need for further studies to replicate these findings, control for factors not covered in this study, further isolate specific effects of neurofeedback, and address how to optimize neurofeedback training.

An open-label study by Monastra, et al (2002) examined the effects of EEG biofeedback and Ritalin on the primary symptoms of ADHD and neuropsychological and electrophysiological measures, while controlling for other commonly provided types of clinical interventions. A total of 100 children, ages 6-19 and their parents participated in the study. Based on parental preference, patients with ADHD participated in a 1-year comprehensive program including medication management, parent counseling and school consultation. Observations were first recorded while participants were still being treated with Ritalin. A second post-treatment assessment was conducted after a 1-week period in which no stimulant therapy was provided. Significant improvement was noted on the TOVA and the ADDES when participants were tested while using Ritalin. Only those who had received EEG biofeedback sustained these gains when tested without Ritalin. The results of the QEEG-Scan revealed significant reduction in cortical slowing only in those participants who had received EEG biofeedback. The authors conclude that the findings of this study are supportive of multimodal treatment models that include parent counseling and EEG biofeedback, in addition to stimulant therapy. They comment that long-term follow-up studies examining the relationship between EEG biofeedback and stimulant dosing patterns are required.

Systematic Reviews/Meta-Analyses: ADHD
A systematic review and double-blind placebo-controlled study conducted by Vollebregt and colleagues (2014) determined if EEG-neurofeedback improves neurocognitive functioning in children with ADHD. First, 10 randomized controlled trials, including a range of different sample sizes and control conditions, were examined. The neurofeedback protocol, as well as the duration, frequency, and number of sessions varied between studies. Three of the ten studies reported significant improvement on at least one neurocognitive variable for the neurofeedback condition superior to the control condition. The authors concluded that these studies had many methodological limitations and the majority failed to show positive neurocognitive effects of neurofeedback. Next, the authors conducted a double-blind, placebo-controlled treatment trial for children aged 8-15 with ADHD. Forty-one children were randomly allocated to EEG-neurofeedback (n = 22) or placebo-neurofeedback (n = 19) for 30 sessions, twice a week. Results from the trial found no significant treatment effect on any neurocognitive variables (sustained attention dots task, visuospatial sequencing, digit span WISC-III, Rey Auditory-Verbal Learning Test, instrumental learning task, time production task, time reproduction task). The authors conclude that the study was unable to establish positive treatment effects on neurocognitive functioning after EEG-neurofeedback when compared to placebo-neurofeedback.

A systematic review of pharmacological and psychosocial treatments for adolescents with ADHD was conducted by Sibley and colleagues (2014). This review included three studies (two controlled trials and one open trial) that evaluated two classes of cognitive enhancement training: neurofeedback or EEG biofeedback, and working memory training. The authors note that all reviewed studies employed methodologically sound multi-informant assessment strategies, but no studies examined intervention effects on impairment. Available symptom effect sizes for the cognitive enhancement training interventions suggested that overall, these treatments did not produce significant gains.

A meta-analytic review of nonpharmacological treatments for ADHD was conducted by Hodgson and colleagues (2014). Included in the 14 selected studies was neurofeedback therapy (3 studies). The authors found that two interventions most commonly resulted in improvement in the treatment groups across a range of different outcomes measures: neurofeedback and behavior modification. Improvement was observed in attention, self-control, and performance on the Digit Span test for neurofeedback treatment. One major limitation noted was a focus on studies comparing treatment and control groups while excluding all studies using within-subjects designs. While minimizing the influence of placebo effects, this reduced the total number of studies. The authors conclude that this meta-analytic study provides initial evidence that nonpharmacological treatments, such as neurofeedback, have potential in the treatment of children with ADHD and that more and better outcome research is necessary.

A systematic review and meta-analysis of 54 studies conducted by Sonuga-Barke and colleagues (2013) determined the efficacy of various dietary and psychological ADHD treatments. Included among the psychological treatments was neurofeedback. The outcome measure was pre- to post-treatment change in total ADHD symptom severity measured at the first post-treatment assessment. Results from ADHD-specific symptom scales were used where available. Of the eight neurofeedback trials with data for most assessments, four reported "probably blinded" assessments. Significant
treatment effects were seen for most assessments. These were substantially reduced and fell short of statistical significance for the “probably blinded” assessments. Sensitivity analysis to test for medication effects was not possible because of the small number of no-medication trials. The authors conclude that based on these results, the value of psychological approaches that directly target neuropsychological processes should be further investigated. Also noted is that evidence of efficacy from blinded assessments is required before behavioral interventions such as neurofeedback are likely to be supported as ADHD treatments.

Moriyama and colleagues (2012) reviewed literature on the effectiveness and specificity of neurofeedback for the treatment of ADHD. The authors found 3 systematics reviews on the use of neurofeedback for ADHD, and 6 randomized controlled trials not been included in these reviews. The authors note that the available studies point to a possible efficacy and specificity of neurofeedback for the treatment of ADHD, but additional large, randomized controlled studies using blind assessment procedures are needed to clarify this issue. Very few studies were found to compute response ratio to neurofeedback, and since sample sizes are small for the majority of the included studies, most do not investigate possible predictors of response. No safety issues emerged with neurofeedback, with studies having low dropout rates and no discontinuation due to side effects. Very few of the studies examined long-term effects of neurofeedback, but the few studies that did found promising results. The authors note that while neurofeedback has been efficacious in the majority of trials conducted so far, there is not enough data to support the use of neurofeedback as a monotherapy for ADHD. The great majority of studies tested neurofeedback as an adjunctive treatment. The authors conclude that although some data suggest possible efficacy and specificity of neurofeedback for the treatment of ADHD, considerable work is still needed to determine the contribution of specific in comparison to nonspecific components, when and how to use neurofeedback in routine clinical practice, and how to implement neurofeedback standards covering the fidelity of the EEG and artifact control methodology.

Arns, et al (2009) conducted a meta-analysis to investigate the effects of neurofeedback and stimulant medication on the core symptoms of ADHD. All included subjects had a primary diagnosis of ADHD and study designs could be controlled, prospective, or retrospective in nature. All neurofeedback was provided in a standardized manner, and no more than two treatment protocols (SCP; theta/beta) were used. Standardized pre- and post-assessment means and standard deviations (SDs) were available for at least 1 of the following domains: hyperactivity, inattentiveness, or CPT commission errors. If there was not sufficient information available, the study was excluded. Fifteen studies met all criteria and were included in the meta-analysis. Two separate effect sizes were calculated - for the controlled between-subject design studies (n = 10), the effect size of the neurofeedback group as compared to the control group was calculated. Effect size was also calculated and plotted for all ADHD children treated with neurofeedback from both the controlled and within-subject designs. For all controlled studies, there were a total of 476 subjects, and for the pre/post-design studies there were a total of 718 subjects. Drop-out rates were reported in 5 studies, and around 10%. Six studies employed randomized allocation of subjects, and 3 studies compared neurofeedback with stimulant medication. Results found that both the prospective controlled studies and those studies employing a pre- and post-design found large effect sizes for neurofeedback (compared to control) on impulsivity and inattention, and a medium effect size for hyperactivity. No differences were found between neurofeedback studies in medicated vs. unmedicated subjects. None of the studies comparing neurofeedback with stimulant medication used random assignment; participants were allowed to self-select the treatment of their preference. As a result, it is noted that more studies using randomization and larger sample sizes are needed to investigate further how neurofeedback compares to stimulants in the treatment of ADHD. The authors also note that further research on the impact of medication on neurofeedback is needed. Long-term effects could not be addressed in the meta-analysis, though several studies did report follow-up results showing that the clinical effects of neurofeedback were stable.

Clinical Trials: Other Behavioral Disorders

Koprivova and colleagues (2013) assessed the effect of neurofeedback (NFB) on EEG and clinical symptoms in patients with obsessive-compulsive disorder (OCD). A randomized, double-blind, parallel design of 20 inpatients with OCD was conducted, with patients undergoing 25 sessions of NFB (n = 10) or sham feedback (SFB; n = 10). All patients were either drug free (n = 5) or medicated with SSRIs (n = 15). Results found that patients receiving NFB showed a higher percentage of improvement on compulsion scores when compared to patients receiving SFB, and that NFB led to a small and non-significant EEG change in the direction of training. Other clinical outcome measures did not differ between the NFB and SFB groups. The authors note that further research is necessary to elucidate the relationship between EEG patterns and their role in treatment responsiveness in patients with OCD.

Kouijzer and colleagues (2013) conducted a randomized controlled trial to evaluate the effects of EEG-biofeedback in autism spectrum disorder (ASD). The randomized pretest-posttest control group design included a blinded active comparator and six month follow-up. A total of 38 participants were randomly assigned to either EEG-biofeedback (n = 13), skin conductance (SC)-biofeedback (n = 12), or wait list group (n = 13). Results found that 54% of the EEG group participants (n = 7) significantly reduced delta and/or theta power and were identified as EEG-regulators. However, in these individuals, no statistically significant reductions of ASD symptoms were observed, though they did
show improvement in cognitive flexibility when compared to participants who regulated SC. The authors conclude that the results are inconclusive with respect to the clinical application of EEG-biofeedback in children and adolescents with ASD.

Knox and colleagues (2011) evaluated the effects of a game-based biofeedback intervention on symptoms of anxiety and depression among a pediatric sample. A total of 24 individuals (aged 9-17 years) reporting symptoms of anxiety or diagnosed with an anxiety disorder completed the study, with 12 individuals participating in a biofeedback intervention and 12 assigned to a wait list comparison group. Results found the intervention group to have reduced anxiety and depression scores on the outcome measures, with the largest effect size found on the depression scale. The authors conclude that, despite the promise of these results, more research is needed to examine whether these outcomes can be replicated in children and youths with anxiety and depression. The authors additionally note that the study contained a small sample size, a non-random assignment to groups, and an unknown contribution of each component of treatment intervention (e.g., psychoeducation, biofeedback) to the outcomes.

Lande and colleagues (2010) conducted a pilot study to investigate the potential effectiveness of heart rate variability biofeedback as a complementary treatment for posttraumatic stress disorder (PTSD). Active duty service members deployed to an area of combat operations were alternately assigned to a treatment as usual (TAU) control group (n = 17) or a group consisting of TAU with the addition of biofeedback (n = 22). Outcomes were measured using the posttraumatic stress disorder checklist (PCL)-Military version and the Zung Self-Rating Depression Scale, administered before treatment and at the conclusion of three weeks of biofeedback therapy. Results found that biofeedback did not produce a measurable improvement in either PTSD or depression scores, though subjective satisfaction with biofeedback was found among many of the participants. The authors conclude that the study’s exploratory design can be strengthened by future research, and that a more empirical study, using strict random sampling techniques and a sham biofeedback unit could improve the potential generalizability of results.

**Systematic Reviews/Meta-Analyses: Other Behavioral Disorders**

Blase and colleagues (2016) analyzed the effectiveness of HRV-BFB (hearthrate variability biofeedback) as an additional psychophysiological treatment for depression and PTSD. The review included 789 studies, including 6RCTs. Results indicate that HRV is popular in the literature, but has not been reviewed systematically. The authors note that there may be clinical improvement when HRVB training is used to treat depression and PTSD when combined with psychotherapy. The authors conclude that additional research is necessary in larger groups for stress-related disorders in psychotherapy.

Bergemann and colleagues (2016) conducted a meta-analysis of studies investigating the efficacy of EEG neurofeedback in the treatment of psychiatric disorders. A total of 30 studies were included (n = 1171) and evaluated neurofeedback for ADHD, autism, obsessive-compulsive disorder, generalized anxiety disorder and depression. The majority of studies included a passive-semi-active control group, with only three placebo-controlled trials identified. Twenty-one of the thirty studies used randomization procedures. The authors’ analysis found small to medium effect sizes for treating the symptoms of ADHD, with varying results for its effect on other diagnoses. The authors conclude that a lack of methodologically sound studies prevents evidence-based conclusions on the efficacy of EEG neurofeedback in the treatment of various psychiatric disorders. They recommend that future studies are carefully planned and executed, including power calculations to establish required sample sizes, randomization, blinding and adequate control conditions, to assess whether neurofeedback is a viable treatment option in the field of psychiatry.

Schoenber and David (2014) conducted a systematic review to explore the current therapeutic use of biofeedback for a range of psychiatric disorders, including addictions, anxiety disorders, autism spectrum disorders, depressive disorders, dissociative disorders, personality disorders, and psychoses. A total of 63 articles were included in the review. EEG biofeedback was used in 32% of the reviewed studies; with 29% incorporating electromyographic (EMG), 16% heart rate variability (HRV) and/or sole respiration, 6% heart rate, 5% electrodermal (EDA) and 3% thermal biofeedback methodologies. Anxiety disorders were the most commonly treated among the reviewed studies (68%). The review found 81% of articles to report some level of clinical amelioration related to biofeedback exposure, and 65% to a statistically significant level of symptom reduction. However, the review highlights a lack of standardization amongst biofeedback studies for psychiatric disorders, and methods/results sections were inconsistent in structure and lacking empirical detail, resulting in the exclusion of several studies from the review. The authors note that Level 1 (“not empirically supported”) studies were not included in the review because of exclusion criteria, potentially skewing the overall evaluation of biofeedback treatments used in psychiatric domains. The authors conclude that further development of standardized controlled methodological protocols tailored for specific disorders and guidelines to generate comprehensive reports may contribute towards establishing the value of biofeedback interventions within mainstream psychiatry.
Holtmann and colleagues (2011) reviewed 15 studies on the effectiveness of neurofeedback as a method of treatment on the core symptoms of autism spectrum disorder (ASD). The review found that the existing evidence does not support the use of neurofeedback in the treatment of ASD. The authors hypothesized that those studies with outcomes in favor of neurofeedback may be showing an improvement in comorbid attention-deficit/hyperactivity disorder symptoms, rather than a true improvement in core ASD symptoms. The authors conclude that a number of methodological limitations will need to be addressed in future studies on neurofeedback in the treatment of ASD.

**Professional Societies**

**American Academy of Pediatrics (AAP):** In the AAP’s Clinical Practice Guideline for the Diagnosis, Evaluation, and Treatment of ADHD in Children and Adolescents (2011), the section titled “Areas for Future Research” includes “study of medications and other therapies used clinically but not approved by the FDA for ADHD, such as electroencephalographic biofeedback.”

The appendix to the AAP’s Clinical Practice Guidelines for the Treatment of ADHD in Children and Adolescents (2011) states that alternative therapies for treatment of ADHD...include “large doses of vitamins, essential fatty acids, and other dietary alterations; chelation; and electroencephalographic (EEG) biofeedback”. The appendix notes that there is “insufficient evidence to determine whether these therapies lead to changes in core symptoms of ADHD or function, and for many of them, there is limited information about their safety. For these reasons, these therapies cannot be recommended”.

AAP guidance material titled “ADHD, What Every Parent Needs to Know” (2011) states that studies on the use of neurofeedback to date have been criticized for lacking appropriate controls or random assignment of test subjects to the treatment/sham treatment groups.

**Institute for Clinical Systems Improvement (ICSI):** Guidance material from the ICSI (Dobie, et al 2012) states that neurofeedback has been demonstrated in one randomized, controlled clinical trial to be significantly better than a computerized attention skills training control and that ADHD symptoms were moderately improved. Long-term benefits have not been definitively proven. The cost and time involved in treatment need to be taken into account. Neurofeedback for ADHD lacks sufficient research support. Treatment response rates have not reached the level shown with psychostimulant medications; therefore neurofeedback cannot be recommended as an alternative to medication use in ADHD.

**American Psychiatric Association (APA):** According to the APA, a substantial number of studies of TMS have been conducted, but most have had small sample sizes, and the studies overall have yielded heterogeneous results. Further complicating the interpretation of the TMS literature is the variability in stimulation intensities (relative to the motor threshold), stimulus parameters (e.g., pulses/second, pulses/session), anatomical localization of stimulation, and number of TMS sessions in the treatment course.

**U.S. FOOD AND DRUG ADMINISTRATION**

Neurofeedback/biofeedback equipment registered with the FDA is considered a medical device. At this time, neurofeedback has been approved by the FDA for purposes of relaxation therapy.

The FDA has granted qEEG a Class II exemption. qEEG analysis involves comparison of the patient’s EEG to a reference population. The norms used for comparison are FDA-registered, and include cross-validation statistics and peer reviewed publications.

**CENTERS FOR MEDICARE AND MEDICAID SERVICES**

A National Coverage Determination (NCD) is available for biofeedback therapy. Biofeedback therapy is covered under Medicare only when it is reasonable and necessary for the individual patient for muscle re-education of specific muscle groups or for treating pathological muscle abnormalities of spasticity, incapacitating muscle spasm, or weakness, and more conventional treatments (heat, cold, massage, exercise, support) have not been successful. This therapy is not covered for treatment of ordinary muscle tension states or for psychosomatic conditions.

**APPLICABLE CODES**

The following list(s) of procedure and/or diagnosis codes is provided for reference purposes only and may not be all inclusive. Listing of a code in this policy does not imply that the service described by the code is a covered or non-covered health service. Benefit coverage for health services is determined by the member-specific benefit plan
document and applicable laws that may require coverage for a specific service. The inclusion of a code does not imply any right to reimbursement or guarantee claim payment. Other Policies and Coverage Determination Guidelines may apply.

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<td>90875</td>
<td>Individual psychophysiological therapy incorporating biofeedback training by any modality (face-to-face with the patient), with psychotherapy (e.g., insight oriented, behavior modifying or supportive psychotherapy); 30 minutes</td>
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<tr>
<td>90876</td>
<td>Individual psychophysiological therapy incorporating biofeedback training by any modality (face-to-face with the patient), with psychotherapy (e.g., insight oriented, behavior modifying or supportive psychotherapy); 45 minutes</td>
</tr>
</tbody>
</table>

*CPT® is a registered trademark of the American Medical Association*

**REFERENCES**


<table>
<thead>
<tr>
<th>Date</th>
<th>Action/Description</th>
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<tr>
<td>12/16/2016</td>
<td>Version 1 (Approved by UMC)</td>
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<tr>
<td>1/10/2018</td>
<td>Annual Update: Updates to formatting, references, coding.</td>
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