Kansas City

An Independent Licensee of the Blue Cross and Blue Shield Association

Neurofeedback

Policy Number: 2.01.28
Origination: 7/2008
Last Review: 9/2014
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Policy

Blue Cross and Blue Shield of Kansas City (Blue KC) will not provide coverage for neurofeedback. This is considered investigational.

When Policy Topic is covered

Not Applicable

When Policy Topic is not covered

Neurofeedback is considered **investigational**.

Description of Procedure or Service

Neurofeedback describes techniques of providing feedback about neuronal activity, as measured by electroencephalogram (EEG) biofeedback or functional magnetic resonance imaging (fMRI), in order to teach patients to self-regulate brain activity. Neurofeedback may utilize several techniques in an attempt to normalize unusual patterns of brain function in patients with central nervous system (CNS) disorders, such as attention deficit/hyperactivity disorder (ADHD), autism spectrum disorder, substance abuse, epilepsy, and insomnia.

Background

Neurofeedback may be conceptualized as a type of biofeedback that has traditionally used the electroencephalogram (EEG) as a source of feedback data. Neurofeedback differs from traditional forms of biofeedback in that the information fed back to the patient (via EEG tracings or fMRI) is a direct measure of global neuronal activity, or brain state, compared to feedback of the centrally regulated physiologic processes, such as tension of specific muscle groups or skin temperature. The patient may be trained to either increase or decrease the prevalence, amplitude, or frequency of specified EEG waveforms (e.g., alpha, beta, theta waves), depending on the changes in brain function associated with the particular disorder. It has been proposed that training of slow cortical potentials (SCPs) can regulate cortical excitability and that using the EEG as a measure of CNS functioning can help train patients to modify or control their abnormal brain activity. Upregulating or downregulating neural activity with real-time feedback of fMRI signals is also being explored.

Neurofeedback is being investigated for the treatment of a variety of disorders including attention deficit/hyperactivity disorder (ADHD), learning disabilities, Tourette syndrome, autism spectrum disorder traumatic brain injury, seizure disorders, menopausal hot flashes, panic and anxiety disorders, fibromyalgia, tinnitus, substance abuse disorders, depression, stress management, migraine headaches, Parkinson's disease and sleep disorders. Two EEG training protocols, training of SCPs and theta/beta training, are typically used in children with ADHD. For training of SCPs, surface-negative SCPs and surface-positive SCPs are generated over the sensorimotor cortex. Negative SCPs reflect increased excitation and occur during states of behavioral or cognitive preparation, while positive SCPs are thought to indicate reduction of cortical excitation of the underlying neural networks and appear during behavioral inhibition. In theta/beta training, the goal is to decrease activity in the EEG theta band (4-8 hertz [Hz]) and increase activity in the EEG beta band (13-20 Hz), corresponding to an alert and

focused but relaxed state. Alpha-theta neurofeedback is typically used in studies on substance abuse. Neurofeedback protocols for depression focus on alpha interhemispheric asymmetry and theta/beta ratio within the left prefrontal cortex. Neurofeedback for epilepsy has focused on sensorimotor rhythm up-training (increasing 12-15 Hz activity at motor strip) or altering SCPs. It has been proposed that learned alterations in EEG patterns in epilepsy are a result of operant conditioning and are not conscious or voluntary. A variety of protocols have been described for treatment of migraine headaches.

Rationale

This policy was originally based on a 1997 TEC Assessment and updated periodically using the MEDLINE database. The most recent literature update was performed through June 2, 2014.

The 1997 TEC Assessment concluded that there were inadequate data to permit conclusions regarding the health outcome effects of neurofeedback for any indication. (1) Among the 19 studies reviewed in the TEC Assessment, few were randomized controlled trials (RCTs), and those that were did not support the efficacy of neurofeedback in improving health outcomes. Literature published since the 1997 TEC Assessment consists of studies that evaluate neurofeedback for a variety of clinical indications, with the greatest amount of scientific literature published on the treatment of attention deficit/hyperactivity disorder (ADHD). Relevant systematic reviews and key randomized or controlled trials of neurofeedback are described here.

ADHD Systematic Reviews. A 2005 review/meta-analysis used criteria from the Association for Applied Psychophysiology and Biofeedback and the International Society for Neuronal Regulation to assess the clinical efficacy of neurofeedback for ADHD. (2) The authors concluded that neurofeedback for ADHD was ranked at level 3 or "probably efficacious" on a scale of 1 to 5 (1 being not empirically supported and 5 being efficacious and specific). The authors noted that benefits were reported in the 5 randomized group studies (totaling 214 patients) included in their analysis; however, the ranking for neurofeedback for ADHD was based on the need for further studies controlled for patient and therapist factors that could unduly influence outcomes.

In 2009, Arns et al published a meta-analysis on neurofeedback and ADHD, concluding that neurofeedback could be considered “efficacious and specific” for ADHD based on level 5 evidence. (3) Fifteen studies met criteria (either between-subject or within-subject design) and were included in the analysis. Initial analysis indicated heterogeneity in study results, which typically would preclude meta-analysis. For this paper, studies were removed from the analysis until heterogeneity was achieved. The adjusted analysis indicated similar effect sizes between neurofeedback and stimulant medication; however, this result was based on nonrandomized studies in which patients chose their treatment; this study design has a high potential for selection bias. (4) Four RCTs that either used a wait-list control or active control group were included in the meta-analysis. One of the studies is a German language report and another is an unpublished PhD thesis (total of 69 children); these have not been reviewed in this policy. The other 2 RCTs included in the systematic review are described next, including 20 and 94 children with ADHD, respectively. (5, 6) Overall, the literature included in this meta-analysis is characterized by small, poor quality studies with high potential for bias. The findings of the meta-analysis are also limited by significant heterogeneity in study results and exclusion of studies due to heterogeneity. Details of the English language RCTs are described next.

A 2011 review of complementary medicine for ADHD indicates that there is only 1 large RCT (Gevensleben et al, reviewed next) that found a significant benefit (ie, with a moderate effect size of 0.6) of neurofeedback for children with ADHD. (7) In comparison, effect sizes in studies that used medication were around 0.8 for methylphenidate and around 1.2 for amphetamine. Three additional small RCTs have been identified which found no significant difference between neurofeedback and either attention skills training, placebo training, or biofeedback relaxation training. (8) Comparison with biofeedback relaxation training suggests that nonspecific factors such as a structured learning environment may contribute to the effects of neurofeedback. (9)

RCTs. In 2013, van Dongen-Boomsma et al reported a well-conducted, randomized, assessor and patient-blinded, placebo-controlled trial of neurofeedback in 41 children age 8 to 15 years. (10) Only children with an electroencephalogram (EEG) that deviated from the normative database were included in the study. Children in the neurofeedback group were given positive feedback for increasing activity in the beta band and decreasing theta activity, while children in the placebo-control group received feedback based on a random EEG signal. Outcomes were assessed by structured interview with the parents, teacher reports, and by the investigator with the Clinical Global Impressions-Improvement scale. Both groups improved over time in ADHD symptoms and clinical scores, with similar improvement in the 2 groups.

A randomized study published in 2006 examined brain activity following neurofeedback in 15 children with ADHD. (5) The experimental subjects learned to inhibit the amplitude of theta waves (4–7 Hz) and increase amplitude of beta waves (15–18 Hz). Five children with ADHD were randomly assigned to a no-treatment control condition. Functional magnetic resonance imaging (fMRI) revealed increased activation of the right anterior cingulate cortex, an area related to selective attention that previously was shown to be altered in children with ADHD. However, it could not be determined whether the change in brain function was related to the specific neural training program (decreasing the amplitude of theta waves and increasing the amplitude of beta waves) or to the additional attentional training received by the experimental group. A 2007 report from Europe compared neurofeedback training of slow cortical potentials (SCPs) (n=17) with a control group (n=13) that participated in a group cognitive/behavior training program. (11) The report stated that randomization was incomplete because the age range in the group program was limited, parents had to be available for intense training during neurofeedback, and some parents had a preference for one type of training. Results showed that children in the neurofeedback group improved more than children who had participated in a group therapy program, particularly improved for attention and cognition. However, parental support was found to account for more of the improvement than neurofeedback training performance

To control for nonspecific effects (attention training) and confounding variables (parental engagement), Gevensleben et al compared neurofeedback with a control intervention of participation in a computerized attention skills training. (6) All children were drug-naïve or drug-free without concurring psychotherapy for at least 6 weeks before starting training. The 2 training conditions were designed to be as similar as possible, using computer games, positive reinforcement by a trainer, homework, and parental encouragement in using the skills/strategies learned during training in real-life situations. Both groups participated in 2 blocks of 9 sessions (approximately 100 minutes per session plus a break), with 2 to 3 sessions per week, and parents were informed that both treatments were expected to be beneficial but were not informed as to which type of training their child had been assigned. A total of 102 children were randomly assigned in a 3:2 ratio; 8 children were excluded due to need for medical treatment or noncompliance with the study protocol by either the children or their parents, resulting in 59 children in neurofeedback and 35 in attention training (92% follow-up). SCPs and theta/beta training were compared by starting with one type of training in the first block and then the other (counterbalanced order) in the second block. Investigator evaluations were performed by the teachers, and thus, the teachers were not blinded to the treatment. At the end of training/testing, there were no significant differences in parents' attitude toward the 2 training conditions or in the perceived motivation of their children. Approximately 40% of the parents either did not know which training their child had participated in or guessed the wrong group. Both parents and teachers rated the neurofeedback group as more improved on the hyperactivity subcomponent of a Strength and Disabilities Questionnaire (eg, SDQ, 19% vs 3%, respectively, improved) and on a German ADHD scale (eg, 26% vs 9%, respectively, improved). Thirty children in the neurofeedback group (52%) and 10 children in the attention training group (29%) improved more than 25% in the German ADHD scale (odds ratio=2.68), which was the primary outcome measure. Other components of the SDQ, including emotional symptoms; conduct problems; peer problems; and prosocial behavior, were not different between the 2 training conditions. No significant differences were noted between the 2 neurofeedback training protocols. Results of this RCT suggested that neurofeedback may have specific effects on attention and hyperactivity beyond those achieved by attention training and parental involvement. The authors noted that future studies

should further address the specificity of effects and how to optimize the benefit of neurofeedback as a treatment module for ADHD.

Six-month follow-up from the RCT previously described was reported in 2010. (6, 12) Of the 94 children who completed treatment, 17 started medication during the follow-up interval, and parents of 16 children did not return the questionnaires. Follow-up was obtained in 61 children (65%) of the original per-protocol 102 children. Although the percentage of dropouts did not differ between the 2 groups, dropouts tended to have higher scores on the German ADHD rating scale (FBB-HKS), particularly in the control group. The difference in dropouts between the groups limits the interpretation of the comparative data, as the scores in the 2 groups included in follow-up were not similar at baseline (eg, baseline FBB-HKS of 1.50 for the neurofeedback group and 1.37 for the control group). The improvement observed in the neurofeedback group after treatment appeared to be preserved at 6-month follow-up. For example, the inattention subscore of the FBB-HKS improved from 2.02 to 1.51 after treatment and remained at 1.49 at 6-month follow-up (moderate effect size of 0.73). The hyperactivity/impulsivity subscore improved from 1.10 to 0.79 after treatment and remained at 0.76 at 6-month follow-up (small effect size of 0.35). The authors of this European study noted that the treatment effects appear to be limited but considered neurofeedback to be potentially effective as a component of a multimodal treatment approach.

Steiner et al randomized 104 children with ADHD age 7 to 11 years to receive neurofeedback, cognitive training, or a no-intervention control condition in their elementary school. (13) Both the neurofeedback and cognitive therapies were administered with commercially available computer programs (45-min sessions 3 times per week), monitored by a trained research assistant. The neurofeedback EEG sensor was embedded in a standard bicycle helmet with the grounding and reference sensors located on the chin straps on the mastoids. No data was presented on the technical performance of this system. There were some differences in baseline measures between the groups, although these differences were not large. The slope of the change in scores over time was compared between groups. Children in the neurofeedback group showed a small improvement on the Conners 3-Parent Assessment Report (effect size [ES]=0.34 for inattention, ES=0.25 for executive functioning, ES=0.23 for hyperactivity/impulsivity), and subscales of the Behavior Rating Inventory of Executive Function Parent Form (BRIEF global executive composite, ES=0.23) when compared with baseline. Interpretation of these findings is limited by the use of a no-intervention control group and lack of parental blinding. Evaluator-blinded classroom observation (Behavioral Observation of Students in Schools) found no sustained change with a linear growth model but a significant improvement with a quadratic model. No between-group difference in change in medication was observed at the 6-month follow-up.

In 2012, Duric et al reported a comparative study of neurofeedback versus methylphenidate in 91 children with ADHD. (14) The children were randomized into 3 groups, consisting of 30 sessions of neurofeedback, methylphenidate, or a combination of neurofeedback and methylphenidate. The neurofeedback sessions focused on increasing cortical beta activity and decreasing theta activity. Parental evaluations found improvements in ADHD core symptoms for all 3 groups, with no significant differences between groups. Alternative reasons for improvement with neurofeedback include the amount of time spent with the therapist and cognitive-behavioral training introduced under neurofeedback.

Section Summary

There are 2 moderate-sized RCTs that have examined neurofeedback in comparison with attention skills training or cognitive therapy. Both studies found a small benefit of neurofeedback, although interpretation of 1 of the studies is limited by the lack of parental blinding and differences in baseline measures, along with a lack of information on the technical performance of the system. A smaller well-conducted, sham-controlled study found no benefit of neurofeedback on standard ADHD outcome measure. Studies that have attempted to use active controls have suggested that at least part of the effect of neurofeedback may be due to attention skills training, relaxation training, and/or other nonspecific effects. Larger sham-controlled studies are needed to evaluate whether neurofeedback

(alone or in combination with other treatments) has beneficial effects for children with ADHD. Durability of any observed effect also needs to be evaluated.

Autism Spectrum Disorder

In a 2009 systematic review of novel and emerging treatments for autism spectrum disorders, neurofeedback received a grade C recommendation, supported by one nonrandomized controlled trial. (15) The only controlled trial identified was a pilot study from 2002 that included 12 children with autism who received neurofeedback and an untreated control group of 12 children who were matched by sex, age, and disorder severity. (16) The study found a 26% reduction in autism symptoms based on the Autism Treatment Evaluation Checklists (A TEC), compared to 3% for the untreated controls. Parental assessments found improvements in all behavioral categories (socialization, vocalization, anxiety, schoolwork, tantrums, and sleep) in the group treated with neurofeedback, while minimal changes were reported in the control group. As discussed above, there is a need for sham controlled trials with neurofeedback training due to the possibility of nonspecific effects (e.g., attention training) and confounding variables (e.g., parental engagement and expectation). No sham-controlled RCTs on neurofeedback for autism spectrum disorders have been identified.

Cognitive Performance

One small (n=6) quasi-randomized, double-blind pilot study was identified that examined whether increasing peak alpha frequency would improve cognitive performance in older adults (70–78 years of age). (17) Control subjects were trained to increase alpha amplitude or shown playback of one of the experimental subject's sessions. Compared with controls, the experimental group showed improvements in speed of processing for 2 of 3 cognitive tasks (Stroop, Go/No-Go) and executive function in 2 tasks (Go/No-Go, *n*-back); other functional measures, such as memory, were decreased relative to controls.

Depression

Linden et al reported a "proof-of-concept" study of neurofeedback with fMRI in 8 patients with major depressive disorder. (18) Four neurofeedback sessions resulted in the upregulation of brain areas that were shown to be involved in the generation of positive emotions (eg, ventrolateral prefrontal cortex and insula). Testing immediately following the fourth session revealed a significant improvement in clinical symptoms (-4.13 points) on the Hamilton Rating Scale for Depression. A subsequently recruited imagery control group underwent a training procedure with the same cognitive strategies (evoke positive memories) without neurofeedback and showed no clinical improvement (+1.0 point).

Epilepsy

A 2009 meta-analysis by Tan et al identified 63 studies on neurofeedback for treatment of epilepsy. (19) Ten of the 63 studies met inclusion criteria; 9 of these studies included fewer than 10 subjects. The studies were published between 1974 and 2001 and used a prepost design in patients with epilepsy refractory to medical treatment; only 1 controlled study was included. The meta-analysis showed a small effect size for treatment (-0.233), with a likelihood of publication bias based on funnel plot. Placebo-controlled RCTs are needed to evaluate the effect of neurofeedback on seizure frequency in patients with epilepsy.

Fibromyalgia

In 2010, Kayiran et al reported a randomized single blind study of neurofeedback versus escitalopram in 40 patients with fibromyalgia. (20) Patients in the neurofeedback group were instructed to widen a river on a computer monitor which corresponded to increasing sensory motor activity and decreasing theta activity. Patients received 5 sessions per week for 4 weeks. The control group received escitalopram for 8 weeks. Outcome measures at baseline and at weeks 2, 4, 8, 16, and 24 included

visual analog scale for pain, Hamilton and Beck Depression and Anxiety Inventory Scales, Fibromyalgia Impact Questionnaire and Short Form-36. Mean amplitudes of EEG rhythms and the theta/sensory motor rhythm were also measured in the neurofeedback group. At baseline, the control group scored higher on the Hamilton and Beck Anxiety Scales and the Hamilton Depression Scale; all other baseline measures were similar between groups. Both groups showed improvements over time, with significantly better results in the neurofeedback group. There were no changes over time in mean amplitudes of EEG rhythms and essentially no change in the theta/sensory motor rhythm ratio (reduced only at week 4). This study is limited by the difference in intensity of treatment and contact with investigators between the neurofeedback and escitalopram groups. Sham-controlled RCTs are needed when assessing the effect of neurofeedback on subjective outcome measures.

Insomnia

In 2010, Cortoos et al published a small (n=17) RCT on the effect of neurofeedback training or biofeedback training (placebo control) on objective and subjective sleep in patients with primary insomnia. (21) Of 158 subjects with sleep complaints who were interested in participating, 131 (89%) were excluded due to study criteria or unwillingness to remain medication-free during the study period. Following polysomnograph (PSG) recorded sleep in the laboratory, all subjects received 20 sessions of therapist-controlled telefeedback training at home over a period of 8 weeks. The neurofeedback group was trained to increase the sensory-motor rhythm (12-15 Hz) and inhibit theta power (4-8 Hz) and high beta power (20-30 Hz). The biofeedback group was trained to decrease electromyographic (EMG) activity, which was equated with the reinforcement of relaxation (placebo control). Both treatments reduced sleep latency by 40% to 45% (22 minutes at baseline) on post-treatment PSG, measured 2 weeks after the end of training. Neurofeedback training reduced wake after sleep onset (54% vs. 13% decrease, respectively; however, no interaction was found on the 2-way analysis of variance [ANOVA]) and increased total sleep time (40 minutes vs less than 5 minutes, respectively, $p<0.05$). This study is limited by the small number of subjects, differences in sleep parameters at baseline, and short follow-up. Additional studies are needed to evaluate this novel treatment approach.

Obsessive-Compulsive Disorder

In 2013, Koprivova et al reported a double-blind randomized sham-controlled trial of independent component neurofeedback in 20 patients with obsessive-compulsive disorder. (22) Independent component neurofeedback is based on the individual diagnosis of pathologic EEG sources and was directed at downtraining of abnormally high activity. All patients were hospitalized and participated in a 6- week standard treatment program that included cognitive-behavioral therapy and 25 neurofeedback or sham biofeedback sessions. The neurofeedback group showed greater reduction of compulsions compared with the sham group (56% vs 21%). However, clinical improvement was not associated with a change in EEG.

Parkinson Disease

Subramanian et al conducted a “proof of principle” study to determine whether fMRI-guided activity increase in the supplementary motor area (SMA) cortex complex would result in improved motor function in patients with early stage Parkinson disease. (23) Patients were instructed to practice the strategy/imagery that was used during the initial neurofeedback (n=5) or control imagery session (n=5) for 2 to 6 months at home. At follow-up, the patients in the fMRI neurofeedback group showed higher activation than imagery control patients in several brain regions and improved motor speed (finger tapping) and clinical ratings of motor symptoms. The imagery control patients showed no control of SMA activation and no motor improvement.

Substance Abuse

A 2008 systematic review of neurofeedback as a treatment for substance abuse disorders described difficulties in assessing the efficacy of this and other substance abuse treatments, including the lack of clearly established outcome measures, differing effects of the various drugs, presence of comorbid

conditions, absence of a criterion standard treatment, and use as an add-on to other behavioral treatment regimens. (24) The authors concluded that alpha-theta training, when combined with an inpatient rehabilitation program for alcohol dependency or stimulant abuse, would be classified as level 3 or “probably efficacious.” This level is based on beneficial effects shown in multiple observational studies, clinical studies, wait-list control studies, or within-subject or between-subject replication studies. The authors also noted that few large-scale studies of neurofeedback in addictive disorders have been reported, and a shortcoming of the evidence for alpha-theta training is that it has not been shown to be superior to sham treatment.

Tourette Syndrome
A 2011 evidence review with clinical guidelines by the European Society for the Study of Tourette Syndrome identified a total of 2 case studies on neurofeedback for Tourette syndrome; this is considered investigational. (25)

Migraine Headaches
Walker reported quantitative EEG (QEEG) for the treatment of migraine headaches in 46 patients. (26) Results were compared with 25 patients who chose not to do neurofeedback and continued antimigraine drug therapy. Since baseline QEEG assessment (all 71 patients) showed a greater amount of the high-frequency beta band (21-30 Hz); the 5 neurofeedback sessions focused on increasing 10-Hz activity and decreasing 21 to 30 Hz targeted individually to brain areas where high-frequency beta was abnormally increased. Patient diaries of headache frequency showed a reduction in migraines in most patients in the QEEG group but not the drug-therapy group. Fifty-four percent reported complete cessation of migraines over 1 year, with an additional 39% reporting a greater than 50% reduction. In comparison, no patients in the drug-therapy group reported a cessation of headaches, and 8% had a reduction in headache frequency of greater than 50%. Sham-controlled RCTs are needed to adequately evaluate this treatment approach.

Summary

The scientific evidence does not permit conclusions concerning the effect of the technology on health outcomes. The largest body of evidence is for treatment of attention deficit/hyperactivity disorder (ADHD), but the available RCTs in that area are not definitive in demonstrating health outcome benefits.

For patients with insomnia, epilepsy, Tourette syndrome, autism spectrum disorder, fibromyalgia, migraine headache, substance abuse disorder, depression, Parkinson disease, or other neurologic disorders, the evidence is poor and a number of questions regarding clinical efficacy remain to be answered before applying neurofeedback techniques. As a result of the deficiencies in the evidence base, neurofeedback is considered investigational.

Practice Guidelines and Position Statements

The American Academy of Pediatrics published a 2011 clinical practice guideline for the diagnosis, management, evaluation and treatment of ADHD in children and adolescents. (27) They state that although EEGbbiofeedback is used clinically, it is not U.S. Food and Drug Administration (FDA)-approved for the treatment of ADHD and needs further research.

The Institute for Clinical Systems Improvement released a 2012 revision of their 2010 guideline: Diagnosis and Management of Attention Deficit Hyperactivity Disorder in primary care for school age children and adolescents. (28) The guideline states that neurofeedback has been demonstrated in 1 RCT to be significantly better than computerized attention skills training. ADHD symptoms were moderately improved, however, long-term benefits have not been definitely proven, and neurofeedback lacks sufficient research support. They conclude that treatment responses have not reached the level shown with psychostimulant medications; therefore neurofeedback cannot be recommended as an alternative to medication use for ADHD.

The National Institute for Health and Care Excellence (NICE) issued a 2013 clinical guideline; Autism; The management and support of children and young people on the autism spectrum. (29) They stated the following treatments were considered but are not recommended: neurofeedback, auditory integration training to manage speech and language problems, omega-3 fatty acids to manage sleep problems, secretin, chelation, and hyperbaric oxygen therapy in any context.

The International Society for Neurofeedback & Research published a 2011 position paper on standards of practice for neurofeedback and neurotherapy. (30) Issues discussed include competency, qualifications of practitioners, scope of practice, informed consent, pretreatment assessment, standards for remote training, recordkeeping and billing, accountability, standards for practitioner training and qualifications to be trained, adequate supervision and coaching of training sessions, ethical advertising, standards for professional societies, and standards for those who sell and manufacture neurofeedback equipment.

Clinical guidelines on behavioral and psychosocial interventions for Tourette syndrome and other tic disorders were published in 2011 by the European Society for the Study of Tourette Syndrome. The guidelines state that neurofeedback is still experimental. (25)

The American Psychological Association (APA) provides general information on biofeedback (including neurofeedback) on their website www/apaonline.org (APA Online), stating that “Biofeedback helps treat some illness, may boost performance, helps people relax, and is even used to help children with Attention Deficit-Hyperactivity Disorder.” (31)

The Association for Applied Psychophysiology & Biofeedback rates neurofeedback as efficacious (level 4 on a scale of 1–5 with 5 being the best) for ADHD, based on several small controlled and moderately large clinical studies showing that neurofeedback significantly helps children with ADHD who have problems with mathematics. (32)

No information on neurofeedback was identified from the American Academy of Child and Adolescent Psychiatry or the American Psychiatric Association

U.S. Preventative Services Task Force Recommendations

Neurofeedback is not a preventive service.

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Billing Coding/Physician Documentation Information

90875	Individual psychophysiological therapy incorporating biofeedback training by any modality (face-to-face with the patient), with psychotherapy (eg, insight oriented, behavior modifying or supportive psychotherapy); approximately 20-30 minutes
90876	Individual psychophysiological therapy incorporating biofeedback training by any modality (face-to-face with the patient), with psychotherapy (eg, insight oriented, behavior modifying or supportive psychotherapy); approximately 45-50 minutes
90901	Biofeedback training by any modality
E0746	Electromyography (EMG), biofeedback device

Additional Policy Key Words

N/A

Policy Implementation/Update Information

7/1/08	New policy; considered investigational.
7/1/09	No policy statement changes.
7/1/10	No policy statement changes.
7/1/11	No policy statement changes.
7/1/12	No policy statement changes.
7/1/13	No policy statement changes.
7/1/14	No policy statement changes.
9/1/14	No policy statement changes.