WHAT IS NEUROFEEDBACK: AN UPDATE

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Written to educate both professionals and the general public, this article provides an update and overview of the field of neurofeedback (EEG biofeedback). The process of assessment and neurofeedback training is explained. Then, areas in which neurofeedback is being used as a treatment are identified and a survey of research findings is presented. Potential risks, side effects, and adverse reactions are cited and guidelines provided for selecting a legitimately qualified practitioner.

INTRODUCTION
In the late 1960s and 1970s it was learned that it was possible to recondition and retrain brainwave patterns (Kamiya, 2011; Sterman, LoPresti, & Fairchild, 2010). Some of this work began with training to increase alpha brainwave activity for the purpose of increasing relaxation, whereas other work originating at University of California, Los Angeles focused first on animal and then human research on assisting uncontrolled epilepsy. This brainwave training is called EEG biofeedback or neurofeedback. Prior to a more detailed discussion, the author will review some preliminary information about brainwave activity. Brainwaves occur at various frequencies. Some are fast, and some are quite slow. The classic names of these EEG bands are delta, theta, alpha, beta, and gamma. They are measured in cycles per second or hertz (Hz). The following definitions, although lacking in scientific rigor, will provide the general reader with some conception of the activity associated with different frequency bands.

Gamma brainwaves are very fast EEG activity above 30 Hz. Although further research is required on these frequencies, we know that some of this activity is associated with intensely focused attention and in assisting the brain to process and bind together information from different areas of the brain. Beta brainwaves are small, relatively fast brainwaves (above 13–30 Hz) associated with a state of mental, intellectual activity and outwardly focused concentration. This is basically a “bright-eyed, bushy-tailed” state of alertness. Activity in the lower end of this frequency band (e.g., the sensorimotor rhythm, or SMR) is associated with relaxed attentiveness. Alpha brainwaves (8–12 Hz) are slower and larger. They are generally associated with a state of relaxation. Activity in the lower half of this range represents a considerable degree the brain shifting into an idling gear, relaxed and a bit disengaged, waiting to respond when needed. If people merely close their eyes and begin picturing something peaceful, in less than half a minute there begins to be an increase in alpha brainwaves. These brainwaves are especially large in the back third of the head. Theta (4–8 Hz) activity generally represents a more daydream-like, rather spacey state of mind that is associated with mental inefficiency. At very slow levels, theta brainwave activity is a very relaxed state, representing the twilight zone between waking and sleep. Delta brainwaves (0.5–3.5 Hz) are very slow, high-amplitude (magnitude) brainwaves and are what we experience
in deep, restorative sleep. In general, different levels of awareness are associated with dominant brainwave states.

It should be noted, however, that each of us always has some degree of each of these various brainwave frequencies present in different parts of our brain. Delta brainwaves will also occur, for instance, when areas of the brain go “off line” to take up nourishment, and delta is also associated with learning disabilities. If someone is becoming drowsy, there are more delta and slower theta brainwaves creeping in, and if people are somewhat inattentive to external things and their minds are wandering, there is more theta present. If someone is exceptionally anxious and tense, an excessively high frequency of beta brainwaves may be present in different parts of the brain, but in other cases this may be associated with an excess of inefficient alpha activity in frontal areas that are associated with emotional control. Persons with Attention-Deficit/Hyperactivity Disorder (ADD, ADHD), head injuries, stroke, epilepsy, developmental disabilities, and often chronic fatigue syndrome and fibromyalgia tend to have excessive slow waves (usually theta and sometimes excess alpha) present. When an excessive amount of slow waves are present in the executive (frontal) parts of the brain, it becomes difficult to control attention, behavior, and/or emotions. Such persons generally have problems with concentration, memory, controlling their impulses and moods, or hyperactivity. They have problems focusing and exhibit diminished intellectual efficiency.

As the reader can see, there can be complexity involved in how the brain is operating. Research (Hammond, 2010b) has found that there is heterogeneity in the EEG patterns associated with different diagnostic conditions such as ADD/ADHD, anxiety, or obsessive-compulsive disorder. For example, scientific research has identified a minimum of three major subtypes of ADD/ADHD, none of which can be diagnosed from only observing the person’s behavior and each of which requires a different treatment protocol. The picture can become even more complicated by the fact that sometimes there are other comorbid problems present, and not simply ADD/ADHD alone. Therefore, appropriate assessment is important prior to beginning to do neurofeedback to determine what EEG frequencies are excessive or deficient, or if there are problems in processing speed or coherence, and in what parts of the brain. Proper assessment allows the treatment to be individualized and tailored to the patient.

Neurofeedback training is EEG (brainwave) biofeedback. During typical training, one or more electrodes are placed on the scalp and one or two are usually put on the earlobes. Then, high-tech electronic equipment provides real-time, instantaneous feedback (usually auditory and visual) about your brainwave activity. The electrodes allow us to measure the electrical patterns coming from the brain—much like a physician listens to your heart from the surface of your skin. No electrical current is put into your brain. Your brain’s electrical activity is relayed to the computer and recorded. Ordinarily, patients cannot reliably influence their brainwave patterns because they lack awareness of them. However, when they can see their brainwaves on a computer screen a few thousandths of a second after they occur, it gives them the ability to influence and gradually change them. The mechanism of action is generally considered to be operant conditioning. We are literally reconditioning and retraining the brain. At first, the changes are short-lived, but the changes gradually become more enduring. With continuing feedback, coaching, and practice, healthier brainwave patterns can usually be retrained in most people. As is reviewed later in the article, most research suggests that significant improvements seem to occur 75 to 80% of the time. The process is a little like exercising or doing physical therapy with the brain, enhancing cognitive flexibility and control. Thus, whether symptoms stem from ADD/ADHD, a learning disability, a stroke, head injury, deficits following neurosurgery, uncontrolled epilepsy, cognitive dysfunction associated with aging, depression, anxiety, obsessive-compulsive disorder, autism, or other brain-related conditions, neurofeedback training offers additional opportunities.
for rehabilitation through directly retraining the electrical activity patterns in the brain. The exciting thing is that even when a problem is biological in nature, there is now another treatment alternative to simply relying on medication. Neurofeedback is also being used increasingly to facilitate peak performance in “normal” individuals, executives, and athletes.

More than a decade ago, Frank H. Duffy, MD, a professor and pediatric neurologist at Harvard Medical School, stated in the journal Clinical Electroencephalography that scholarly literature had already suggested that neurofeedback “should play a major therapeutic role in many difficult areas. In my opinion, if any medication had demonstrated such a wide spectrum of efficacy it would be universally accepted and widely used” (Duffy, 2000, p. v). “It is a field to be taken seriously by all” (p. vii). Considerable research has been published since that time. This article, written to educate both professionals and the general public about the field of neurofeedback, provides an overview of this literature without seeking to cite every publication with all their methodological details.

ASSESSMENT PRIOR TO NEUROFEEDBACK TRAINING

Some people wish that they could simply buy their own neurofeedback equipment and train themselves or their children. As is explained later in this article, this is fraught with potential for harm or ineffectiveness. To be done properly, neurofeedback needs to be conducted or supervised by someone with specialized expertise concerning brain function and who is knowledgeable about much more than simply how to operate equipment and software. As just mentioned, for training to be successful and side effects avoided, it is vitally important for an assessment to be performed and the training to be individualized to the distinctive brainwave patterns and symptoms of each person. Everyone does not need the same training at the same locations, and research has shown that a person’s brainwave patterns simply cannot be distinguished by only observing the person’s behavioral symptoms. Therefore, prior to doing neurofeedback training, legitimate licensed clinicians will want to ask questions about the clinical history of the client or patient. Occasionally in more serious cases they may suggest doing neuropsychological or psychological testing. Competent clinicians (Hammond et al., 2011) will also do a careful assessment and examine brainwave patterns. Some practitioners may do an assessment by placing one or two electrodes on the scalp and measuring brainwave patterns in a limited number of areas. Other clinicians perform a more comprehensive evaluation by doing a quantitative electroencephalogram (QEEG) brain map where 19 or more electrodes are placed on the scalp.

A QEEG is an assessment tool to objectively and scientifically evaluate a person’s brainwave function. The procedure usually takes about 60 to 75 min and consists of placing a snug cap on the head, which contains small electrodes to measure the electrical activity coming from the brain. This is done while the client is resting quietly with his or her eyes closed, eyes open, and sometimes during a task. Afterward, a careful process is used to remove as completely as possible artifacts that occurred when the eyes moved or blinked, from body movement, or tension in the jaw, neck, or forehead. The brainwave data that were gathered are then statistically compared to a sophisticated and large normative database that provides scientifically objective information on how the brain should be functioning at the client’s age. This assessment procedure allows the professional to then determine in a scientific, objective manner whether a client’s brainwave patterns are significantly different from normal, and if so, how and where they differ.

Since the 1970s and 1980s there has been a great deal of research with QEEG for a wide range of problems. Abundant evidence, summarized in Thatcher (2010), has verified the reliability of QEEG evaluation, and hundreds of scientific studies have been published using QEEG evaluations. These studies have found the QEEG to have documented ability to aid in the evaluation of conditions such as mild traumatic brain injury (TBI; and sports-related
concussions), ADD/ADHD, learning disabilities, depression, obsessive-compulsive disorder, anxiety, panic disorder, drug abuse, autism, and a variety of other conditions (including schizophrenia, stroke, epilepsy, and dementia; e.g., Alper, Prichep, Kowalik, Rosenthal, & John, 1998; Amen et al., 2011; Barry, Clarke, Johnstone, McCarthy, & Selikowitz, 2009; Clarke, Barry, McCarthy, & Selikowitz, 2001; Clarke et al., 2007; Harris et al., 2001; Hoffman et al., 1999; Hughes & John, 1999; Newton et al., 2004; Thatcher, 2010; Thatcher et al., 1999). QEEG has even been able to predict treatment outcomes from interventions with conditions such as ADD/ADHD (Suffin & Emory, 1995), and alcoholism and drug abuse (Bauer, 1993, 2001; Prichep, Alper, Kowalik, John, et al., 1996; Prichep, Alper, Kowalik, & Rosenthal, 1996; Winterer et al., 1998). The American Psychological Association has also endorsed QEEG as being within the scope of practice of psychologists who are appropriately trained, and the International Society for Neurofeedback and Research (ISNR) has similarly endorsed its use by qualified health care professionals who are appropriately trained (Hammond et al., 2004) and created standards for the use of QEEG in neurofeedback. Persons who are certified in this assessment specialty may be identified through either the EEG & Clinical Neuroscience Society (http://www.ecnsweb.com/provider-directory.html) or the Quantitative Electroencephalography Certification Board (http://www.qeegboard.org).

**NEUROFEEDBACK TRAINING**

Once the assessment is complete and treatment goals have been established, most commonly one or more electrodes are placed on the scalp and one or more on the earlobes for neurofeedback training sessions. The trainee then usually watches a display on the computer screen and listens to audio tones, sometimes while doing a task such as reading. These training sessions are designed assist the person to gradually change and retrain their brainwave patterns. For example, some persons may need to learn to increase the speed or size of brainwaves in specific areas of the brain, whereas other individuals need training to decrease the speed of and amplitude of their brainwaves. Commonly initial improvements begin to be noticed within the first five to 10 sessions. Length of treatment may only be 15 to 20 sessions for anxiety or insomnia, but with other conditions such as ADD/ADHD or learning disabilities it will more often involve 30 to 50 sessions, depending on the severity of the problem. Each session usually lasts about 20 to 25 min once equipment is attached. In treating very complex conditions or when multiple disorders or diagnoses are present, a clinician cannot always stipulate in advance how many treatment sessions may be required.

**SPECIALIZED TYPES OF NEUROFEEDBACK**

There are also several innovative forms of neurofeedback that should be explained. They each differ in distinctive ways from the traditional neurofeedback methods that have just been described, and yet each represents important and fascinating advances in our technology.

**Slow Cortical Potentials Training**

Speaking very technically for a moment, slow cortical potentials are the positive or negative polarizations of the EEG in the very slow frequency range from .3 Hz to usually about 1.5 Hz. They may be thought of as the direct current baseline on which the alternating current EEG activity rides. There is generally a negative shift in direct current potentials that occurs during cognitive processing (to create excitatory effects) and positive slow cortical potentials occur during inhibition of cortical networks. During and prior to an epileptic seizure, for example, the cortex is electronegative, and this same kind of hyperexcitability tends to be seen before many migraines. After a seizure, when the cortex is fatigued, it tends to be electro-positive. Slow cortical potential neurofeedback training has been done (e.g., Kotchoubey, Blankenhorn, Froscher, Strehl, & Birbaumer, 1997; Kotchoubey et al., 2001; Strehl et al., 2006), particularly in Europe, with
epilepsy and ADHD. This type of neurofeedback may also hold strong potential in the treatment of migraine (Kropp, Siniatchkin, & Gerber, 2002). In this training, one electrode is placed in the center of the top of the head and one behind each ear, while the client focuses on changing a visual display on the computer (Strehl, 2009).

THE LOW ENERGY NEUROFEEDBACK SYSTEM

The Low Energy Neurofeedback System (LENS; Hammond, 2007b; Larsen, 2006; Ochs, 2006) is a unique and passive form of neurofeedback that produces its effects through feedback that involves a very tiny electromagnetic field, which only has a field strength of $10^{-18}$ watts/cm$^2$. This feedback is so small that it is the equivalent of only $\frac{1}{400}$th of the strength of the input we receive from simply holding an ordinary cell phone to the ear and only about the intensity of the output coming from a watch battery. It is delivered in 1-s intervals down electrode wires while the patient remains relatively motionless, usually eyes closed. This feedback is adjusted 16 times a second to remain a certain number of cycles per second faster than the dominant brainwave frequency. Most preliminary research and clinical experience are encouraging with articles published on LENS treatment of conditions such as TBI (Hammond, 2010c; Schoenberger, Shiflett, Esdy, Ochs, & Matheis, 2001), fibromyalgia (C. C. S. Donaldson, Sella, & Mueller, 1998; Mueller, Donaldson, Nelson, & Layman, 2001), anger (Hammond, 2010a), restless legs syndrome (Hammond, in press), ADD/ADHD, anxiety, depression, insomnia and other conditions (Larsen, 2006; Larsen, Harrington, & Hicks, 2006). LENS has even been used to modify behavioral problems in animals (Larsen, Larsen, et al., 2006). Advantages of the LENS approach include that it commonly seems to produce results faster than traditional neurofeedback, and it can be used with very young children and with individuals who are less motivated and who do not have the impulse control or stamina required with other neurofeedback approaches.

Hemoencephalography

There are two different hemoencephalography (HEG) systems that provide feedback, which is believed to influence cerebral blood flow (Toomim & Carmen, 2009). Preliminary research consisting of case series reports on the HEG applications appears encouraging (Carmen, 2004; Coben & Pudolsky, 2007b; Duschek, Schuepbach, Doll, Werner, & Reyes Del Paso, 2010; Friedes & Aberbach, 2003; Mize, 2004; Sherrill, 2004; Toomim et al., 2004), perhaps especially with migraine.

Live Z-Score Neurofeedback Training

Live Z-score training is a more recent innovation that usually utilizes two, four, or more electrodes on the head. Continuous calculations are being computed comparing the way that the brain is functioning on different variables (e.g., power, asymmetries, phase-lag, coherence) to a scientifically developed normative database. Feedback is then based on these moment-to-moment statistical comparisons to norms for the patient’s approximate age group. As with other methods of neurofeedback, the feedback that is provided is designed to guide the brain toward normalized function. This feedback often consists of observing a DVD where the picture dims and flickers when the person is not doing as well and becomes more clear and bright when his or her brain is functioning closer to norms. At this point, most of what has been published on this approach are case series data (Collura, 2008a, 2008b, 2009; Collura, Juan, Tarrant, Bailey, & Starr, 2010; Collura, Thatcher, Smith, Lambos, & Stark, 2009), with the exception of a new controlled study showing positive results with insomnia (Hammer, Colbert, Brown, & Illoï, 2011), but these preliminary results, which include pre- and posttreatment QEEGs, are very encouraging. As this is being written, an expansion of this approach has become available wherein an entire electrode cap with 19 electrodes can also be used for training.

LORETA Neurofeedback Training

LORETA refers to low resolution electromagnetic tomography. This is a kind of QEEG
analysis that provides an estimation of the location of the underlying brain generators (e.g., the anterior cingulate, insula, fusiform gyrus) of the patient’s EEG activity within a frequency band. Very preliminary research (Cannon & Lubar, 2007; Cannon et al., 2007; Cannon et al., 2006; Congedo, Lubar, & Joffe, 2004) has been published about this approach. It does require more labor-intensive preparation where an entire electrode cap with 19 electrodes must be applied in every session. It is believed that this approach may have potential to improve outcomes in difficult cases and/or shorten the length of treatment, and a preliminary report (Cannon & Lubar, 2011) suggests that changes may be enduring.

Functional MRI Neurofeedback

Functional magnetic resonance imaging (fMRI) is a very sophisticated type of neuroimaging that examines brain activation to evaluate brain functioning (unlike the MRI, which examines brain structure). A fascinating scientific advancement in the last several years has been utilization of the fMRI for neurofeedback (Caria et al., 2007; deCharms, 2007; deCharms et al., 2004; deCharms et al., 2005; Haller, Birbaumer, & Veit, 2010; Johnston, Boehm, Healy, Goebel, & Linden, 2010; Rota et al., 2009; Weiskopf et al., 2004; Weiskopf et al., 2003; Yoo et al., 2006). An advantage of fMRI neurofeedback is that it can examine functioning at deep subcortical areas of the brain. However, the serious practical disadvantage of fMRI neurofeedback is that it would be incredibly expensive and with equipment that costs approximately $1 million or more, as well as extreme expenses associated with the day-to-day operation of such equipment, this approach does not appear to be something that will hold realistic clinical promise as a treatment option in the foreseeable future.

AREAS OF APPLICATION FOR NEUROFEEDBACK TREATMENT ADD/ADHD

Since the late 1970s, neurofeedback has been researched, refined, and tested with ADD/ADHD and learning disabilities. Clinical work by Dr. Joel Lubar and his colleagues (e.g., Lubar, 1995) at the University of Tennessee as well as many others has repeatedly demonstrated that it is possible to retrain the brain. In fact, one randomized controlled study (Levesque, Beauregard, & Mensour, 2006) documented with fMRI neuroimaging the positive changes in brain function in ADHD children that mirrored their behavioral changes following neurofeedback treatment. This and the research cited next all provide strong support that demonstrate the effectiveness of neurofeedback in treating ADD/ADHD. Whereas the average stimulant medication treatment study follow-up is only 3 weeks long, with only four long-term follow-up medication studies that lasted 14 months or longer, Lubar (1995) published 10-year follow-ups on cases and found that in about 80% of clients, neurofeedback can substantially improve the symptoms of ADD and ADHD and that these changes are maintained.

Rossiter and LaVaque (1995) found that 20 sessions of neurofeedback produced comparable improvements in attention and concentration to taking Ritalin. Fuchs, Birbaumer, Lutzenberger, Gruzelier, and Kaiser (2003) and Rossiter (2005) likewise demonstrated that neurofeedback produced comparable improvements to Ritalin. Drechsler et al. (2007) found slow cortical potentials training superior to group therapy with ADHD children. Neurofeedback has also been found in randomized controlled studies to be superior to EMG biofeedback (Bakhshayesh, 2007). In a 1-year follow-up, control group study, Monastra, Monastra, and George (2002) found that neurofeedback produced superior improvements compared to Ritalin, not requiring continuation of the medication. In a randomized controlled study, Leins et al. (2007) demonstrated that 30 sessions of slow cortical potentials training or of traditional neurofeedback were both effective in producing cognitive, attentional, behavioral, and IQ improvements, which remained stable 6 months after treatment.

Gevensleben et al. (2009b) in a randomized controlled study documented the superiority of neurofeedback training (effect size = .60)
compared with computerized attention skills training (which would have placebo control characteristics). Behavioral and attentional improvements were found to be stable on 6-month follow-up in research studies reported by Strehl et al. (2006) and Gevensleben et al. (2010), and the latter found that neurofeedback training produced superior results to computerized attention skills training, as did Holtmann et al. (2009).

Two randomized, double-blind placebo controlled studies (deBeus & Kaiser, 2011; deNiet, 2011) have documented the effectiveness of neurofeedback with ADHD. Other recent, large randomized controlled studies (Gevensleben et al., 2009a; Wrangler et al., 2010) should also do much to dispel concerns that improvements from neurofeedback training simply reflect nonspecific placebo factors. These studies demonstrated protocol-specific changes in electrophysiological brain function using EEG and sophisticated event-related potential measures, replicating some earlier findings (Heinrich, Gevensleben, Freisleder, Moll, & Rothenberger, 2004) and showing distinct neuronal mechanisms involved with different training techniques. A 2-year follow-up (Gani, Birbaumer, & Strehl, 2008) of the Heinrich research found that not only were improvements in attention and behavior stable but that some parent ratings had shown continued improvement during the 2 years. Continuing improvement on 6-week and 12-week follow-ups were also found after the completion of LENS treatment of adult ADD/ADHD by deNiet (2011) in a randomized, double-blind placebo controlled study. Thus follow-up evaluations ranging from 3 months to 10 years after treatment (Gani et al., 2008; Heinrich et al., 2004; Lubar, 1995; Monastra et al., 2002; Strehl et al., 2006) provide strong support that improvements from neurofeedback with ADD/ADHD should be enduring, unless of course something such as a head injury or drug abuse were to occur to negative alter brain function.

A recent meta-analysis (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009) concluded that neurofeedback treatment of ADD/ADHD meets criteria for being classified as an efficacious and specific treatment—the highest level of scientific validation (La Vaque et al., 2002). In comparison to neurofeedback, a meta-analysis (Schachter, Pham, King, Langford, & Hoher, 2001) of randomized controlled studies of medication treatment for ADD/ADHD concluded that the studies were of poor quality, had a strong publication bias (meaning that drug company funded studies that failed to support the effectiveness of their product tended to never be submitted for publication), and often produced side effects. They further indicated that long-term effects (beyond placebo effects) for longer than a 4-week follow-up period were not demonstrated.

A recent comprehensive review (Drug Effectiveness Review Project, 2005) of medication treatment for ADD/ADHD concluded that there was no evidence on the long-term safety of the medications used in ADD/ADHD treatment and that good quality evidence is lacking that drug treatment improves academic performance or risky behaviors on a long-term basis, or in adolescents or adults. The latter conclusions were also reached by Joughin and Zwi (1999). The largest randomized controlled multisite study compared medication treatment, “routine community care,” and behavior therapy. Outcome raters were not blinded, introducing a bias, and most subjects in community care were also on medications. At 14-month follow-up (MTA Cooperative Group, 1999), all groups showed improvements, and medication produced better improvements in attention and hyperactivity (the latter only on parent ratings), but not in aggression, social skills, grades, or parent–child relations. The ratings provided by the only blinded rater (a classroom observer), however, showed no difference between groups, and on 3-year follow-up (Swanson et al., 2007) there was no difference on any outcome measures between groups, findings that were confirmed on 8 year follow-up (Molina et al., 2009). Studies (e.g., Swanson et al., 2007) have confirmed loss of appetite and growth suppression as a side effect of medication treatment, along with other side effects such as increased heart rate and blood pressure, insomnia, loss of emotional responsiveness,
dizziness, headache, and stomachache. In the MTA study, 64% of children reported side effects, 11% of them moderately severe and 3% severe. Side effects associated with ADD/ADHD medications are also so common that less than 50% of children maintain prescribed dosages for more than 6 months (Hoagwood, Jensen, Feil, Vitiello, & Blatara, 2000).

In light of these findings, neurofeedback seems well validated as providing a noninvasive and relatively side effect free treatment alternative for ADD/ADHD. In the long run it is also very cost effective. Some individuals express concern about the cost of neurofeedback being greater than the expense involved in drug treatment. Research has shown, however, that the costs associated with medication treatment are actually quite sizable. For instance, a study (Marchetti et al., 2001) of six different medications for ADD/ADHD treatment found that the average cost per school-aged patient was $1,678 each year. Another study (Swensen et al., 2003) examined the health care costs in more than 100,000 families where ADHD was either present or not present. They found that in families where a member had ADHD, the direct costs of health care expenditures plus indirect costs (such as work loss) averaged $1,288 per year higher for the other family members (who had not been diagnosed as having ADD/ADHD) in comparison with members of families where ADHD was not present. This would mean that the cost of medication just cited, combined with indirect costs each year for a family with two children, one of whom had ADHD, would be $5,542.

Neurofeedback training for ADD/ADHD is commonly found to be associated with decreased impulsiveness/hyperactivity, increased mood stability, improved sleep patterns, increased attention span and concentration, improved academic performance, and increased retention and memory, and with a much lower rate of side effects. It is fascinating to note that ADD/ADHD or learning disability studies that have evaluated IQ pre- and posttreatment have commonly found IQ increases following neurofeedback training. These improvements ranged from an average of 9 IQ points improvement in one study (Linden, Habib, & Radojevic, 1996), to an average improvement of 12 IQ points in a study by L. Thompson and Thompson (1998), a mean of 19 IQ points in another study (Tansey, 1991b), and even up to an average increase of 23 IQ points in a study by Othmer, Othmer, and Kaiser (1999).

Learning and Developmental Disabilities

With regard to learning disabilities, Fernandez et al. (2003) demonstrated in a placebo-controlled study that neurofeedback was an effective treatment, and the improvements were sustained on 2-year follow-up (Becerra et al., 2006). An additional report by Fernandez (Fernandez et al., 2007) on 16 children with learning disabilities documented significant EEG changes 2 months after neurofeedback compared to a placebo-control group where there were no EEG changes, and 10 of 11 children in the neurofeedback treatment group showed objective changes in academic performance compared with one in five children in the placebo group. Other articles have also been published on the value of neurofeedback with learning disabilities (Orlando & Rivera, 2004; Tansey, 1991a; Thornton & Carmody, 2005). A randomized controlled study with children with dyslexia (Breteler, Arns, Peters, Giepmans, & Verhoeven, 2010) documented significant improvement in spelling, and Walker (2010a; Walker & Norman, 2006) found significant improvements in reading ability in 41 dyslexia cases. In the first 12 cases reported by Walker (Walker & Norman, 2006) after 30 to 35 sessions, all the children had improved at least two grade levels in reading ability. Barnea, Rassis, and Zaidel (2005) identified improvements in reading ability in learning disability children after 20 sessions.

Although controlled research has not been done, Surlmeli and Ertem (2007) evaluated whether QEEG-guided neurofeedback could be helpful with Down Syndrome children. All eight children who completed up to 60 treatment sessions (one child dropped out after only eight sessions) showed significant improvement...
in attention, concentration, impulsivity, behavior problems, speech and vocabulary, and on QEEG measures. Surmeli and Ertem (2010) treated 23 children diagnosed with mild to moderate mental retardation with 80 to 160 QEEG-guided neurofeedback sessions. Twenty-two of 23 showed clinical improvement on the Developmental Behaviour Checklist, and 19 of 23 showed improvement on the Wechsler Intelligence Scale for Children and a computerized test of attention.

Cognitive and Memory Enhancement

Neurofeedback also has documented results for cognitive and memory enhancement in normal individuals (Angelakis et al., 2007; Boulay, Sarnacki, Wolpaw, & McFarland, 2011; Egner & Gruzelier, 2003; Egner, Strawson, & Gruzelier, 2002; Fritson, Wadkins, Gerdes, & Hof, 2007; Gruzelier, Egner, & Vernon, 2006; Hanslmayer, Sauseng, Doppelmayr, Schabus, & Klimesch, 2005; Hoedlmoser et al., 2008; Keizer, Verment, & Hommel, 2010; Rasey, Lubar, McIntyre, Zoffuto & Abbott, 1996; Vernon et al., 2003; Zoefel, Huster, & Herrmann, 2010). Neurofeedback to enhance cognitive functioning and to counter the effects of aging has been referred to as “brain brightening” (Budzynski, 1996). Ros, Munneke, Ruge, Gruzelier, and Rothwell (2010) produced evidence that neurofeedback training with normal persons may enhance neuroplasticity.

Uncontrolled Epilepsy

Medication treatment of epilepsy is successful only in completely controlling seizures in two thirds of patients (Iasemidis, 2003), and the long-term use of many antiseizure medications can have health risks. When medication treatment is not successful, neurosurgery is often recommended, but it has limited success (Witte, Iasemidis, & Litt, 2003). In addition, many epilepsy patients are also women of child-bearing age who wish to have children but fear the effects of medications on the fetus. Therefore, a treatment option other than or in addition to medication and surgery would be desired. Research has shown that when medication is insufficient to control the occurrence of seizures, neurofeedback can offer an additional modality that can be added to treatment, which has the potential to assist in bringing seizures under control, allowing dosage levels of medications to be reduced, and helping to avoid invasive brain surgery.

Research in this area began in the early 1970s and is extensive and rigorous, including blinded, placebo-controlled, cross-over studies (reviewed in Sterman, 2000, and in a meta-analysis by Tan et al., 2009). The samples in the studies that have been done typically consist of the most severe, out-of-control, medication-treatment-resistant patients. However, even in this most severe group of patients, research found that neurofeedback training on average produces a 70% reduction in seizures. In these harsh cases of medically intractable epilepsy, neurofeedback has been able to facilitate greater control of seizures in 82% of patients, often reducing the level of medication required, which can be very positive given the long-term negative effects of some medications. Many patients, however, may still need to remain on some level of medication following neurofeedback.

More recently Walker and Kozlowski (2005) reported on 10 consecutive cases, and 90% were seizure free after neurofeedback, although only 20% were able to cease taking medication. In another group of 25 uncontrolled epilepsy patients (Walker, 2008), 100% became seizure free following QEEG-guided neurofeedback, with 76% no longer requiring an anticonvulsant for seizure control on follow-up, which averaged 5.1 years. Walker (2010b) reported on still an additional 20 patients with intractable seizures, 18 of which were seizure free following neurofeedback training, whereas two continued to report occasional seizures. Two of the 18 patients remained on a single anticonvulsant medication. The average length of follow-up in these cases was 4 years. In this same report, Walker indicated that he had seen nine women who wished to stop taking anticonvulsants to become pregnant, and all nine have remained seizure free for an average of 6 years.
TBI and Stroke

Concussions and head injuries that cause emotional, cognitive, and behavioral problems occur as a result of many things such as motor vehicle accidents, war (Trudeau et al., 1998), and sports (McCrea, Prichep, Powell, Chabor, & Barr, 2010; McKee et al., 2009), including football (Amen et al., 2011), doing headers in soccer (Tysvaer, Stroll, & Bachen, 1989), and boxing (Ross, Cole, Thompson, & Kim, 1983).

Neurofeedback treatment outcome studies of closed and open head injuries have been published (Ayers, 1987, 1991, 1999; Bounias, Laibow, Bonaly, & Stubbeltine, 2001; Bounais, Laibow, Stubbeltine, Sandground, & Bonaly, 2002; Byers, 1995; Hammond, 2007a, 2007b, 2010c; Hoffman, Stockdale, Hicks, & Schwaninger, 1995; Hoffman, Stockdale, & Van Egren, 1996a, 1996b; Keller, 2001; Laibow, Stubbeltine, Sandground, & Bounais, 2001; Schoenberger et al., 2001; Thornton, 2000; Tinius & Tinius, 2001), as well as with stroke (Ayers, 1981, 1995a, 1995b, 1999; Bearden, Cassisi, & Pineda, 2003; Cannon, Sherlin, & Lyle, 2010; Doppelmayr, Nosko, Pecherstorfer, & Fink, 2007; Putnam, 2001; Rozelle & Budzynski, 1995; Walker, 2007; Wing, 2001), but further high-quality research needs to be done. One article (Hammond, 2007b) reported a case of moderate severity TBI treated with the LENS, which resulted in the complete reversal of posttraumatic anosmia (complete loss of sense of smell) of 9 years’ duration, which was previously unheard of, as well as significant clinical improvement in postconcussion symptoms.

A recent research review (Thornton & Carmody, 2008) particularly suggests that QEEG-guided neurofeedback is superior to neurocognitive rehabilitation strategies and medication treatment in the rehabilitation of TBI. Traditionally physical medicine and rehabilitation physicians tell head injury patients that 1 year after a TBI they cannot expect further improvement and must simply adjust to their deficits. Clinical experience and research thus far clearly indicate that neurofeedback may often produce significant improvements even many years after a head injury. The accumulating evidence indicates that neurofeedback offers a valuable additional treatment in the rehabilitation of head injuries and with athletes who have suffered concussions.

Alcoholism and Substance Abuse

EEG investigations of alcoholics (and the children of alcoholics) have documented that even after prolonged periods of abstinence, they frequently have lower levels of alpha and theta brainwaves and an excess of fast beta activity. This suggests that alcoholics and their children tend to be hardwired differently from other people, making it difficult for them to relax. Following the intake of alcohol, however, the levels of alpha and theta brainwaves increase. Thus individuals with a biological predisposition to develop alcoholism (and their children) are particularly vulnerable to the effects of alcohol because, without realizing it, alcoholics seem to be trying to self-medicate in an effort to treat their own brain pathology. The relaxing mental state that occurs following alcohol use is highly reinforcing to them because of their underlying brain activity pattern. Several research studies now show that the best predictor of relapse is the amount of excessive beta brainwave activity that is present in both alcoholics and cocaine addicts (Bauer, 1993, 2001; Prichep, Alper, Kowalik, John, et al., 1996; Prichep, Alper, Kowalik, & Rosenthal, 1996; Winterer et al., 1998).

Recently, neurofeedback training to teach alcoholics how to achieve stress reduction and profoundly relaxed states through increasing alpha and theta brainwaves and reducing fast beta brainwaves has demonstrated promising potential as an adjunct to alcoholism treatment. Peniston and Kulkosky (1989) used such training in a study with chronic alcoholics compared to a nonalcoholic control group and a control group of alcoholics receiving traditional treatment. Alcoholics receiving 30 sessions of neurofeedback training demonstrated significant increases in the percentages of their EEG that was in the alpha and theta frequencies, and increased alpha rhythm amplitudes. The neurofeedback
treatment group also demonstrated sharp reductions in depression when compared to controls. Alcoholics in standard (traditional) treatment showed a significant elevation in serum beta-endorphin levels (an index of stress and a stimulant of caloric [e.g., ethanol] intake), whereas those with neurofeedback training added to their treatment did not demonstrate this increase in beta-endorphin levels. On 4-year follow-up checks (Peniston & Kulkosky, 1990), only 20% of the traditionally treated group of alcoholics remained sober, compared with 80% of the experimental group who had received neurofeedback training. Furthermore, the experimental group showed improvement in psychological adjustment on 13 scales of the Millon Clinical Multiaxial Inventory compared to the traditionally treated alcoholics who improved on only two scales and became worse on one scale. On the 16-PF personality inventory, the neurofeedback training group demonstrated improvement on seven scales, compared to only one scale among the traditional treatment group. Similar positive results with 92% sobriety on 21-month follow-ups were reported by Saxby and Peniston (1995) in 14 depressed alcoholics, and encouraging results were reported on 3-year follow-ups in a treatment program with native Americans (Kelley, 1997).

Scott, Kaiser, Othmer, and Sideroff (2005) conducted a randomized controlled study with 121 individuals undergoing an inpatient substance abuse program. The patients received 40 to 50 treatment sessions. Persons who had neurofeedback added to their treatment remained in therapy significantly longer—an important factor in the treatment of substance abuse. On 1-year follow-up, 77% of patients receiving neurofeedback remained sober versus only 44% of traditional treatment patients. Significant differences were found in measures of attention and in seven scales on the Minnesota Multiphasic Personality Inventory–2 compared with improvement on only one scale in those receiving traditional treatment. Reports from a similar treatment program (Burkett, Cummins, Dickson, & Skolnick, 2005) with 270 homeless crack cocaine addicts showed that the addition of neurofeedback to treatment more than tripled the length of stay in the recovery center. On 1-year follow-up of the 94 patients who completed treatment, 95.7% were now maintaining a residence, 93.6% were employed or in schooling, 88.3% had no further arrests, and 53.2% had been alcohol and drug free 1 year, whereas another 23.4% had used alcohol or drugs only one to three times, corroborated by urinalysis.

Arani, Rostami, and Nostratabadi (2010) compared results from 30 sessions of neurofeedback being provided to opioid dependent patients undergoing outpatient treatment (methadone or Buprenorpine maintenance), compared with a control group that received outpatient treatment alone. Patients receiving neurofeedback showed significantly more improvements in outcome measures (e.g., of hypochondriasis, obsessing, interpersonal sensitivity, aggression, psychosis, anticipation of positive outcome, and desire to use drugs) and on QEEGs. Preliminary research (Horrell et al., 2010) has suggested that neurofeedback may also have potential to reduce drug cravings in cocaine abusers.

The evidence reviewed validates the immense potential that neurofeedback treatment has to likely double if not triple the outcome rates in alcoholism and substance abuse treatment when it is added as an additional component to a comprehensive treatment program (Sokhadze, Cannon, & Trudeau, 2008). It may have real potential in not only treating but also remediating some of the serious damage to the brain that occurs through drug abuse (e.g., Alper et al., 1998; Prichet, Alper, Kowalik, & Rosenthal, 1996; Struve, Straumanis, & Patrick, 1994).

Antisocial Personality and Criminal Justice

Quirk (1995) reported reduced recidivism using a combination of neurofeedback and galvanic skin response biofeedback. Smith and Sams (2005) showed improvements in attention and behavior in a group of juvenile offenders, and a study in a Boys Totem Town project with seven juvenile felons (Martin & Johnson, 2005) improvements were noted on a variety of measures. Most recently, Surmeli and Ertem
(2009) presented a case series of 13 patients who received from 80 to 100 neurofeedback treatment sessions guided by QEEG findings. Outcomes were measured with the Minnesota Multiphasic Personality Inventory, a test of attention, QEEG results, and interviews with family members. Twelve of the 13 patients showed significant improvement, which was maintained on 2-year follow-up. The abnormal representation of learning disabilities, ADHD, head injuries, childhood abuse, alcoholism, and substance abuse in an incarcerated offender population (Wekerle & Wall, 2002; Wilson & Cumming, 2009) and of alcoholism and drug abuse in domestic violence (Lin et al., 2009) would suggest considerable potential for the use of neurofeedback, particularly given the high recidivism rates that attest to the limited effectiveness of traditional psychotherapies and pharmacology treatment. This will be another fruitful area for further research.

Posttraumatic Stress Disorder

Peniston and Kulkosky (1991) added thirty 30-minute sessions of alpha/theta neurofeedback training to the traditional VA hospital treatment provided to a group of posttraumatic stress disorder Vietnam combat veterans, and then compared them at 30-months posttreatment with a contrast group who received only traditional treatment. On follow-up, all 14 traditional treatment patients had relapsed and been rehospitalized, whereas only three of 15 neurofeedback training patients had relapsed. Although all 14 patients who were on medication and were treated with neurofeedback had decreased their medication requirements by follow-up, among the patients receiving traditional treatment, only one patient decreased medication needs, two reported no change, and 10 required an increase in psychiatric medications. On the Minnesota Multiphasic Personality Inventory, neurofeedback training patients improved significantly on all 10 clinical scales—dramatically on many of them—whereas there were no significant improvements on any scales in the traditional treatment group. One study (Huang-Storms, Bodenhamer-Davis, Davis, & Dunn, 2006) has also reported positive improvements in 20 adopted children with histories of abuse and/or neglect. Improvements were noted in externalizing and internalizing problems, social problems, aggressive and delinquent behavior, anxiety/depression, thought problems, and attentional problems. Neurofeedback seems very promising with posttraumatic stress disorder, but further corroborating research is needed.

Autism and Asperger’s Syndrome

There is a quite significant body of research that has now appeared on the neurofeedback treatment of autism and Asperger’s Syndrome (Coben & Myers, 2010; Coben & Pudolsky, 2007a; Jarusiwiwcz, 2002; Knezevic, Thompson, & Thompson, 2010; Kouijzer, de Moor, Gerrits, Buitelaar, & van Schie, 2009; Kouijzer, de Moor, Gerrits, Congedo, & van Schie, 2009; Kouijzer, van Schie, de Moor, Gerrits, & Buitelaar, 2010; Pineda et al., 2007; Pineda et al., 2008; Scolnick, 2005; Sichel, Fehmi, & Goldstein, 1995).

L. Thompson, Thompson, and Reid (2010) reported on a case series of 150 Asperger’s Syndrome patients and nine autism spectrum disorder patients who received 40 to 60 sessions, commonly with some supplementary peripheral biofeedback. They found very statistically significant improvements in measures of attention, impulsivity, auditory and visual attention, reading, spelling, arithmetic, EEG measures, and an average full scale IQ score gain of 9 points. Some of the studies just cited were control group studies. There has also been a placebo-controlled study (Pineda et al., 2008), and there have been 6-month (Kouijzer et al., 2010) and 1-year follow-ups (Kouijzer et al., 2009) documenting maintenance of positive results. A review of neurofeedback with autism spectrum problems, which includes a review of unpublished papers presented as scientific meetings, has been published by Coben, Linden, and Myers (2010). In an as-yet-unpublished study cited by those authors using neurofeedback and HEG training, Coben found a 42% reduction in overall autistic symptoms, including a 55% decrease in social interaction deficits and improvements in communication and social
interaction deficits of 55% and 52%, respectively. Overall, neurofeedback has positive research support as a beneficial treatment with autism spectrum problems, with findings of positive changes in brain function, attention, IQ, impulsivity, and parental assessments of other problem behaviors such as communication, stereotyped and repetitive behavior, reciprocal social interactions, and sociability. Although neurofeedback is certainly not a cure for these conditions, it appears to usually produce significant improvements in these chronic conditions.

**Anxiety and Depression**

Encouraging preliminary research has been published for the effectiveness of neurofeedback in treating anxiety with 10 controlled studies that have been identified (Hammond, 2005c; Moore, 2000). Of the eight studies of anxiety that were reviewed, seven found positive changes. Another study (Passini, Watson, Dehnel, Herder, & Watkins, 1977) used only 10 hr of neurofeedback with anxious alcoholics and found very significant improvements in state and trait anxiety compared to a control group, with results sustained on 18-month follow-up. A randomized, blinded, controlled study (Egner & Gruzelier, 2003) was done with performance anxiety at London’s Royal College of Music. They evaluated the ability of alpha/theta neurofeedback to enhance musical performance in high-talent-level musicians when they were performing under stressful conditions where their performance was being evaluated. When compared with alternative treatment groups (physical exercise, mental skills training, Alexander Technique training, and two other neurofeedback protocols that focused more on enhancing concentration), only the alpha/theta neurofeedback group resulted in enhancement of real-life musical performance under stress. Similar randomized controlled studies reducing performance anxiety have been conducted with musical performance (Egner & Gruzelier, 2003), ballroom dance performance (Raymond, Sajid, Parkinson, & Gruzelier, 2005), and performance in singing (Kleber, Gruzelier, Bensch, & Birbaumer, 2008; Leach, Holmes, Hirst, & Gruzelier, 2008). In a randomized, placebo-controlled study with medical students (Raymond, Varney, Parkinson, & Gruzelier, 2005) neurofeedback enhanced mood, confidence, feeling energetic and composed.

Neurofeedback has also been shown with objective measures to improve depression (Baehr, Rosenfeld, & Baehr, 2001; Hammond, 2001a, 2005b; Hammond & Baehr, 2009). The degree to which depressed patients were able to normalize their EEG activity during neurofeedback has been found to significantly correlate with improvement in depressive symptoms (Paquette, Beauregard, & Beaulieu-Prevost, 2009). A blinded, placebo-controlled study (Choi et al., 2011) demonstrated the superiority of neurofeedback over a placebo treatment in reducing depression while improving executive function. However, more research is needed on the use of neurofeedback with depression.

**Insomnia**

A randomized, controlled study (Hoedlmoser et al., 2008) demonstrated that only 10 neurofeedback sessions focused on reinforcing the SMR resulted in an increase in sleep spindles and reduced sleep latency. Because memory consolidation occurs during sleep, this study also documented improved memory in the subjects. This study replicated findings some earlier studies (Berner, Schabus, Wienerroither, & Klimesch, 2006; Sterman, Howe, & MacDonald, 1970). Hammer et al. (2011) published a randomized, single-blind controlled study documenting the effectiveness of 20 sessions of live Z-score training in the treatment of insomnia. Individualized neurofeedback was also shown in control group studies by Hauri (1981; Hauri, Percy, Hellekson, Hartmann, & Russ, 1982) to have long-lasting effects with insomnia patients. A recent randomized control group study (Cortoos, De Valck, Arns, Brelteer, & Cluydt, 2010) of primary insomnia patients found an average of 18 sessions of home neurofeedback training administered over the Internet produced a significant improvement in the time required to fall asleep and a significant improvement in total sleep time as measured in a sleep lab compared with a control group. Even three
schizophrenic or schizoaffective patients with disturbed sleep all showed improvement in sleep quality when compared with a control group (Cortoos et al., in press).

**Headaches and Migraine**

Walker (2011) reported on 71 recurrent migraine cases who consulted a neurological practice. Forty-six of the patients consented to QEEG-guided neurofeedback treatment, whereas 25 chose drug treatment. Excess higher frequency beta was present in all cases. At 1-year follow-up, 54% of the neurofeedback group experienced complete cessation of migraines compared with no one in the medication treatment group. In the neurofeedback group, 39% experienced a reduction of greater than 50% in migraines (compared with 8% with drug treatment), and a reduction of less than 50% was found in 4% of patients (compared to 20% with medication treatment). Sixty-eight percent of the medication treatment group reported no change in headache frequency, whereas only one patient (2%) receiving neurofeedback reported no reduction in frequency. Siniatchkin, Hierundar, Kropp, Gerber, and Stephani (2000) found a significant reduction in the number of days per month with a migraine in children treated with slow cortical potentials training versus a waitlist control group. Carmen (2004) reported improvement of more than 90% in migraine sufferers who completed at least six sessions of HEG training. For Stokes and Lappin (2010), 70% of migraine patients experienced at least a 50% reduction in frequency on more than 1-year follow-up from a combination of 40 neurofeedback sessions combined with HEG training. Tansey (1991a) published four case reports. Although encouraging, further controlled research is needed.

**Peak or Optimal Performance Training**

Neurofeedback is also being utilized in peak performance training (Vernon, 2005). For example, in a randomized, blinded controlled study (Egner & Gruzelier, 2003) neurofeedback significantly enhanced musical performance, and a similarly designed study (Raymond, Sajid, et al., 2005) documented significant improvements in ballroom dance performance. Such results have also been reported with golf (Arns, Kleinnijenhuis, Fallahpour, & Breteler, 2007), archery (Landers, 1991; Landers et al., 1994), improving fast reaction time and visuospatial abilities (which has relevance to athletic performance; Doppelmayr & Weber, 2011; Egner & Gruzelier, 2004), improving singing performance (Kleber et al., 2008; Leach et al., 2008), acting performance (Gruzelier, Inoue, Smart, Steed, & Steffert, 2010), and improvements in radar-monitoring tasks (Beatty, Greenberg, Diebler, & O’Hanlon, 1974). One fascinating study (Ros et al., 2009) compared training to either increase SMR or alpha and theta brainwave frequencies in ophthalmic microsurgeons in training, compared to a waitlist (no-treatment) group. In only eight sessions of SMR training the physicians demonstrated significant improvements in surgical skill, decreases in anxiety, and a 26% reduction in surgical task time. Research documenting improvements in cognitive and memory performance has already been reviewed earlier. The potential of neurofeedback applications for optimal performance will be very a fruitful area for further research.

**Other Clinical Applications of Neurofeedback Training**

Preliminary reports have also been published on the use of neurofeedback with chronic fatigue syndrome (Hammond, 2001b); Tourette’s (Tansey, 1986); obsessive-compulsive disorder (Hammond, 2003, 2004; Surmeli, Ertem, Eralp, & Kos, 2011); Parkinson’s tremors (M. Thompson & Thompson, 2002); tinnitus (Croce, Forti, & Bo, 2011; Dohrmann, Elbert, Schlee, & Weisz, 2007; Gosepath, Nafe, Ziegler, & Mann, 2001; Schenk, Lamm, Gundel, & Ladwig, 2005; Weiler, Brill, Tachiki, & Schneider, 2001); pain (Ibric & Dragomirescu, 2009; Jensen, Grierson, Tracy-Smith, Bacigalupi, & Othmer, 2007; Sime, 2004); physical balance, swallowing, gagging, and incontinence (Hammond, 2005a); children with histories of abuse and neglect (Huang-Storms et al., 2006) or reactive attachment disorder (Fisher, 2009); cerebral palsy (Ayers, 2004); restless legs and
periodic limb movement disorder (Hammond, in press); physical and emotional symptoms associated with Type I diabetes mellitus (Monjezi & Lyle, 2006); essential tremor; and for “chemo fog” (Raffa & Tallarida, 2010; Schagen, Hamburger, Muller, Boogerd, & van Dam, 2001) following chemotherapy or radiation treatments.

Mixed results have been found with neurofeedback treatment of fibromyalgia. An uncontrolled trial (Mueller et al., 2001) with 30 patients with fibromyalgia (using an early version of LENS) found significant improvements in mood, clarity, and sleep. C. C. S. Donaldson et al. (1998) used an earlier version of LENS (and a small amount of EMG biofeedback) and reported significant improvement in 77% of patients’ long-term follow-ups, but again this was an uncontrolled case series. In contrast, these results were not confirmed by Kravitz, Esty, Katz, and Fawcett (2006) in a double-blind, placebo-controlled study, and Nelson et al. (2010) found improvements in pain, fatigue, and cognitive clouding, and increased activity in comparison to a sham placebo control group, but the effects were not enduring. On the other hand, Kayiran, Dursan, Dursun, Ermutlu, and Karamursel (2010), in a randomized, blinded, control group study, compared 20 sessions of neurofeedback to treatment with Lexapro and found that both treatments produced significant symptomatic improvements, but the benefits were significantly greater in the neurofeedback group.

Research has shown that it is possible for schizophrenics to participate in neurofeedback training (Guzelier, 2000; Gruzelier et al., 1999; Schneider et al., 1992) and clinical experience with chronic schizophrenics (Bolea, 2010; Cortoos et al., in press; M. Donaldson, Moran, & Donaldson, 2010; Surmeli, Ertem, Eralp, & Kos, in press) provides encouragement that this may be an additional treatment intervention which holds potential.

IS MORE PLACEBO CONTROLLED RESEARCH NEEDED?

Despite the considerable research cited in this article, there are many areas where more controlled outcome research is still needed in the application of neurofeedback to various problems. Placebo-controlled studies are often regarded as the very highest level of scientific validation. It can be assumed that positive results from neurofeedback are due to a combination of expectancy (placebo) effects and effects specific to the neurofeedback treatment (Hammond, 2011; Perreau-Linck, Lessard, Levesque, & Beauregard, 2010), because placebo effects appear to be an active ingredient in virtually every therapeutic modality. We know, however, that there are improvements very specific to neurofeedback because there are several placebo-controlled studies that have demonstrated significant efficacious and specific effects beyond placebo influences in neurofeedback training (Raymond, Varney, et al., 2005), including with learning disabilities (Becerra et al., 2006; Fernandez et al., 2003), ADD/ADHD (deBeus & Kaiser, 2011; deNiet, 2011), anxiety (Raymond, Varney, et al., 2005), epilepsy (Lubar et al., 1981), sleep latency and declarative learning (Hoedlmoser et al., 2008), cognitive enhancement in the elderly (Angelakis et al., 2007), autism (Pineda et al., 2008), and depression (Choi et al., 2011), although one preliminary study did not find such effects (Lansbergen, van Dongen-Boomsma, Buitelaar, & Slaats-Wijmenga, 2010). Certainly animal studies (e.g., Sterman, 1973; Larsen, Larsen, et al., 2006) also suggest that neurofeedback has therapeutic effects independent of placebo effects. It would not be anticipated that cats would form positive expectancies about being more seizure resistant simply because an experimenter was putting electrodes on their heads.

In spite of the placebo-controlled studies we have in neurofeedback, some academic researchers (e.g., Loo & Barkley, 2005), insurance companies, and proponents of medication treatment have complained that there should be more placebo-controlled research on neurofeedback, even though medical ethicists (Andrews, 2001; Lurie & Wolfe, 1997; Rothman, 1987), neurofeedback advocates (La Vaque, 2001), and the Declaration of Helsinki (World Medical Association, 2000)
have expressed the view that requiring placebo-controlled studies in conditions where there is a known effective treatment already available is considered unethical. The primary benefit of placebo-controlled studies is that they clarify the mechanism of action by which a treatment works, but they are not necessary to determine the effectiveness of a treatment (e.g., the degree of improvement in attention and behavior in ADD/ADHD, and in comparison with stimulant drugs).

When considering how well validated common medical and psychiatric treatments actually are, it is enlightening to learn that only 11% of 2,711 cardiac medical treatment recommendations are based on multiple randomized controlled studies (Tricoci, Allen, Kramer, Califf, & Smith, 2009) and only 41% are based on evidence from a single randomized trial or nonrandomized studies, whereas 48% are simply based on “expert opinion” or only case studies. As yet a further example, the public is generally unaware of the fact that studies (summarized in Kirsch, 2010, and Moncrieff, 2009) of psychiatric medication treatment of depression have concluded that they are only mildly (18%) more effective than a placebo (and yet frequently associated with side effects and a withdrawal syndrome). Despite these facts, insurance companies accept medication treatment for depression and a large proportion of medical treatments as being well established and effective. These facts do not mean that more neurofeedback outcomes studies are desirable and needed, but it creates an important perspective that much of current medical and psychiatric treatment practice does not rest on as much sound scientific evidence as is commonly assumed.

ADVERSE EFFECTS, SIDE EFFECTS, AND HOME TRAINING

Mild side effects can sometimes occur during neurofeedback training. For example, occasionally someone may feel fatigued, spacey, or anxious; experience a headache; have difficulty falling asleep; or feel agitated or irritable. Sometimes such side effects may occur because the training session is too long (Matthews, 2007; Ochs, 2007). Many of these feelings pass within a short time after a training session. If clients make their therapists aware of such feelings, they can alter training protocols and usually quickly eliminate such mild side effects.

Selecting a Qualified Practitioner

It is possible, however, for more significant negative effects to occur (Hammond & Kirk, 2008; Hammond, Stockdale, Hoffman, Ayres, & Nash et al., 2001; Todder, Levine, Dwolatzky, & Kaplan, 2010), particularly if training is not being conducted or supervised by a knowledgeable, certified (http://www.bcia.org) professional who will individualize the training. A “one-size-fits-all” approach that is not tailored to the individual will undoubtedly pose a greater risk of either being ineffective or of producing an adverse reaction. Due to the heterogeneity in the brainwave activity (e.g., Clarke et al., 2001; Hammond, 2010b; Prichep et al., 1993) within broad diagnostic categories (e.g., ADD/ADHD, head injuries, depression, autism, or obsessive-compulsive disorder) the treatment requires individualization, and research is increasingly showing that different treatment protocols have differential effects (e.g., Angelakis et al., 2007; Egner & Gruzelier, 2004; Gevensleben et al., 2009a, 2009b; Gruzelier & Egner, 2005; Hauri, 1981; Hauri et al., 1982; Heinrich et al., 2004; Ros et al., 2010; Wrangler et al., 2010) on the brain.

Thus, it is emphasized once again that everyone does not need the same treatment and that if training is not tailored to the person, the risk is greater of it being ineffective or very infrequently even detrimental. For instance, Lubar et al. (1981) published a reversal double-blind controlled study with epilepsy which documented that problems with seizure disorder could be improved with neurofeedback, but they could also be made worse if the wrong kind of training was done. Similarly, Lubar and Shouse (1976, 1977) documented that ADD/ADHD symptoms could improve but also be worsened if inappropriate training was done. As yet another example in the treatment of ADD/ADHD, it was found that when a nonindividualized approach was used (Steiner,
Sheldrick, Gotthelf, & Perrin, 2011) with one electrode embedded in a helmet compared with computerized attention training, only modest equivalent results were found. In contrast, when individualized neurofeedback was compared with computerized attention training (Gevensleben et al., 2010; Gevensleben et al., 2009a, 2009b; Holtmann et al., 2009), neurofeedback was significantly more effective than the skills training.

Therefore, seeking out a qualified and certified professional who will do a comprehensive assessment of brain function (e.g., with a QEEG or careful assessment of the raw EEG activity) is deemed to be vitally important. If the practitioner indicates that they do a “brain scan” or QEEG, it is important to determine whether the EEG data are actually being statistically compared to a normative database rather than simply being roughly measured.

If you are seeking help for a psychological, psychiatric, or medical problem like those discussed in this article, the ISNR (Hammond et al., 2011) has recommended that you determine that the practitioner you select is not only certified but also licensed or certified for independent practice in your state or province as a mental health or health care professional. An increasing number of unqualified and unlicensed persons are managing to obtain neurofeedback equipment and seeking to basically practice psychology and medicine without a license. It has unfortunately become a “buyer beware” marketplace.

In this regard, some individuals are now renting and leasing home training equipment. It is strongly recommended that training with equipment at home should be done only under the regular consultation and supervision of a legitimately trained and certified professional, and preferably home training should occur only following closely supervised training that has taken place in the office for a period of time (Hammond et al., 2011). It is important to caution the public that if this is not done, some negative effects (and a higher probability of ineffective results) could occur from such unsupervised self-training. It is important to remember that the impressive success documented in most of the research on neurofeedback is based on work conducted by qualified professionals, following individualized assessment, and with training sessions that are supervised by a knowledgeable therapist rather than with unsupervised sessions taking place in an office or at home. Supervised training sessions where the patient is coached have been found to produce significantly better outcomes than unsupervised sessions (Hammond, 2000).

REFERRAL SOURCES

Readers may identify certified practitioners who are doing neurofeedback training by consulting the website for the Biofeedback Certification International Alliance (http://www.bcia.org) and by examining persons who are licensed and listed in the membership directory for ISNR (http://www.isnr.org). In addition to the references included in this article, the ISNR website also includes a comprehensive bibliography of outcome literature on neurofeedback, which is periodically updated.

REFERENCES


Doppelmayr, M., Nosko, H., Pecherstorfer, T., & Fink, A. (2007). An attempt to increase


Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O. ... Heinrich, H.


Sime, A. (2004). Case study of trigeminal neuralgia using neurofeedback and peripheral
biofeedback. *Journal of Neurotherapy, 8*(1), 59–71.


Academy of Child & Adolescent Psychiatry, 46, 1015–1027.


