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Stella B. Legarda, MD¹, Doreen McMahon, MD²,
Siegfried Othmer, PhD³, and Sue Othmer, BCIAC³

Abstract

Trends in alternative medicine use by American health care consumers are rising substantially. Extensive literature exists reporting on the effectiveness of neurofeedback in the treatment of autism, closed head injury, insomnia, migraine, depression, attention deficit hyperactivity disorder, epilepsy, and posttraumatic stress disorder. We speculated that neurofeedback might serve as a therapeutic modality for patients with medically refractory neurological disorders and have begun referring patients to train with clinical neurofeedback practitioners. The modality is not always covered by insurance. Confident their child's medical and neurological needs would continue to be met, the parents of 3 children with epilepsy spectrum disorder decided to have their child train in the modality. The children's individual progress following neurofeedback are each presented here. A proposed mechanism and practice implications are discussed.

Keywords

neurology, epilepsy, neurofeedback, electroencephalogram

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Historical Overview

According to the Centers for Disease Control and Prevention (CDC), at least 14 million children in the United States have chronic brain disorders for which there is no medical treatment, representing 17% of all children between birth and 19 years of age.¹ Consumer spending in alternative therapies, among these biofeedback, has increased to 34 billion dollars in 2009² compared with 27 billion dollars in 1997 (representing a 45.2% increase from 1990), when the rate increased from 36.3% to 46.3%.³

Neurofeedback is brain biofeedback. More specifically, it is electroencephalographically (EEG) recorded brain activity biofeedback. It has reported benefit in managing disorders in adults such as epilepsy, migraine, depression, chronic insomnia, traumatic brain injury, and posttraumatic stress syndrome.⁴⁻⁹ A recent meta-analysis review concluded that a significant reduction in seizure frequency occurs in adults with epilepsy who train with neurofeedback ($P \leq .001$).¹⁰ The vast majority of clinical neurofeedback work with children has been for attentional complaints and related behavioral problems.^{11,12} In 2007, a randomized trial indicated clinical efficacy of neurofeedback in managing children with attention deficit hyperactivity disorder (ADHD)¹³ and a recent meta-analysis review of the neurofeedback literature reporting on this therapy in children with ADHD supports this finding.¹⁴

In practice, clinical neurofeedback is EEG operant conditioning.^{8,15} The individual receiving neurofeedback is given a computerized video program to watch (a game or a show), the content of which is driven by real-time EEG "behavior" at examiner-selected EEG frequencies. Only by abiding enough in the selected frequencies does the video-game or show proceed normally (reward). By training in the selected frequencies the individual "learns" to enter a better regulated state, sometimes for the first time.

First researched by Serman and colleagues in the 1960s on cats,¹⁵ the premise was to alter mammalian physiologic states by conditioning EEG-derived neural network activation/de-activation dynamics. Grounded in Skinnerian operant conditioning the immediate target of Serman's method was *brain*

¹ Georgetown University Medical Center, Departments of Pediatrics and Neurology, Washington, DC, USA

² NOVA Neurointegrative Medicine, Maclean, Virginia, USA

³ EEG Institute (Biofeedback Certification Institute of America, BCIAC), Woodland Hills, California, USA

Corresponding Author:

Stella B. Legarda, MD, Pediatrics and Neurology, Georgetown University Hospital, 3800 Reservoir Road, NW, 2nd Floor, Pasquerilla Healthcare Center, Washington, DC 2000, USA

Email: m.stella.legarda@gunet.georgetown.edu

“operant” behavior (EEG activity), as opposed to overt physiologic and motoric behaviors previously targeted by either Pavlovian or Skinnerian methods. Sterman theorized this to be feasible by training cats in specific, accessible, measurable and recordable EEG frequencies, particularly those that reflected motoric excitability. By virtue of electrode placement on the sensorimotor strip, he subjected cats to this training by rewarding them (with chicken broth and milk) when they produced somatomotor rhythms (SMR) of 12–19 Hz. In a later experiment, Sterman showed that compared with other cats also injected with toxic monomethylhydrazine (a rocket fuel) the cats who underwent SMR training took twice as long to exhibit expected toxicity (seizures). Sterman concluded that seizure thresholds changed in the cats that had experienced SMR training.

Since Sterman’s classic experiments and his later studies reporting efficacy of neurofeedback in humans with epilepsy,^{16,17} an explosion of unsupported claims regarding the health benefits of neurofeedback has made it difficult to discern any actual effect. New York Times science reporter, Jim Robbins attempted to unravel the claims and rally the science behind neurofeedback in the well written book *A Symphony in the Brain*,¹⁸ now in its second edition. Independent national news reports of chronic illness success stories after training with neurofeedback and televised interviews of neurofeedback clinicians with their clients on shows like *Dr. Phil* are likewise informing public healthcare consumers. The medical community, however have largely ignored the claims.

Clinical Neurofeedback

The practice of neurofeedback promotes real-time EEG-selected frequencies and trains the individual in these examiner-selected frequencies. How successfully the patient abides in these frequencies is measured during the session. We do not know what is happening in the brain during neurofeedback, much as we do not know where many abnormal and variant EEG findings ultimately originate given the parallel, transcallosal, and hierarchical processing that co-occur in the central nervous system at any given moment.

There are few objective measures for the effects of neurofeedback. The quantitative electroencephalogram (QEEG) is used by many in the field to guide treatment and monitor response to neurofeedback. The QEEG represents approximately 60 seconds (2–5 minutes at most) of artifact-free routine awake background EEG that is computer analyzed to generate an integrated spectral map of frequencies, amplitudes, and their relative power. This QEEG baseline activity is then compared statistically with normative databases (with respect to spectral amplitude, spectral power, relative band amplitude, hemispheric asymmetry, and coherence).¹⁹ To quote a respected pioneer in clinical neurofeedback: “If there is focal excessive power in a frequency band, it may be downtrained. If there is a focal deficiency in power, it may be uptrained. Similarly, significantly decreased coherence between brain areas may be uptrained, and significantly increased coherences may be

downtrained.”²⁰ (Coherence in QEEG is defined as “a measure of phase synchrony or shared activity between spatially distant generators.”)^{19,21}

Functional assessment commonly includes a continuous performance test. The Tests of Variables of Attention (TOVA[®]) is an individually administered set of computerized choice reaction time tests developed to assess attention, maintenance of vigilance, impulse control, and consistency of nervous system functioning.²² Testing with the TOVA at baseline and following several neurofeedback sessions allows for performance comparisons. Additionally, symptom profiles have been devised that are scored at baseline and before each neurofeedback session.²³ Individuals who train in neurofeedback most often report a subjectively felt state shift (calmness, alertness, clarity) that is experienced immediately and can last days. Adverse experiences can also occur. State shifts might be induced that are not comfortable for the individual. EEG electrode placement close to the eye sometimes unintentionally trains for decreased blinking and can cause visual discomfort and even visible conjunctival changes. Headaches and changes in energy levels have also been reported (personal observations).

Case Report

We are reporting on data for 3 individuals who were referred for clinical neurofeedback. After an initial review of the neurofeedback literature several patients with medically refractory neurological disorders were recommended to train with neurofeedback while remaining under physician supervision by 1 of the authors (S.L.). Two patients had well-controlled epilepsy with significant life-altering comorbidities (presented at the American Epilepsy Society meeting, December 2010).²⁴ A third individual with epilepsy is also discussed (not previously reported).

Study Profiles

Case A. A 19-year-old male with refractory epilepsy managed by vagus nerve stimulation and 3 anticonvulsants, zonisamide, oxcarbazepine, and rufinamide who also suffered comorbidities of insomnia, depression, chronic daily migraine type headaches with intermittent exacerbations, and ADHD. Before initiating neurofeedback these comorbidities were managed by multiple medications (see Table 1). He would also present for unanticipated clinic visits at least weekly to receive acute headache management in the form of intravenous nalbuphine with promethazine. Case A’s past EEGs revealed bilateral frontal-temporal paroxysmal theta and multiphasic sharp potentials involving predominantly Fp2/F4, F8 (standard 10–20 nomenclature for right lateral frontal EEG scalp electrode placements) and homologous regions (Fp1/F3, F7, left lateral frontal), occurring independently and rarely synchronously.

Case B. A 6-year-old ex-32 weeks premature male with cerebral palsy (status post birth asphyxia, walking independently with bilateral ankle-foot orthoses), autism (nonverbal, behavioral

Table 1. Assessments at Baseline and After Several Neurofeedback Sessions

Case	Sex	Age (y)	Test						
			TOVA	QEEG	Observer ratings	Medications	Sites trained	ORF	
A	Male	19	Significant for ADHD plus disinhibition	F7/T3/T4 F3/F4 T4/T6/O2 Excessive low Frequencies	No seizures/ VNS implanted 6 months ago Anxiety score 4 (low result) OCD score 0 At least weekly acute clinic visits for IV Nubain/ Phenergan to abort headache Headache rating 8-10 Sleep difficulty rating 10	Triazolam Quetiapine Hydrocodone-acetaminophen Amitriptyline Sertraline Methylphenidate Oxcarbazepine Rufinamide Zonisamide Nalbuphine/ Promethazine prn	T3-T4 C3-C4	1.5 Hz All sessions	
Post-NF 13 sessions Patient discontinued due to financial hardship			Not done	Not done	Not done Headache rating 4-6 (a 2 after sessions) Not one stat clinic visit for IV meds Sleep difficulty 6 Autism profile score 59	Oxcarbazepine Rufinamide Zonisamide Triazolam prn			
B	Male	6	Unable	Diffuse theta Frontal delta	59		Sertraline Catapres Topiramate Risperidone Fluoxetine	T3-T4 C3-C4 T4-P4 T4-Fp2	0.001 Hz 21 sessions
Continuing sessions Post-NF 21 sessions			Not done	Not done	22		Topiramate Catapres prn		0.0001 Hz
C	Female	16			Diurnal and nocturnal seizures, 2-3 x/ week			T3-T4	10 Hz
Pre-NF			"ADD" -4.09	Sharp wave discharges at CZ/C3, T5, F4	Spacey, unfocused Struggling in school (GPA 3.0) Sleep disorder, volatile moods, fatigue	None		FP1-T3 CZ-T4 C4-T4	39 sessions
Continuing sessions Post-NF			Normal	Not done	Better focus, moods stabilized, sleep improved, school improvements (GPA 3.9)				0.0001 Hz
44 sessions			4.72		Driver's license, no seizures				

TOVA = Tests of Variable Attention; QEEG = quantitative EEG (did not guide therapy); ORF = optimal reinforcement frequency; OCD = obsessive compulsive disorder; ADD = attention deficit disorder; ADHD = attention deficit hyperactivity disorder; pre-NF = before neurofeedback; Post-NF = following neurofeedback; GPA = general point average.

stereotypy of bringing hands up to each side of face, with major social and developmental challenges in both cognitive and motor domains), profound mental retardation, and remote symptomatic epilepsy managed by topiramate. The patient had recently presented with “new onset seizures” and failed an initial trial on oxcarbazepine before achieving seizure control with topiramate. A distressing chronic symptom was intermittent aggressive self-stimulation in the form of repetitively hitting himself in the head with his fists to the point of bruising and bleeding. He also had a severe insomnia, keeping the household awake most nights. He failed sequential medication trials to control these injurious behaviors (see Table 1). Case B’s EEG revealed a multifocal epileptic encephalopathy with maximum spike activity and slowing at T5 and P3 (left posterior temporal and left parietal).

Case C. A 16-year-old female who experienced her first grand mal at age 5 years. Thereafter, she had repeatedly witnessed diurnal “staring” seizures, most lasting less than 5 seconds. Continuous video-monitoring documented staring behaviors to occur as frequently as 4–5 times per minute accompanied by bilateral rhythmic frontal slow delta. Nocturnal seizures occurred 2–3 times a week and were witnessed as falling out of bed, thrashing, and crying out. She experienced 1 more grand mal episode at age 15 years. Interictal EEG showed C4/T4 (right central–temporal) sharp and slow wave complexes. Magnetic resonance imaging (MRI) of her brain revealed an asymmetry of the hippocampal gyri, right larger than left. She was initially diagnosed with benign rolandic epilepsy then complex partial epilepsy with secondary generalization accompanied by problems of inattention, depressed mood, and sleep disturbances. Levetiracetam and lamotrigine caused allergic reactions. Oxcarbazepine, even at subtherapeutic doses caused somnolence. Case C refused to take further medications and would not follow a ketogenic diet.

Methods

Baseline performance tests consisted of symptom profiles, TOVA, QEEG, and other observer evaluations (see Table 1). The TOVA and QEEG were repeated after 20 sessions and compared with pretreatment baseline. Symptom profiles were reviewed at each session.

In all subjects, the QEEG was not found to be as useful as the symptom profiles in guiding the individual neurofeedback protocols. All subjects declined a posttreatment QEEG because of costs.

The computer system used for clinical neurofeedback was Cygnet® neurofeedback software integrated with Somatic Vision videogames and run by means of Windows (XP or Vista) operating system using standard personal computer (PC) desktops and high-resolution monitors.

The optimal reinforcement frequency (ORF)²³ was determined for the individual at the beginning of each neurofeedback session based on subjective reporting by the patient and/or observer ratings of behavioral alertness (the ORF being within the clinical EEG band, including the infra-slow region ≤ 0.05 Hz).^{25,26} Training at their ORF occurred with bipolar placement of EEG electrodes at T3–T4 (left and right temporal scalp electrode placements) according to standard

10–20 nomenclature, applied after scalp preparation (Nu-Prep®) using standard electrode paste (Ten20 Conductive®) to secure 3 sintered silver/silver chloride scalp electrodes at T3, T4, and at Fpz (a vertex prefrontal scalp electrode placement) as “ground” electrode. The T3–T4 bipolar recording was used to bias the training toward EEG desynchronization, theorizing that promotion of enhanced coherence at the target frequency can be contraindicated for individuals with a susceptibility to seizures. Subsequently, each patient was scheduled to receive at least 20 separate 30-minute neurofeedback sessions over a period of 4–8 weeks.

Rationale

Anterior temporal training (T3–T4) was the starting placement in all sessions to effect general calming and stabilizing of both limbic and other physiological functions. These effects are believed to be mediated through temporal lobe connections with the insula and limbic circuitry. Because the individuals in this study had epilepsy, central training (C3–C4, left and right central electrode placements) was also performed to impact somatosensory regions involved in seizures.²³ In Case B, right temporal-prefrontal training (T4–Fp2) was performed to improve control of basic emotional regulation, specifically emotional reactivity. Right temporal-parietal training (T4–P4) was also performed to calm sensory hypersensitivities and improve sensory integration. Parietal training is also believed to increase social–emotional awareness and empathy.²³ In Case C, the right centrottemporal regions (underlying central vertex to temporal scalp electrode placements, Cz–T4/C4–T4) were specifically trained to address the regional hyper-excitability (C4/T4 spikes) the EEG had demonstrated. Training at T3–Fp1 (left temporal–prefrontal) was also performed to benefit executive control and focus.

Case A. Trained optimally at 1.5 Hz on average, and was specifically trained at T3–T4 and intermittently at C3–C4. After 2 sessions, the implanted vagus nerve stimulator (VNS) device was temporarily inactivated during all subsequent neurofeedback sessions.

Case B. Trained optimally at infra-slow EEG frequencies, with a bandpass extending down to 0.001 Hz (the lowest available frequency at the time of this report) and specifically at T3–T4, C3–C4, T4–P4, and T4–Fp2.

Case C. Trained optimally at 8.5–11.5 Hz with inhibit bands from 0 to 40 Hz for a total of 39 sessions at T3–T4, Cz–T4, C4–T4, and T3–Fp1, then at 0.0001 Hz to include training at T4–P4.

Results (see Table 1)

Case A

At baseline, Case A scored “significantly deviant” on the TOVA for attention deficit and hyperactivity disorder and behavioral disinhibition. He had low scores for anxiety and obsessive compulsive parameters. He rated headache severity as 8–10 and subjective sleep difficulty as 10 (subjective range, 1–10). Excessive slow frequencies were found on QEEG at F7/T3 as well as at F3/F4 and T4/T6/O2.

During his first 2 sessions of neurofeedback, Case A experienced less than expected response. Subsequently his VNS device was inactivated before each neurofeedback session and

reactivated after each session was completed. This individual completed only 13 sessions due to financial constraints. His ratings for headache and sleep decreased to 6. His medications were gradually discontinued over time except for the anticonvulsants (see Table 1), and he stopped coming to the clinic for intravenous pain control medications.

Case B

At baseline Case B scored 59 in his autism profile. The QEEG revealed diffuse theta and frontal delta.

Case B completed 21 sessions. His autism profile score after 21 sessions was 22, an improvement of 63% from baseline (see Table 1). Improvements included the return of a normal sleep cycle. He continues to train in neurofeedback with a band pass now extending down to 0.0001 Hz.

Case C

At baseline, Case C had a TOVA “suggestive of ADHD” (-4.09) with slow response times that were highly variable (1st percentile). Impulsivity scores were especially deviant in the first half of the test. The QEEG showed independent sharp wave discharges at Cz/C3, T5 (left posterior temporal) and F4 (right mid-frontal). Case C’s irregular seizure activity began to decrease in frequency in that the nocturnal events resolved after 15 neurofeedback sessions. After 20 sessions, she appeared to remain fully alert during the day, becoming more fully engaged in daily activities. No observable seizure activity has occurred for over 2 years. School grade point average rose from 3.0 to 3.9, and the TOVA score normalized (+4.72). Case C’s sleep and moods stabilized; she participates in after-school activities, and she holds a driver’s license. Case C continues with neurofeedback on a maintenance basis.

Proposed Mechanism

We propose that clinical neurofeedback at infra-slow frequencies (below 0.1 Hz) engages the known biological very slow frequencies^{25,26} that are understood to modulate cortical excitability.²⁷ These have been routinely filtered out during standard EEG recordings (the slow phasic direct current (DC) shifts distort the visualized EEG baseline) and thus have been relatively neglected by EEG researchers. Using DC-coupled EEG scalp recordings, spontaneous (nonevoked) infraslow oscillations from 0.02 to 0.2 Hz have been demonstrated to be strongly correlated with interictal epileptic events and with faster EEG activities and K complexes in individuals with and without epilepsy.²⁷ These very slow frequencies are termed the ISO (infra-slow oscillations)^{27,28} as well as “infra-slow rhythmic oscillations,”²⁵ and participate in the modulation of neuronal frequencies such as alpha and mu (or somatomotor, SMR) rhythms.²⁸ The intimate relationships between apparently independent EEG phenomenology can serve to explain why the varied clinical approaches to neurofeedback (e.g., training either at infra-slow or at SMR frequencies) are met with wide claims of

benefit. Most investigators believe the ISO is non-neuronal in origin.^{28–30} Adenosine deficiency in the central nervous system is an established pathologic hallmark of epilepsy^{31,32}; recent elegant studies demonstrate the ISO to be dependent on adenosine, derived from the adenosine triphosphate (ATP) of astrocytes.²⁸ The ISO has been found to orchestrate the asynchronous “stadium wave” type firing of thalamocortical sensory neurons, is induced by moderate stimulation of metabotropic glutamate receptors as well as acetylcholine receptors, and is unaffected by *N*-methyl-D-aspartic acid (NMDA), alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA), and gamma-aminobutyric acid (GABA) blockers. It disappears when G-protein-activated inwardly rectifying potassium (GIRK) channels are blocked and in other ways demonstrates properties approaching the *resting membrane potential* of neurons.²⁸

Vagus nerve stimulation is an established exogenous source for inducing “central nervous system regulation” in individuals with epilepsy.³³ Direct low frequency stimulation (0.5 Hz) to ictal zones in human cortex has been shown to suppress seizures.^{34,35} Unlike these methods, clinical neurofeedback evokes the slow rhythm, or at least promotes it or merely engages with it. The technique is noninvasive and exercises the individual in the task of regulation, promoting endogenous self-regulation of the individual’s central nervous system resting state networks.

In summary, the reinforcement challenge during neurofeedback aims to adjust the “set point” of an individual’s arousal level and attain long-term stability in the individual’s habitual arousal state. Theoretically, enhanced stability is achieved because the challenge induces a beneficial shift in coherence relationships within central nervous system resting state networks. It does this in the following way. The ISO is differentially amplified (by basic EEG method) and the net signal is reinforced during neurofeedback to induce the shift. Thus the net signal being differentially amplified (and reinforced) directly reflects central nervous system resting network coherence relationships and biasing the training toward desynchronization of network activity (by the method described in this report) directly supports long-term central nervous system stability. In our patient-driven method using symptom profiles we find this process to be especially effective when the reinforcement engages spectral components of the slow cortical potential down to the region of milli-Hertz. The ISO is likely affected by a variety of physiologic states³⁶ and inducements²⁸ including neurofeedback. The point is neurofeedback does impact the ISO, an impact that *in first order* is mediated by brain activity.

Conclusions

The results in all 3 cases support the hypothesis that neurofeedback is a useful therapeutic modality for managing catastrophic neurological disorders such as epilepsy and its comorbidities. Indeed, Case C, who remains seizure-free and lives a normal young adult life 2 years after beginning neurofeedback, demonstrates that neurofeedback alone can effect

epilepsy control. Multiple medications were discontinued following neurofeedback therapy averting potential adverse effects from polypharmacy. Case A completed only 50% of his planned sessions (due to cost) and still experienced up to 40% reduction in his comorbid symptoms. It is likely that the mechanisms giving rise to epilepsy in both cases A and B were also positively impacted given that the comorbidities improved; first, because of supportive literature in this regard,^{4,17,20} and second, because Case C's epilepsy came under control without the use of pharmacotherapy.

These are case studies. The therapy is not widely covered by insurance, limiting its heuristic evaluation. There is a need for controlled studies to investigate the reported merits of neurofeedback in managing epilepsy and other pervasive neurological disorders. Physician-driven protocols investigating the process by which clinical neurofeedback exerts its effects can shed light on mechanisms of epilepsy spectrum disorders and other central nervous system disordered states.

Practice Implications

The co-occurrence of central nervous system disorders fundamentally reflects an intrinsic dysregulated state. The ability of patients to train with neurofeedback to reinforce an optimal baseline of central nervous system self-regulation decreases the need for multiple prescriptions, decreases the incidence of potentially adverse drug interactions, improves overall quality of life and promotes a sense of self-empowerment and of self-healing. The success of neurofeedback in this regard, fully researched and better understood, can lead to a more integrative understanding of the multi-level mechanisms underlying central nervous system disordered states such as epilepsy and its comorbidities.

The reported benefits of neurofeedback in managing neurological disorders such as autism syndromes, attention deficit hyperactivity disorder, postconcussion syndromes and insomnia without the need for pharmacotherapy^{7,11,12,21} suggest that current neurological services would be augmented by this modality. The therapy offers a nonpharmacologic intervention similar to the ketogenic diet for children with epilepsy who do not tolerate or respond well to established antiepileptic drugs. The practice is Food and Drug Administration-approved as "biofeedback" therapy and physician provision of this therapy nullifies additional out-of-pocket costs to the patient.

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Author Contributions

Dr. Legarda provided medical supervision and actively guided the neurofeedback protocols for cases A and B. Dr. McMahon is certified in the Othmer Method of neurofeedback and contributed the history and data for Case C. Sue Othmer, BCIAC (Biofeedback Certification Institute of America), is Clinical Director and Dr. Siegfried Othmer is Chief Scientist of the EEG Institute, Woodland Hills, California.

Their combined experience and training of clinicians in the field of clinical neurofeedback contributed to both the clinical methodology and writing of this manuscript.

Declaration of Conflicting Interests

The authors declared a potential conflict of interest (eg, a financial relationship with the commercial organizations or products discussed in this article) as follows: Dr. and Mrs. Sue Othmer's son is CEO of EEG Info, a company involved in the development of technology for clinical neurofeedback.

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Ethical Approval

A case report, meaning a retrospective analysis of 1–3 clinical cases, does not require prospective Institutional Review Board review and approval, as it is not viewed by the Georgetown University Institutional Review Board to be human subject research within the meaning of Georgetown University policies and procedures and the Common Rule (CFR Title 45, Part 46).

References

1. Boulet SL, Boyle CA, Schieve LA. Health care use and health and functional impact of developmental disabilities among US children, 1997–2005. *Arch Pediatr Adolesc Med.* 2009;163:19–26.
2. Dyess D. Health News. American Spending on Alternative Medicine Continues to Skyrocket. July 31, 2009. <http://www.healthnews.com/natural-health/alternative-medicine/american-spending-alternative-medicine-continues-skyrocket-3514.html>. Accessed Month day, year.
3. Eisenberg Davis RB, Ettner SL, Appel S, et al. Trends in alternative medicine use in the United States, 1990–1997. *JAMA.* 1998;280:1569–1575.
4. Serman, MB, Egner T. Foundation and practice of neurofeedback for the treatment of epilepsy. *Appl Psychophysiol Biofeedback.* 2006;31:21–35.
5. Walker J. QEEG-guided neurofeedback in migraine headaches. *J Clin EEG Neurosci.* 2010;42:59–61.
6. Kwon JS, Youn T, Jung HY. Right hemisphere abnormalities in major depression: quantitative electroencephalographic findings before and after treatment. *J Affect Disord.* 1996;40:169–173.
7. Feinstein B, Serman MB, MacDonald LR. Effects of sensorimotor rhythm training on sleep. *Sleep Res.* 1974;3:134.
8. Thatcher RW. EEG operant conditioning (biofeedback) and traumatic brain injury. *Clin Electroencephalogr.* 2000;1:38–44.
9. Peniston EG, Kulkosky PJ. Alpha-theta brainwave neurofeedback therapy for Vietnam veterans with combat-related post-traumatic stress disorder. *Med Psychotherapy.* 1991;4:47–60.
10. Tan G, Hornby J, Hammond DC, et al. Meta-analysis of EEG biofeedback in treating epilepsy. *J Clin EEG Neurosci.* 2009;40:173–179.
11. Fernandez T, Herrera W, Harmony T, et al. EEG and behavioral changes following neurofeedback treatment in learning disabled children. *Clin Electroencephalogr.* 2003;34:145–152.

12. Strehl U, Leins U, Goth G, et al. Self-regulation of slow cortical potentials: a new treatment for children with attention-deficit/hyperactivity disorder. *Pediatrics*. 2006;118:1530-40.
13. Gevensleben H, Holl B, Albrecht B, et al. Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. *J Child Psychol Psychiatry*. 2009;7:780-789.
14. Arns M, de Ridder S, Strehl U, et al. Efficacy of neurofeedback treatment in ADHD: the effects on inattention, impulsivity and hyperactivity: a meta-analysis. *Clin EEG Neurosci*. 2009;40:180-189.
15. Serman MB, Wyrwicka W, Roth SR. Electrophysiological correlates and neural substrates of alimentary behavior in the cat. *Ann N Y Acad Sci*. 1969;157:723-739.
16. Serman MB, MacDonald LR. Effects of central cortical EEG feedback training on incidence of poorly controlled seizures. *Epilepsia*. 1978;19:207-222.
17. Serman MB, MacDonald LR, Stone RK. Biofeedback training of the sensorimotor EEG rhythm in man: effects on epilepsy. *Epilepsia*. 1974;15:395-417.
18. Robbins J. *A Symphony in the Brain: The Evolution of the New Brain Wave Biofeedback*. Revised Edition. New York, NY: Grove Press; 2008.
19. Budzynski TH, Budzynski HK, Evans JR, Abarbanel A. *Introduction to Quantitative EEG and Neurofeedback: Advanced Theory and Applications*. 2nd ed. San Francisco, CA: Academic Press (Elsevier); 2009.
20. Walker JE, Kozlowski G. Neurofeedback treatment of epilepsy. *Child Adolesc Psychiatr Clin N Am*. 2005;14:153-176.
21. Walker JE, Norman CA, Weber RK. Impact of QEEG-guided coherence training for patients with a mild closed head injury. *J Neurotherapy*. 2002;6:31-43.
22. Greenberg LM, Waldmant ID. Developmental normative data on the test of variables of attention (T.O.V.A.). *J Child Psychol Psychiatry*. 1993;34:1019-1030.
23. Othmer S. *Protocol Guide for Neurofeedback Clinicians: Optimizing Electrode Placements and Reward Frequencies Step-By-Step Through Assessment and Training*. 2nd ed. Woodland Hills, CA: EEG Institute; 2008.
24. Legarda S, Ford S, Kondratyev A, Gale K. Nonpharmacologic management of epilepsy comorbidities. Abstract 2.068 2010. Paper presented at American Epilepsy Society 64th Annual Meeting. www.aes.net.org.
25. Aladjalova NA. Infra-slow rhythmic oscillations of the steady potential of the cerebral cortex. *Nature*. 1957(May):957-959.
26. Monto S, Palva S, Voipio J, Palva JM. Very slow EEG fluctuations predict the dynamics of stimulus detection and oscillation amplitudes in humans. *J Neurosci*. 2008;28:8268-827.
27. Vanhatalo S, Palva JM, Holmes MD, et al. Infralow oscillations modulate excitability and interictal epileptic activity in the human cortex during sleep. *Proc Natl Acad Sci U S A*. 2004;101:5053-5057.
28. Lorincz ML, Geall F, Boa Y, et al. ATP-dependent infra-slow (<0.1 Hz) oscillations in thalamic networks. *PLoS One*. 2009;4:e4447.
29. Parri HR, Gould TM, Crunelli V. Spontaneous astrocytic Ca²⁺ oscillations in situ drive NMDAR-mediated neuronal excitation. *Nat Neurosci*. 2001;4:803-812.
30. Parri HR, Crunelli V. Pacemaker calcium oscillations in thalamic astrocytes in situ. *Neuroreport*. 2001;12:3897-3900.
31. Li T, Ren G, Lusardi T, et al. Adenosine kinase is a target for the prediction and prevention of epileptogenesis in mice. *J Clin Invest*. 2008;118:571-582.
32. Boison D. Adenosine augmentation therapy for epilepsy. *Epilepsia*. 2010;51(Suppl):97.
33. Lundgren J, Amark P, Blennow G, et al. Vagus nerve stimulation in 16 children with refractory epilepsy. *Epilepsia*. 1998;39:809-813.
34. Schrader LM, Stern JM, Wilson CL, et al. Low frequency electrical stimulation through subdural electrodes in a case of refractory status epilepticus. *Clin Neurophysiol*. 2006;117:781-788.
35. Luders, HO. *Deep Brain Stimulation and Epilepsy*. New York, NY: Martin Dunitz; 2004.
36. Mantini D, Perrucci MG, Del Gratta C, Romani GL, Corbetta M. Electrophysiological signatures of resting state networks in the human brain. *Proc Natl Acad Sci U S A*. 2007;104: 3170-13175.