

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/8163422>

# Neurofeedback with anxiety and affective disorders

Article in *Child and Adolescent Psychiatric Clinics of North America* · February 2005

DOI: 10.1016/j.chc.2004.07.008 · Source: PubMed

---

CITATIONS

171

---

READS

2,198

1 author:



**D. Corydon Hammond**

University of Utah

106 PUBLICATIONS 2,162 CITATIONS

SEE PROFILE

Some of the authors of this publication are also working on these related projects:



Easing back [View project](#)

# **Neurofeedback with Anxiety and Affective Disorders**

## **Journal of Child & Adolescent Psychiatry Clinics of North America, Jan. '05**

(This article has been lightly edited by David Dubin, MD to make it more accessible to a general public.)

D. Corydon Hammond, PhD, ABEN/ECNS  
Physical Medicine and Rehabilitation, University of Utah School of Medicine,

Compelling evidence exists for a neurophysiologic basis for obsessive-compulsive disorder (OCD). There is also strong research evidence also indicates that there are functional brain abnormalities associated with anxiety and panic disorder [28–30] and post-traumatic stress disorder (PTSD) [31].

There is a strong reliance in psychiatry on the use of medication for the treatment of depression and anxiety, although some evidence currently suggests that medication may not be as effective in treating these conditions as has often been believed [44–48].

Similarly, Greist [49] estimated the degree of symptomatic improvement in OCD from treatment with serotonin drugs to only be 30%. Goodman et al [44] similarly found that symptom amelioration in OCD treatment with serotonin uptake inhibitors is approximately 35% on average and that only 50% of patients experience this partial improvement.

In light of this brief review and the fact that an increasing number of patients and parents seem interested in non-medication treatment alternatives that still address the underlying biologic factors associated with depression, anxiety, and obsessive compulsive disorder (OCD), it would be desirable to find a treatment that also would help address the biologic aspects of mental health disorders. Neurofeedback holds promise for offering such an alternative.

### **What is neurofeedback?**

Neurofeedback is EEG biofeedback or brain wave training. Nothing intrusive is introduced into the brain. The sensors simply measure the ongoing brain wave activity.

Ordinarily we are unable to reliably influence our brain wave activity because we lack awareness of it. When we are able to see representations of our brain wave activity on a computer screen a few thousandths of a second after it occurs, however, it allows us to modify our brain wave patterns through operant

conditioning.

The patient is placed in front of a computer screen. The computer display may be as complex as a computer/video game type of display. It also may be as simple as two bar graphs, one representing slow and inefficient brain wave activity and the other representing efficient, beta brain wave activity. The patient concentrates on the screen. When the inappropriate activity decreases slightly and the appropriate activity increases slightly, a pleasant tone might be heard.

At first, changes in brain wave activity are transient. As sessions are repeated, enduring changes are gradually seen.

EEG biofeedback (neurofeedback) has been found to be effective in modifying brain function and producing significant improvements in clinical symptoms in children, adolescents, and adults who have several different biologic brain disorders.

These conditions include epilepsy, attention deficit disorder and attention deficit hyperactivity disorder (ADHD), and learning disabilities and have included up to 10-year follow-ups of patients [57].

## **Neurofeedback for anxiety**

A review of the literature on the neurofeedback treatment of anxiety disorders by Moore [58] identify eight studies of generalized anxiety disorder.

The best studies of neurofeedback with anxiety were three outcome studies [59] with phobic (test) anxiety. In each study, the group that received alpha EEG enhancement training demonstrated significant reductions in test anxiety. In comparison, the untreated control group and the relaxation training group experienced no significant reduction.

In another study, with alpha training the anxiety scores dropped significantly compared with a non-treatment group. Moore [58] concluded in his review that a placebo effect was present in these neurofeedback studies but that alpha and theta enhancement training provided additional effects beyond placebo and are effective treatments for anxiety disorders.

Passini et al [70] used 10 hours of alpha neurofeedback training, comparing 25 anxious patients (23 of whom were alcoholics) with a control group of 25 anxious patients (22 of whom were also alcoholics), most of whom were seeking treatment at a Veterans Administration hospital brief treatment unit. The alpha neurofeedback training produced significant changes in state and trait anxiety compared with controls.

An 18-month follow-up of those patients was published, with virtually identical

results of lower anxiety still found, which validated that the anxiety changes from alpha neurofeedback were enduring [71].

**Two neurofeedback outcome studies have focused on chronic PTSD.** In a randomized, controlled group study [73], 30 30-minute sessions of alpha-theta EEG biofeedback training were added to the traditional Veterans Administration hospital treatment that was provided to a group of 15 Vietnam combat veterans with PTSD. The study compared them after treatment and at follow-up with a contrast group of 14 veterans who only received traditional treatment.

In addition to the posttreatment testing, on a monthly basis, patients and informers were contacted for a full 30-month follow-up period to determine if there had been PTSD symptoms (eg, flashbacks, nightmares, anxiety attacks, depression).

At follow-up, all 14 traditional treatment patients had experienced relapse, whereas only 3 of 15 neurofeedback training patients had experienced relapse.

All 14 patients who were treated with neurofeedback had decreased their medication requirements at follow-up, whereas in contrast, only 1 traditional treatment patient had decreased medication needs, 2 reported no change, and 10 required more medications.

Neurofeedback training patients improved significantly on all ten MMPI clinical scales—in many instances dramatically—but there were no significant improvements on any scales in the traditional treatment group.

In another Veterans Administration hospital uncontrolled study [74], 20 Vietnam veterans with chronic PTSD, all with alcohol abuse, were randomly selected. All patients showed frequent (eg, two to three times per week) episodes of PTSD and had been hospitalized for PTSD an average of five times.

They were treated with 30 30-minute sessions of alpha-theta neurofeedback training. Follow-up interviews occurred with the patients and their wives or family members on a monthly basis for 26 months. In that time, only 4 of the 20 patients reported a few (one to three) instances of recurrence of nightmares or flashbacks, and the other 16 patients had no recurrence of PTSD symptoms.

## **Neurofeedback for depression**

Although reports to date on the application of neurofeedback to depression only represent uncontrolled case reports, they provide encouragement that neurofeedback may hold potential for treating mildly to severely depressed patients and that unlike medication, it may enduringly modify the functional brain abnormality associated with biologic predisposition to depression.

## **Clinical experience and further case examples**

Based on clinical experience with more than 25 patients with dysthymia, in which most of them have been followed for between 6 and 24 months, neurofeedback has seemed to be successful in producing significant and enduring change in approximately 80% of the patients. There have been no published research or clinical reports on the use of neurofeedback in a pediatric depression sample. Because the biologic marker of a frontal alpha asymmetry has been found in multiple studies with children and infants [38–41] of depressed mothers, and because there is abundant evidence that children respond to neurofeedback training for other conditions, it is reasonable to expect that this approach would be beneficial with depressed children.

There are widespread clinical reports of improvements in mood among children treated with neurofeedback for ADHD, which further supports the expectation that neurofeedback may be effective with childhood depression. There also are reports of improvements in bipolar disorder.

Neurofeedback seems to involve minimal risk of side effects or adverse reactions [84], and it is less invasive than antidepressant medication or transcranial magnetic stimulation.

## **Anxiety and insomnia**

In most cases, anxiety and insomnia are readily treated with neurofeedback [58,59,85–88]. One of the first improvements that parents often notice is that the child falls asleep more easily and remains asleep. With anxiety patients, neurofeedback training often is done eyes closed while listening to auditory feedback, and in a sense it resembles high-technologic meditation training.

As a case example, a patient was referred by a physician who was a headache specialist, indicating that everything that could be done with medication seemed to have been done. The patient had a lengthy history of several migraines weekly, which had progressed to daily migraines. She had been given a self-hypnosis tape to use for anxiety management, but she complained that her mind was so busy that she was unable to obtain much relaxation from the tape. After 20 30-minute sessions of inhibiting fast beta and reinforcing alpha activity in the parietal area, she was off all her prescription medications. She sensed a migraine trying to begin approximately twice weekly but would take over-the-counter medication and could use the self-hypnosis tape successfully to abort the headache. She felt more relaxed in general and reported no longer feeling compelled to do two things at once.

## Summary

As reviewed in other articles, the neuroscience technology known as EEG biofeedback (or neurofeedback) has considerable research support in areas such as uncontrolled epilepsy and attention deficit disorder and ADHD. In evaluating the studies in the overall broad area of the neurofeedback treatment of anxiety disorders, EEG biofeedback qualifies for the evidence-based designation of being an efficacious treatment [62]. When separate anxiety disorders are individually evaluated, the areas of phobic anxiety, generalized anxiety, and PTSD each qualify for designation as being a probably efficacious treatment.

Currently there are only reports of cases and series of cases on the treatment of depression and OCD and no published reports thus far on treatment of bipolar disorder. Despite the lengthy follow-ups and use of objective measures, neurofeedback treatment for depression and OCD is not yet empirically supported. EEG biofeedback is an exciting, cutting-edge technology that offers an additional treatment alternative for modifying dysfunctional, biologic brain patterns that are associated with various psychiatric conditions.

## References

- [1] Baxter L, Phelps M, Mazziotta J. Local cerebral glucose metabolic rates in obsessive-compulsive disorder: a comparison with rates in unipolar depression and in normal controls. *Arch Gen Psychiatry* 1988;44:211 – 8.
- [2] Baxter L, Phelps M, Mazziotta J, Guze BH, Schwartz JM, Selin C. Local cerebral glucose metabolic rates in obsessive-compulsive disorder. *Arch Gen Psychiatry* 1987;44:211 – 8.
- [3] Benkelfat C, Phelps M, Mazziotta J, Guze BH, Schwartz JM, Selin RM. Local cerebral glucose metabolic rates in obsessive-compulsive disorder patients treated with clomipramine. *Arch Gen Psychiatry* 1990;147:846 – 8.
- [4] Harris GJ, Pearlson GD, Hoehn-Saric R. Single photon emission computer tomography in obsessive-compulsive disorder. *Arch Gen Psychiatry* 1993;50(6):498 – 501.
- [5] Machlin SR, Harris GJ, Pearlson GD. Elevated medial-frontal cerebral blood flow in obsessive-compulsive patients: a SPECT study. *Am J Psychiatry* 1991;148:1240 – 2.
- [6] Nordahl TE, Benkelfat C, Semple WE, Gross M, King AC, Cohen RM. Cerebral glucose metabolic rates in obsessive-compulsive disorder. *Neuropsychopharma* 1989;2:23 – 8.
- [7] Perani D, Colombo C, Bressi S, Bonfanti A, Grassi F, Scarone S, et al. 18[F]FDG PET study in obsessive-compulsive disorder: a clinical/metabolic correlation study after treatment. *Br J Psychiatry* 1995;156:244 – 50.
- [8] Piacentini J, Bergman RL. Obsessive-compulsive disorder in children. *Psychiatr Clin N Am* 2000;23(3):519 – 33.
- [9] Rauch SL, Whalen PJ, Dougherty D, Jenike MA. Neurobiologic models of obsessive-

- compulsive disorder. In: Jenike MA, Baer WE, Minichiello WE, editors. Obsessive-compulsive disorders: practical management. St. Louis: Mosby; 1998. p. 222 – 53.
- [10] Rubin RT, Villaneuva-Meyer J, Anath J. Regional <sup>133</sup>Xe cerebral blood flow and cerebral <sup>99m</sup>Tc-HMPAO uptake in unmedicated obsessive-compulsive disorder patients and matched normal control subjects: determination by high-resolution single-photon emission computed tomography. *Arch Gen Psychiatry* 1992;49:695 – 702.
- [11] Sawle GV, Hymas NF, Lees AJ. Obsessive slowness: functional studies with positron emission tomography. *Brain* 1991;114:2191 – 202.
- D.C. Hammond / *Child Adolesc Psychiatric Clin N Am* 14 (2005) 105–123 119
- [12] Saxena S, Brody AL, Schwartz JM, Baxter LR. Neuroimaging and frontal-subcortical circuitry in obsessive-compulsive disorder. *Br J Psychiatry* 1998;35:26 – 38.
- [13] Swedo SE, Schapiro MG, Grady CL. Cerebral glucose metabolism in childhood onset obsessive-compulsive disorder. *Arch Gen Psychiatry* 1989;46:518 – 23.
- [14] Szeszko PR, Robinson D, Alvir JM, Bilder RM, Lencz T, Ashtari M, et al. Orbital frontal and amygdala volume reductions in obsessive-compulsive disorder. *Arch Gen Psychiatry* 1999; 56(10):913 – 9.
- [15] Kuskowski MA, Malone SM, Kim SW, Dysken MW, Okaya AJ, Christensen KJ. Quantitative EEG in obsessive-compulsive disorder. *Biol Psychiatry* 1993;33:423 – 30.
- [16] Leocani L, Locatelli M, Bellodi L, Fornara C, Henin M, Magnani G, et al. Abnormal pattern of cortical activation associated with voluntary movement in obsessive-compulsive disorder: an EEG study. *Am J Psychiatry* 2001;158(1):140 – 2.
- [17] Mas F, Prichep LS, John ER, et al. Neurometric quantitative electroencephalogram subtyping of obsessive compulsive disorders. In: Mauer K, editor. *Imaging of the brain in psychiatry and related fields*. Berlin: Springer-Verlag; 1993. p. 277 – 80.
- [18] Perros R, Young E, Ritson J, Price G, Mann P. Power spectral EEG analysis and EEG variability in obsessive-compulsive disorder. *Brain Topogr* 1992;4(3):187 – 92.
- [19] Prichep LS, Mas F, John ER, et al. Neurometric subtyping of obsessive compulsive disorders in psychiatry: a world perspective. In: Stefanis CN, Rabavilas AD, Soldatos CR, editors. *Proceedings of the VIII World Congress of Psychiatry*. Athens, October 12–19, 1989. New York: Elsevier Science; p. 557–62.
- [20] Prichep LS, Mas F, Hollander E, Liebowitz M, John ER, Almas M, et al. Quantitative electroencephalography (QEEG) subtyping of obsessive compulsive disorder. *Psychiatr Res* 1993;50(1):25 – 32.
- [21] Silverman JS, Loychik SG. Brain-mapping abnormalities in a family with three obsessive-compulsive children. *J Neuropsychiatr Clin Neurosci* 1990;2:319 – 22.

- [22] Simpson HB, Tenke CE, Towey JB, Liebowitz MR, Bruder GE. Symptom provocation alters behavioral ratings and brain electrical activity in obsessive-compulsive disorder: a preliminary study. *Psychiatr Res* 2000;95(2):149 – 55.
- [23] Gehring WJ, Himle J, Nisenson LG. Action-monitoring dysfunction in obsessive-compulsive disorder. *Psychol Sci* 2000;11:1 – 6.
- [24] Hajcak G, Simons RF. Error-related brain activity in obsessive-compulsive undergraduates. *Psychiatry Res* 2002;110:63 – 72.
- [25] Malloy P, Rasmussen S, Braden W, Haier RJ. Topographic evoked potential mapping in obsessive-compulsive disorders: evidence of frontal lobe dysfunction. *Psychiatry Res* 1989; 28(1):63 – 71.
- [26] Posner MI, Rothbart MK. Attention, self-regulation and consciousness. *Philos Trans R Soc Lond B Biol Sci* 1998;353:1 – 13.
- [27] Ursu S, van Veen V, Siegle G, MacDonald A, Stenger A, Carter C. Executive control and self- evaluation in obsessive-compulsive disorder: an event-related fMRI study. Presented at the Cognitive Neuroscience Society Meeting. New York, March 2001.
- [28] Heller W, Etienne MA, Miller GA. Patterns of perceptual asymmetry in depression and anxiety: implications for neuropsychological models of emotion and psychopathology. *J Abnorm Psychol* 1995;104:327 – 33.
- [29] Heller W, Nitschke JB, Etienne MA, Miller GA. Patterns of regional brain activity differentiate types of anxiety. *J Abnorm Psychol* 1997;106(3):376 – 85.
- [30] Wiedemann G, Pauli P, Dengler W, Lutzenberger W, Birbaumer N, Buckkremer G. Frontal brain asymmetry as a biological substrate of emotions in patients with panic disorders. *Arch Gen Psychiatry* 1999;56:78 – 84.
- [31] Brown D, Schefflin AW, Hammond DC. *Memory, trauma treatment, and the law*. New York: WW Norton; 1998.
- [32] Davidson RJ. Affective style and affective disorders: perspectives from affective neuroscience. *Cognition and Emotion* 1998;12:307 – 30.
- [33] Davidson RJ. Emotion and affective style: hemispheric substrates. *Psychol Sci* 1992;3:39 – 43.
- [34] Davidson RJ. Cerebral asymmetry, emotion and affective style. In: Davidson RJ, Hugdahl K, editors. *Brain asymmetry*. Boston: MIT Press; 1995. p. 361 – 87.
- [35] Baehr E, Rosenfeld JP, Baehr R. The clinical use of an alpha asymmetry protocol in the neurofeedback treatment of depression: two case studies. *J Neurotherapy* 1997;2(3):10 – 23.
- [36] Rosenfeld JP, Cha G, Blair T, Gotlib I. Operant biofeedback control of left-right frontal alpha power differences. *Biofeedback Self Regul* 1995;20:241 – 58.
- [37] Henriques JB, Davidson RJ. Left frontal hypoactivation in depression. *J Abnorm Psychol* 1991; 100:534 – 45.
- [38] Dawson G, Grofer Klinger L, Panagiotides H, Hill D, Spieker S. Frontal lobe activity and affective behavior of infants of mothers with depressed symptoms. *Child Dev* 1992;63:725 – 37.
- [39] Dawson G, Grofer Klinger L, Panagiotides H, Spieker S, Frey K. Infants of mothers with de- pressed symptoms: electroencephalographic and behavioral findings related to attachment status. *Dev Psychopathol* 1992;4:67 – 80.
- [40] Field T, Fox N, Pickens J, Nawrocki R. Relative right frontal EEG activation

- in 3- to 6-month- old infants of “depressed” mothers. *Dev Psychopathol* 1995;26:7 – 14.
- [41] Jones NA, Field T, Fox NA, Lundy B, Davalos M. EEG activation in 1-month-old infants of depressed mothers. *Dev Psychopathol* 1997;9:491 – 505.
- [42] Henriques JB, Davidson RJ. Regional brain electrical asymmetries discriminate between previously depressed and health control subject. *J Abnorm Psychol* 1990;99:22 – 31.
- [43] Davidson RJ. Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology* 1998;35:607 – 14.
- [44] Goodman WK, McDougle CJ, Price LH. Pharmacotherapy of obsessive compulsive disorder. *J Clin Psychiatry* 1992;53(Suppl):29 – 37.
- [45] Goodman WK, Price LH, Rasmussen SA, Mazure C, Fleischmann RL, Hill CL, et al. The Yale- Brown obsessive ompulsive scale. I. Development, use, and reliability. *Arch Gen Psychiatry* 1989;46:1006 – 11.
- [46] Goodman WK, Price LH, Rasmussen SA, Mazure C, Delgado P, Heninger GR, et al. The Yale- Brown obsessive compulsive scale. II. Validity. *Arch Gen Psychiatry* 1989;46:1012 – 6.
- [47] Jenike MA, Baer L, Ballantine T, Martuza RL, Tynes S, Giriunas I, et al. Cingulotomy for refractory obsessive-compulsive disorder: a long-term follow-up of 33 patients. *Arch Gen Psychiatry* 1991;48:548 – 55.
- [48] Hughes JR, John ER. Conventional and quantitative electroencephalography in psychiatry. *J Neuropsychiatr Clin Neurosci* 1999;11(2):190 – 208.
- [49] Greist JH. Treatment of obsessive compulsive disorder: psychotherapies, drugs, and other somatic treatment. *J Clin Psychiatr* 1990;51(8):44 – 50.
- [50] Ackerman DL, Greenland S. Multivariate meta-analysis of controlled drug studies for obsessive- compulsive disorder. *J Clin Psychopharmacol* 2002;22(3):309 – 17.
- [51] Rauch SL. Neuroimaging research and the neurobiology of obsessive-compulsive disorder: where do we go from here? *Biol Psychiatry* 2000;47:168 – 70.
- [52] DeRubeis RJ, Gelfand LA, Tang TZ, Simons AD. Medications versus cognitive behavior therapy for severely depressed outpatients: mega-analysis of four randomized comparisons. *Am J Psychiatry* 1999;156:1007 – 13.
- [53] Antonuccio DO, Danton WG, DeNelsky G. Psychotherapy vs. medication for depression: challenging the conventional wisdom with data. *Professional Psychology: Research and Practice* 1995;26:574 – 85.
- [54] Hollon SD, Shelton RC, Loosen PT. Cognitive therapy and pharmacotherapy for depression. *J Consult Clin Psychol* 1991;59:88 – 99.
- [55] Foa EB, Franklin ME. Obsessive-compulsive disorder. In: Barlow DH, editor. *Clinical handbook of psychological disorders*. 3rd edition. New York: Guilford Press; 2001. p. 209 – 63.
- [56] Whitsett SF, Lubar JF, Holder GS, et al. A double-blind investigation of the relationship between seizure activity and the sleep EEG following EEG biofeedback training. *Biofeedback Self Regul* 1982;7:183 – 209.
- [57] Lubar JF. Neurofeedback for the management of attention

- deficit/hyperactivity disorders. In: Schwartz MS, editor. Biofeedback: a practitioner's guide. New York: Guilford Press; 1995. p. 493 – 522.
- [58] Moore NC. A review of EEG biofeedback treatment of anxiety disorders. *Clin Electroencephalogr* 2000;31(1):1 – 6.
- [59] Garrett BL, Silver MP. The use of EMG and alpha biofeedback to relieve test anxiety in college students. In: Wickramasekera I, editor. Biofeedback, behavior therapy, and hypnosis. Chicago: Nelson-Hall; 1976.
- [60] Chambless DL, Baker MJ, Baucom DH, Beutler LE, Calhoun KS, Crits-Christoph P, et al. Update on empirically validated therapies. *Clin Psychol* 1998;51(1):3 – 16.
- [61] Chambless D, Hollon SD. Defining empirically supported therapies. *J Consult Clin Psychol* 1998;66:7 – 18.
- [62] La Vaque TJ, Hammond DC, Trudeau D, Monastera V, Perry J, Lehrer P. Template for developing guidelines for the evaluation of the clinical efficacy of psychophysiological interventions. *J Neurotherapy* 2002;6(4):11 – 23.
- [63] Benson K, Hartz AJ. A comparison of observational studies and randomized, controlled trials. *N Engl J Med* 2000;342(25):1878 – 86.
- [64] Concato J, Shah N, Horwitz RJ. Randomized, controlled trials, observational studies, and the hierarchy of research designs. *N Engl J Med* 2000;342(25):1887 – 92.
- [65] Britton A, McPherson K, KcKee M, Sanderson C, Black N, Bain C. Choosing between randomized and non-randomized studies: a systematic review. *Health Technol Assess* 1998; 2(13):1 – 124.
- [66] Lurie P, Wolfe S. Unethical trials of interventions to reduce perinatal transmission of the human immunodeficiency virus in developing countries. *N Engl J Med* 1997;337(12):853 – 6.
- [67] Rothman DJ. Ethical and social issues in the development of new drugs and vaccines. *Bull N Y Acad Med* 1987;63(6):557 – 68.
- [68] La Vaque TJ, Rossiter T. The ethical use of placebo controls in clinical research: the Declaration of Helsinki. *Appl Psychophysiol Biofeedback* 2001;26(1):23 – 37.
- [69] Linden M, Habib T, Radojevic V. A controlled study of the effects of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. *Biofeedback Self Regul* 1996;21(1):35 – 49.
- [70] Passini FT, Watson CG, Dehnel L, Herder J, Watkins B. Alpha wave biofeedback training therapy in alcoholics. *J Clin Psychol* 1977;33(1):292 – 9.
- [71] Watson CG, Herder J, Passini FT. Alpha biofeedback therapy in alcoholics: an 18-month follow-up. *J Clin Psychol* 1978;34(2):765 – 9.
- [72] Egner T, Gruzelier JH. Ecological validity of neurofeedback: modulation of slow wave EEG enhances musical performance. *Neuroreport* 2003;14(9):1221 – 4.
- [73] Peniston EG, Kulkosky PJ. Alpha-theta brainwave neuro-feedback therapy for Vietnam veterans with combat-related post-traumatic stress disorder. *Medical Psychotherapy* 1991;4:47 – 60.

- [74] Peniston EG, Marrinan DA, Deming WA, Kulkosky PJ. EEG alpha-theta synchronization in Vietnam theater veterans with combat-related post-traumatic stress disorder and alcohol abuse. *Advances in Medical Psychotherapy* 1993;6:37 – 50.
- [75] Hammond DC. QEEG-guided neurofeedback in the treatment of obsessive compulsive disorder. *Journal of Neurotherapy* 2003;7(2):25 – 52.
- [76] Hammond DC. Treatment of obsessional OCD with neurofeedback. *Biofeedback* 2004;32:9 – 12.
- [77] Rosenfeld JP. EEG biofeedback of frontal alpha asymmetry in affective disorders. *Biofeedback* 1997;25(1):8 – 25.
- [78] Baehr E, Rosenfeld JP, Baehr R. Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of mood disorders: follow-up study one to five years post therapy. *Journal of Neurotherapy* 2001;4(4):11 – 8.
- [79] Allen JJ, Iacono WG, Depue RA, Arbisi P. Regional electroencephalographic asymmetries in bipolar seasonal affective disorder before and after exposure to bright light. *Biol Psychiatry* 1993;33:642 – 6. D.C. Hammond / *Child Adolesc Psychiatric Clin N Am* 14 (2005) 105–123 122
- [80] Gotlib IH, Ranganath C, Rosenfeld JP. Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion* 1999;12:449 – 78.
- [81] 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 – 73.
- [82] Hammond DC. Neurofeedback treatment of depression with the Roshi. *Journal of Neurotherapy* 2000;4(2):45 – 56.
- [83] Hammond DC. Neurofeedback treatment of depression and anxiety. *J Adult Dev*, in press.
- [84] Hammond DC, Stockdale S, Hoffman D, Ayers ME, Nash J. Adverse reactions and potential iatrogenic effects in neurofeedback training. *Journal of Neurotherapy* 2001;4(4):57 – 69.
- [85] Hardt JV, Kamiya J. Anxiety change through electroencephalographic alpha feedback seen only in high anxiety subjects. *Science* 1978;201:79 – 81.
- [86] Feinstein B, Sterman MB, MacDonald LR. Effects of sensorimotor rhythm training on sleep. *Sleep Research* 1974;3:134.
- [87] Sterman MB. Effects of sensorimotor EEG feedback on sleep and clinical manifestations of epilepsy. In: Beatty J, Legewie H, editors. *Biofeedback and behavior*. New York: Plenum Press; 1977. p. 167 – 200.
- [88] Sterman MB, Howe RD, Macdonald LR. Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. *Science* 1970;167:1146 – 8.
- [89] Duffy FH. The state of EEG biofeedback therapy (EEG operant conditioning) in 2000: an editor's opinion [editorial]. *Clin Electroencephalogr* 2000;31(1):v – viii. D.C. Hammond / *Child Adolesc Psychiatric Clin N Am* 14 (2005) 105–123 123