#### A Review of Research on Depression and Neurofeedback Training

The following entry on depression and neurofeedback training is excerpted from an article published in the peer reviewed journal **Child and Adolescent Psychiatric Clinics of North America** 14 (2005) pp 105-123 The article is written for an audience of child and adolescent clinicians, but reviews the general research literature, usually conducted on adults, for neurofeedback and depression.

# Neurofeedback with Anxiety and Affective Disorders

D. Corydon Hammond, PhD, ABEN/ECNS

Physical Medicine and Rehabilitation, University of Utah School of Medicine, PM&R 30 No 1900 East, Salt Lake City, UT 84132-2119, USA

### (Excerpted section on Depression specifically)

In relation to the research reviewed earlier on the presence of a frontal alpha asymmetry in depression, Rosenfeld [77] developed a neurofeedback protocol for modifying this asymmetry. This ALAY protocol (which stands for alpha asymmetry; F4 \_ F3/F3 + F4, with a reference electrode at Cz) has been used in case studies [35,36,78] with encouraging preliminary results, but no controlled research has been conducted. Baehr et al [78] did 1- to 5-year follow-ups on patients treated with the ALAY protocol and documented that the changes in depression were enduring and that the frontal alpha asymmetry not only had changed at the end of treatment but that this physiologic asymmetry continued to be reversed on long-term follow-ups. This is of particular relevance because several studies [42,79–81] have found that after pharmacologic treatment that produced a remission of depression, the frontal alpha asymmetry remained unchanged, which suggests that patients in drug treatment continue to have a biologic vulnerability to future depression.

A different protocol for modifying the frontal alpha asymmetry also was developed in association with a successful case report with an 8.5-month followup [82]. In this protocol electrodes are placed at Fp1 (on the left forehead) and F3 (approximately 2.5–3 inches straight above Fp1). During the training, slow brain wave activity is inhibited in the alpha and theta frequency bands during reinforcement of 15- to 18-Hz beta for the first 20 to 22 minutes of each training session, after which the reinforcement frequency band is decreased to 12 to 15 Hz for the final 8 to 10 minutes of each session. A 2-year follow-up of the initial case found that the depression remained in remission.

This second protocol has continued to be used clinically in the treatment of depression during the past 5 years, and there is a new report with a sample of nine consecutive patients who were treated with it [83]. All the patients in this series were relatively medication resistant and had been diagnosed with dysthymic

disorder. They were all administered the MMPI and screened with the ALAY 114 D.C. Hammond / Child Adolesc Psychiatric Clin N Am 14 (2005) 105–123 protocol to verify the presence of the frontal alpha asymmetry associated with a biologic predisposition to depression. This screening takes approximately 15 minutes, and researchers have found that percentage scores of more than 60 indicate that there is no predisposition to depression, whereas scores of 58 or less indicate the presence of a predisposition [80]. The mean percentage score in the recent sample was 40.1, and their mean on the MMPI Depression scale (scale 2) was 93.8 T-scores. From the beginning, one patient seemed to have questionable motivation and dropped out after five sessions. The other eight patients received an average of 10.4 hours of training (20.8 30-minute sessions). No other psychotherapy was provided. After treatment, there was a mean decrease in the depression scale of 28.8 T-scores.

Improvement was categorized using the following criteria. Less than 60 Tscores on the depression scale was considered as representing normal, 60 to 70 Tscores represented mild depression, 71 to 80 T-scores represented moderate depression, 81 to 90 T-scores represented serious depression, and 91 T-scores and above represented severe depression. According to these criteria, overall this was a severely depressed patient sample. One patient was judged to have improved from being severely depressed to being normal, and two improved from being seriously depressed to normal. Three of the patients were judged to have improved from a severe to a mild level of depression, and one improved from moderately depressed to mildly depressed. In one case, a severely depressed individual only manifested mild improvement. He had lost his wife to cancer a year earlier, and this loss seemed to need further attention. He was referred for more traditional psychotherapy. All the patients had been treated with several antidepressant medications without substantive effect, and most of the patients were on medication at the beginning of neurofeedback training but not at the conclusion. The average length of individual follow-up of the eight patients was 1 year (range, 4 months to 2 years), at which time improvements had been maintained. Classifying the patient who only mildly improved as a failure, 87.5% of the cases improved, and if the drop-out is included as a failure, then 77.8% of the case series made significant improvements.

Patients in many of the published medication studies are moderately depressed, whereas in this case series, seven of the eight patients were classified as seriously to severely depressed, and only one patient was moderately depressed. The cases in the ALAY protocol studies [83] were only in the mild range of depression, with scores in the 62 to 64 T-score range on the MMPI, which also is reflected in their ALAY scores, which averaged 51.3, whereas the case series reported by Hammond [83] had a mean ALAY score of 40.1. Although reports to date on the application of neurofeedback to depression only represent uncontrolled case reports that are not sufficiently rigorous to receive one of the levels of evidence-based support, 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 a biologic predisposition to depression. Controlled research seems warranted.

## Clinical experience and further case examples Depression

A case example illustrates the use of this second neurofeedback protocol with depression. Dan was an engineer in his 30s. He had originally entered treatment for a circumscribed complaint of fear of public speaking, which had been successfully treated in five sessions with self-hypnosis training. A year later he returned and indicated that he had experienced depression for many years but that it had been getting worse. His ALAY score of 36.1% indicated an extreme frontal alpha asymmetry, and his MMPI depression scale of 92 T-scores confirmed his severe depression. After informed consent, neurofeedback was started with the depression protocol. After three sessions he said that despite having had a difficult week at work, "I have been feeling a lot better. It's hard to believe that it's working this quickly." He explained that he had been skeptical about the possibility of neurofeedback being successful and was particularly surprised that he already could feel a difference. In clinical experience with this protocol, most patients can begin to perceive a difference in their depression level after three to six 30-minute training sessions. Usually by 10 to 12 30-minute sessions they feel significant improvement, and by 20 to 22 sessions treatment is completed. Dan indicated after five sessions that he was still feeling depressed but that it was improving. After seven sessions he reported sleeping better, and after eight sessions he said that several people at work had commented on seeing a difference in him and had said, "We were worried about you there for a while." He explained that previously he had attributed his depression to his work situation but that his work had not changed and his depression was much improved. He continued to improve steadily. His total treatment consisted of 19 30-minute neurofeedback training sessions. Fig. 3 displays his before and after MMPI changes. His depression (scale 2) had decreased from a severe level (92 Tscores) to a mild, perhaps subclinical level (63 T-scores). The rest of his MMPI profile reflects changes that have been found in most cases after using this treatment protocol. His anxiety, obsessional rumination, and feelings of inferiority and inadequacy (as reflected in scales 7 and A) decreased, whereas ego strength (Es scale) increased. His withdrawal and feelings of alienation from people (scale 8) decreased and he changed from being moderately introverted and quiet (scale 0) to being on the mean between introversion and extroversion. The MMPI has proved to be a much better outcome measure than using a depression scale alone because it has illuminated the many other dimensions on which change has occurred. On the MMPI, a decrease in withdrawal and introversion (scales 8 and 0) commonly accompany the decline in depression, which would be anticipated because an area of the brain is being activated that is also associated with approach motivation. Dan's changes were maintained at 6.5-month followup, at which time he took a new job in another state.

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 anecdotal 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.

#### 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 obsessivecompulsive disorder. In: Jenike MA, Baer WE, Minichiello WE, editors. Obsessive-compulsive disorders: practical management. St. Louis7 Mosby; 1998. p. 222–53.
- [10] Rubin RT, Villaneuva-Meyer J, Anath J. Regional 133Xe cerebral blood flow and cerebral 99m-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. Berlin7 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 selfevaluation 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, Scheffin AW, Hammond DC. Memory, trauma treatment, and the law. New York7 WW Norton; 1998.
- [32] Davidson RJ. Affective style and affective disorders: perspectives from affective neuroscience. Cognition and Emotion 1998;12:307–30.
- 120 D.C. Hammond / Child Adolesc Psychiatric Clin N Am 14 (2005) 105-123
- [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. Boston7 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 depressed 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-monthold

- 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, Pricee 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 York7 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.
- D.C. Hammond / Child Adolesc Psychiatric Clin N Am 14 (2005) 105-123 121
- [57] Lubar JF. Neurofeedback for the management of attention deficit/hyperactivity disorders. In: Schwartz MS, editor. Biofeedback: a practitioner's guide. New York7 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. Chicago7 Nelson-Hall: 1976
- [60] Chambless DL, Baker MJ, Baucaom 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, Monastra 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 RI. 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 followup. 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.
- 122 D.C. Hammond / Child Adolesc Psychiatric Clin N Am 14 (2005) 105-123
- [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 York7 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