NEUROFEEDBACK FOR THE TREATMENT OF DEPRESSION

CURRENT STATUS OF THEORETICAL ISSUES AND CLINICAL RESEARCH

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INTRODUCTION

Interest in the relationship between neural functions and emotions has focused particularly on comparisons of activation in the left and right dorsolateral frontal cortices. Beginning with the investigations of Davidson and colleagues (1985, 1989, 1990a, 1992) the alpha EEG frequency has been used as a measure of hypoarousal, and there has been general agreement among researchers that mood is more positive when the right dorsolateral prefrontal cortex is more hypoactive than the left dorsolateral prefrontal cortex. A practical application of this theoretical approach was developed when Dr. J. Peter Rosenfeld and colleagues designed an experiment in which they demonstrated that cortical asymmetry (A-score) could be modified in normal subjects by using a simple operant conditioning program (Rosenfeld, Cha, Blair, & Gotlieb, 1995). Their findings, in which 9 of 13 subjects were successfully trained, were replicated by Allen, Harmon-Jones & Cavendar (2001). Encouraged by these results, neurofeedback protocols were developed to try with a small group of clinical patients who were suffering from various mood disorders (Baehr & Baehr, 1997a, Baehr,

Rosenfeld, Baehr. 1997b), (Hammond, 2000, 2005a, b).

In this chapter we will review the extensive theoretical literature that has been published since Davidson=s original investigations. We will review the results of the Baehr group first study and present follow-up data from one to ten years post therapy. In addition, we will report on studies (Baehr, Rosenfeld, Baehr & Earnest, 1999) utilizing the asymmetry protocol to study shifts in mood (Baehr, 2000), and studies of asymmetry changes in women who suffer from premenstrual dysphoric disorder (Baehr, Rosenfeld, Miller, & Baehr, 2004). We will also report on other asymmetry protocols designed to alleviate depression (Hammond, 2000, 2005a, b).

A Review of the Literature

Frontal EEG Asymmetry and Emotions:

The development of a theory regarding cortical frontal asymmetry and emotions, proposed by Davidson and colleagues (1985, 1989, 1990a, 1992) evoked the interest of scientific community worldwide. An approach –avoidance model, developed by Davidson (1990b,1993, 1998a,b, 1999 a,b) was based on studies demonstrating that hyperarousal in the left frontal cortex was associated approach behavior and positive mood, while hyperarousal in the right prefrontal cortex was associated with avoidance behavior and negative mood. (Sutton & Davidson, 1997) An advantage of this model was that it provided a way of assessing both normal affective behavior and also pathological states such as depression.

A scale designed to measure behavioral inhibition and behavioral activation, (Carver et al., 1994) provided an objective way to test Davidson's theoretical approach. While data supported a significant relationship of left frontal activation and the behavioral activation scale (BAS), no statistically significant relationship between right frontal activation and the behavioral

inhibition scale (BIS) was found (Harmon-Jones & Allen, 1997, Coan & Allen, 2003). The possibility that hypoarousal in the right frontal cortex may be related to the BIS was not considered in this study. Davidson (2004) suggests that there is a relationship between the arousal of the prefrontal cortex (PFC) and affect. He states that arousal in the PFC is related to its ability to inhibit its input from the sub-cortical centers. In a complicated circuitry involving the amygdala, the hippocampus, the cingulate and the left and right dorsolateral cortex and other structures, he explains that the influx of negative stimuli can be modulated in this way.

In 2003 and 2004 Coan and Allen reviewed over 80 articles published since Davidson's original work, basically supporting the findings that there is a relationship between resting EEG asymmetry in the frontal cortex and in emotions or emotion-related constructs. They reexamined the many existing studies for the purpose of investigating state and trait features of frontal cortical asymmetry, and then, in the later study, looked at activation in the right frontal cortex in terms of influencing behavior. While many studies supported Davidson's results (e.g., Allen, Urry, Hitt, Coan, 2004a; Harmon –Jones 2000; McFarland, Shankman, Tenke, Bruder, Klein, 2006; Pizzagalli, Sherwood, Henriques, Davidson, 2005; Sutton, Davidson, 1997; Tomarken, Davidson, 1994), not all researchers were able to replicate them (Allen et al., 2004a, Hagemann, Naumann, Becker, Maier, Bartussek, 1998; Hagemann, Naumann, Lurken, Becker, Maier, Bartussek, 1999; Hagemann, Naumann, Thayer, 2001; Debener, Beauducel, Nessler, Brocke, Heilemann, Kayser, 2000; Reid, Duke, Allen. 1998; Allen, Coan, Nazarian, 2004 b; Davidson, 1998; Hagemann, 2004; Coan and Allen, 2004). The consensus is that a number of factors may contribute to the discrepancies, such as, comorbidities, differences in referencing (Cz, linked ears or an average reference), artifacting, statistical methods, montage, methodology [EEG, MRI,

Petscan, Glucose Metabolism, blood flow], and selection of subjects [Davidson's criteria for inclusion was for subjects who had extreme or stable asymmetry over time] and individual differences in response to the testing situation [Coan and Allen, 2006, Harmon-Jones, 2006, Papousek & Schulter, 2006]).

A new approach to studying the functional aspects of frontal asymmetry was developed to resolve some of the inconsistent findings. Coan and Allen (2006) proposed a capability model of individual differences in frontal EEG asymmetry. They point out that all of the studies to date have depended upon resting (inherent) asymmetry that might be classified as dispositional models of personality. The implication is that basic traits will determine how an individual will behave in different situations. The capability model on the other hand is an alternative, interactive way to look at approach and avoidance states when the individual is confronted with an emotionally significant task. By presenting emotional challenges to an individual one can determine the degree to which individuals may utilize approach and avoidance responses. EEG correlations with affect were most significant when subjects were presented with emotions of fear and sadness. The reactions to emotional manipulation were so clear that issues of reference schemes were minimized.

Frontal EEG Asymmetry and Mood Disorders

In citing research on stroke patients where the severity of depression was related to the location of a lesion (left frontal pole lesions were associated with greater negative feelings than lesions near the right frontal pole) a theory was developed regarding left frontal hypo-activation and depression (Robinson et al., 1984, Henriques & Davidson, 1991, Gotlib, Ranganath, Rosenfeld, 1998). Numerous studies have explored the ramifications of this theoretical construct.

Inheritability and stability of the asymmetry trait have been studied (Coan, 2003). Developmental studies showed that infants increased with right frontal arousal reacted more to maternal separation than did those with left frontal activation (Tomarken, Davidson, Henriques, 1990), and adult studies demonstrated that right frontal arousal was associated with negative responses to film clips (Tomarken et al., 1990, Allen, Harmon-Jones, Cavender, 2001). Brief mood shifts brought about by happy and sad thoughts resulted in changes in frontal asymmetry in both depressed and normal control subjects (Baehr, 2002). Daily changes in frontal alpha asymmetry were shown to correlate with changes in affect in therapy sessions (Rosenfeld et al, 1996). Right frontal activation measured in a resting state was found in adults with current, remitted and past depressions (Henriques and Davidson, 1990, 1991; Gotlib et al., 1998). Right frontal activation was found in non-depressed adolescent daughters of depressed mothers (Tomarken, Diehter, Garber, Simien, 2004). The left frontal alpha asymmetry has also been found in infants under the age of one that were born to depressed mothers (Dawson et al., 1992a, 1992b), even as young as at 3-6 months (Field et al., 1995) and at 1 month of age (Jones, Field, Fox, Lundy, & Davalos, 1997). This may result from either a genetic predisposition to depression that has been passed on, and/or it may result from an over- or under-activation of brain areas that mediate different emotions in the infant whose frontal lobe begins to be increasingly active at about 8 months of age. However, genetic studies of twins provided only limited evidence of heritability of frontal asymmetry patterns (Smit, Posthuma, Boomsma, DeGeus, 2006, Anokhin, Heath, Myers, 2006). For the most part, these studies imply that right frontal cortical EEG may predict both state and trait individual differences in affect, although not all studies confirm these results (Debener et al., 2000, Allen et al., 2004; Vuga, et al., 2006).

Allen et al., (2003) studied subjects over an 8 to 16 week period. They acknowledged that while trait-like aspects of alpha asymmetry were characteristic of depressed individuals, state changes also occurred. Changes were not related to changes in the severity of the depression. However, Askew (2001) found a strong correlation between alpha asymmetry scores and the Beck depression Inventory ($P<0.0001$) and on the MMPI-II Depression Scale ($P<0.0001$).

The roles of early experiences and plasiticity as factors in patterns of asymmetry were explored by Davidson, (1994). Vuga et al., (2006) studied long–term stability for frontal EEG asymmetry in adults with a history of depression and non-depressed controls. They found that resting asymmetry reflected a moderately stable condition in adults. Deldin and Chiu, (2005) found that depressed persons could respond to a brief cognitive restructuring task, with positive changes also occurring in alpha asymmetry.

An asymmetry with more fast frequency activity in the right hemisphere has even been found to remain in the architecture of sleep in depression (Armitage et al., 1992, 1993, 1995). Research has further suggested that the right hemisphere may be specialized for processing negative affect (Ahern & Schwartz, 1985; Joseph, 1999; Ladavas, Nicoletti, Umilta, et al., 1984; Schwartz, Davidson, & Maer, 1975).

Along with the frontal electrophysiology findings in depression there also seems to be an inverse relationship between frontal alpha asymmetry and parietal asymmetries. More specifically, depressed patients without significant anxiety appear to have decreased right parietal activation (more alpha at P4 than at P3) (Allen et al., 1993; Bruder et al., 1995; Davidson et al., 1985; Henriques & Davidson, 1990, 1997; Schaffer et al., 1983; Tenke et al., 1993). These findings are also congruent with neuropsychological test findings that have consistently identified

right parietotemporal deficits in functioning in depressed subjects (Bruder, 1995; Heller et al., 1995; Jaeger et al., 1987). Recent research (Bruder, Tenke, Warner, & Weissman, 2007) has also verified an inverse parietal alpha asymmetry in grandchildren of depressed parents and grandparents compared with controls without a parental and grandparental depression history.

In summary, in spite of inconsistencies, there is overwhelming support for theories relating EEG frontal cortical asymmetry (state and/or trait dependent) and emotions, both normal and pathological. These studies have provided a substantial theoretical basis for research and for the development of clinical EEG biofeedback protocols designed to train depressed subjects to alter their brainwave patterns.

Clinical Use of Asymmetry protocols for treatment of depression:

Baehr/Rosenfeld Studies

There are assessments (as opposed to treatment outcome studies) that have used the Rosenfeld protocol. Eleven non-depressed age-matched controls and 13 depressed patients participated in a study to compare frontal alpha asymmetry mean for a baseline session with the percentage of time in the session when asymmetry scores were greater than zero. It was found that the percent index was a better discriminator of the two groups than was the asymmetry score. (Baehr et al., 1998). An A-score of 58% is the cut off point between depressed and non-depressed subjects. As mentioned, Askew (2001) also provided further validation for this measure in finding a strong correlation between alpha asymmetry scores and the Beck depression Inventory and the MMPI-II Depression Scale.

The phenomenon of brainwave asymmetry and emotions has been further explored by Baehr and colleagues (2000). Twenty-two subjects, including 14 patients being treated for

depression and 8 non-depressed subjects were asked to think of a happy or sad thought while alpha asymmetry measures were being taken at cortical sites F3 and F4, referenced to CZ. Sixty-three percent of the subjects demonstrated the ability to change their frontal alpha asymmetry in accordance with voluntarily produced thought change. The remaining 37 % of the subjects remained either in the positive alpha asymmetry range (R>L), or in the negative alpha asymmetry range (L>R).

In a study of premenstrual dysphoric disorder (Baehr, Rosenfeld, Miller, & Baehr, 2004a) evidence was found of changes of frontal asymmetry during the luteal phase of the menstrual cycle that was consistent with severe mood swings. They observed two monthly cycles for 5 women diagnosed as having PMDD and one monthly cycle for five non-PMDD control subjects. They found that the Asymmetry percent scores for the 5 PMDD women, and for the five control subjects before and after the Luteal phase were typically within the normal non-depressed range, however the asymmetry scores for the PMDD group fell into the negative range during the Luteal period while the control subjects remained stable.

Replication Studies

A single case study of an adolescent patient who was treated for depression using the EEG asymmetry biofeedback protocol, replicated our results. (Earnest, (1999). In a study of eighteen women using the asymmetry protocol, John Allen et al. (2001), demonstrated that frontal asymmetry could be manipulated to train subject's left or right frontal alpha asymmetry. Self reported emotional responses to film clips and interpretation of facial expressions were consistent with expressions predicted from the EEG training efforts.

Two other papers (Hammond, 2000, 2005b) have been published on the neurofeedback

treatment of depression, both of which built on the same robust foundation of frontal asymmetry research. Hammond (2000) utilized a protocol designed to increase left frontal beta arousal (15-18 Hz and 12-15 Hz) in the left frontal cortex at electrode sites FP1 and F3 in a successful single case study. He later reported (Hammond, 2005b) on the successful treatment of seven of eight subjects using the same protocol.

Baehr, Rosenfeld, Baehr and Earnest, (1999) presented the results of a pilot study in a previous edition of this book A review of that research along with follow-up data will be presented , followed by a detailed review of Hammond's research.

Review of Previous Research

In 1994 Baehr and Rosenfeld introduced their protocol to five depressed patients who were being treated with psychotherapy, and a sixth patient who was seen in another clinic. (Detailed case studies are presented in chapter 8 of the previous edition of this book [Baehr et al, 1999]). They agreed to participate in a study to assess the effectiveness of this approach. (Baehr et al, 1997b, Baehr et al., 1999).[1] Using the alpha asymmetry protocol, they assessed and trained depressed patients to reallocate brainwave amplitude so that the amplitude of alpha was greater in the right frontal cortex then in the homogolous left frontal cortex. The application of this protocol requires scalp electrodes at 2 active sites, F3 and F4, a reference at Cz, and the ground at Fz. (Most of the above reported studies have used a standard EEG montage utilizing 19 or more sites).

Procedures

The Beck Depression Index (BDI), and the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) were administered to assess emotional functioning before and after a series of

approximately 30 sessions of EEG asymmetry training designed to increase the difference between right and left alpha magnitude.[2],[3] Prior to neurofeedback training the patients were trained to use diaphragmatic breathing exercises and autogenic suggestions such as "I feel quite relaxed", and "warmth is flowing down my arms into my hands and fingers" to promote relaxation and hand warming. This technique serves to reduce EEG artifacts caused by muscle tension. The patients were also encouraged to focus their thoughts on pleasant, unemotional imagery during EEG training sessions. They sat in a reclining chair with their feet elevated, and were encouraged to maintain a relaxed state, closing their eyes and moving as little as possible.

The patients were seen once or twice a week for hour-long sessions for an average of 30 sessions that consisted of approximately 50% brainwave biofeedback followed by 50% psychotherapy. During biofeedback, scalp sites F3 and F4, referenced to Cz, were recorded. Impedances were 5k ohms or less, as measured by an EIM electrode impedance meter. The threshold was set at zero so that A - scores below zero represented greater left than right alpha magnitude, and A- scores above zero represented the reverse asymmetry. Alpha rhythm reflects cortical hypoarousal; therefore an increase in left frontal activation corresponds to decreased alpha and a positive change in the asymmetry score. To assess significant A-score change, we rely on our previous study (Baehr et al., 1998), in which we found that A-scores >58% of time over threshold were typical of non-depressed normal control subjects, while A -scores < 58% percent of the time were representative of the depressed population.

The EEG data for A-score training were recorded either on a 4-channel unit or on a Neurosearch 24 channel unit (both by the Lexicor Corp). Fast Fourier Transforms (FFTs) were derived on Blackman-Harris windowed analog signals over 1-second epochs (Harris, 1978). This

device also outputs the mean value over the entire session each day as a mean Asymmetry score which is manifested as a positive or negative asymmetry score and as a mean percentage score, reflecting the percentage of time that the difference between the right and left alpha magnitude is greater than zero, (A-score >0). A bell tone or a clarinet tone that fluctuates in pitch, (the greater the A-score, the higher the tone), was used as a reinforcement when the Asymmetry score exceeded zero.

Results

Each subject was his own control in this study that utilized pre and post treatment data. Five of the six subjects were able to increase their percent of time over threshold to the normal range. The sixth subject, whose depression was diagnosed as non-endogenous, increased her asymmetry score but fell just short of reaching the cut-off score of < 58%. Four of the six subjects showed significant improvement on the BDI and on the MMPI. A comparison of the patients'= pre and post MMPI-2 Depression Scales indicate a significant change. For three patients the pre to post depression score differences exceeded two times the standard error of measurement, (SEm), and for one patient, one (SEm) Table 1a).

Five of the six subjects scored above 9 on the BDI, while four of the six scored below 9 in the posttest (Table 1a. Scores below 9 on the BDI are considered to be within the normal range.).

Longitudinal Data

Five of the six subjects were available for follow up. In our first longitudinal study three subjects (Bob, Celia and Ann Rose in Table 1b) were evaluated one to five years after therapy (Baehr et al, 2001). All subjects maintained asymmetry scores and Beck Depression scores in the normal

range. (Table 1b)

Three subjects were available for evaluation ten years after finishing therapy. One of these subjects, Ann Rose, participated in both follow-up studies. Katie was the formerly Bi-Polar patient and Catherine was formerly diagnosed as having unipolar depression. All subjects maintained asymmetry scores in the normal range and all had Beck depression Scale scores in the normal range. (Table 1c)

Insert Tables 1a, 1b and 1c

Treatment of a Bipolar Patient

Research has shown that bipolar disorder quite commonly has a different EEG pattern than unipolar depression, where alpha is often reduced (Knott & Lapierre, 1987; Clementz et al., 1994), beta elevated (John et al., 1988; Prichep & John, 1986), and where manic and depressive phases may be characterized by different EEG patterns (Flor-Henry & Koles, 1984; Koek et al., 1999).

Thus while we may wish to view the Baehr/Rosenfeld asymmetry protocol as a significant treatment innovation for mood disorders, it is apparent that it does not work in the same way for everyone. For example, in the case of the person diagnosed with bipolar depression, improvement occurred in terms of eliminating mood swings, but the patient remained in a dysphoric state at the end of the 30 sessions of treatment. This case also was complicated by reactions to psychotropic medications. She remained in neurotherapy and psychotherapy for an additional five years. Her asymmetry eventually normalized and at a ten-year follow-up she reported that she was neither bipolar nor dysphoric. Her asymmetry ratio was within the normal range, >58%, (Baehr, 1998) as was her BDI score of 2. She continues to use a minimal amount

of medication.

Adjunctive Treatments with the Baehr/Rosenfeld Asymmetry Protocol

Training Breath and Heart Rate Variability. Since the first Baehr study, they have refined the relaxation techniques prior to training with the Asymmetry protocol. All of the sessions begin with ten minutes of training to balance the sympathetic and parasympathetic arousal of the autonomic nervous system, as described by Elliott & Edmonson (2006). Using a combination of programs, patients are trained to use breathing to regulate heart rate variability (Elliott & Edmonson, 2006; Childre & McCraty, 1999). When successful, patients report that they feel calmer and in better control of responses to stress and depression. Validation of this technique has been reported in two new studies: Rotenberg, Clift, Bolden, & Salomon, (2007) found a relationship between major depression and respiratory sinus arrhythmia, and Karavodas, Lehrer, Vaschillo, Vaschillo, Marin, Buyske, Malinovsky, Radvanski, Hassett (2007) found that improvement in symptoms of major depressive disorder occurred with short term biofeedback treatment to increase heart rate variability.

Other Adjunctive Therapies. The Baehr group considers neurofeedback training for depression as one part of a comprehensive treatment protocol, which may also include entrainment devices such as audio-visual stimulation, nutritional counseling, exercise programs and ongoing psychotherapy. The lab setting where the neurofeedback treatment occurs and the alliance with the therapist also may be important factors, as yet unanalyzed, in the treatment situation. Some question is raised as to whether the positive effects observed would also occur in a lab setting where a therapist was not present.

This group is currently assessing alpha asymmetry in a normal population. They believe

that the crucial next step for their research is to demonstrate that appropriate depressed control subjects do not improve clinically as much as clinical subjects who are administered the asymmetry protocol.

The Hammond Depression Protocol

Hammond (2000) used the Baehr/Rosenfeld protocol described above for 3 sessions with a case of medication resistant depression accompanied by anxiety and obsessional rumination. The patient's score on the protocol was 36.1, representing a severe asymmetry. However, as chance would have it, the patient had considerable difficulty in changing his asymmetry score, which was actually worsening in his scores in sessions 2 and 3. Hammond was familiar with the frontal asymmetry EEG research, as well as neuroimaging research (e.g., Baxter et al., 1985, 1989; Bench et al., 1992, 1993; George et al., 1994; Liotti et al., 2000) and evidence from work by Sterman (1999, also later published in Sterman and Kaiser, 2001) suggesting that the area anterior to electrode site F3 also appears to be hypoactive in depression. Therefore, he considered what other neurofeedback treatment strategy might address this asymmetry.

It was decided to reinforce beta arousal while inhibiting alpha and theta arousal in the left frontal area at electrode sites Fp1 and F3. Within one session the patient reported sensing an improvement. At the completion of treatment the MMPI depression scale had improved from 97 t-scores to 56 t-scores. Somatic symptoms (gastritis, headaches, aches and pains, and preoccupation with health) dramatically improved, as did his over-emotionality, anxiety and rumination, and fatigue. MMPI and reports from the patient demonstrated that he had become less withdrawn, more active, sociable, and less distrustful. There literally was more "approach" behavior, and the changes were maintained at 8 ½ month follow-up.

As a result of this successful case outcome this protocol continued to be used. Clinical experience demonstrated that occasionally a patient reported becoming over-activated from the reinforcement of 15-18 Hz beta, reporting feeling somewhat more irritable, anxious, and having some difficulty falling asleep. Therefore, the protocol was modified so that while inhibiting alpha and theta activity, 15-18 Hz beta was reinforced for 20-22 minutes and then the reinforcement band was changed to 12-15 Hz for the last 8-10 minutes. No further over-activiation side effects were seen after that modification. On occasion a patient was also found to demonstrate considerable delta activity in the left frontal area, in which case delta might also be inhibited.

Following other successful clinical experiences, Hammond (2005b) reported on another case series of 9 consecutive patients with a primary presenting complaint of depression, which was confirmed on the Minnesota Multiphasic Personality Inventory (MMPI). The only other selection criterion was that each patient had a screening assessment of 3 two minute samples with the Baehr/Rosenfeld protocol to ascertain the presence and degree of the frontal asymmetry (predisposition to depression). As reviewed earlier in this chapter, percentage scores greater than 60 suggest that there is not a predisposition to depression, while percentage scores of 58 or less suggest the presence of a predisposition. The mean percentage score for this sample was 40.05, and their mean score on the MMPI Depression scale was 93.75 t-scores—a serious level of depression. Whereas patients in medication studies are often moderately depressed, 7 of the 8 patients in this series were judged to be seriously to severely depressed, with only one being moderately depressed. Cases cited by Baehr et al. (1997, 2001) involved relatively mild depression in the 62-64 t-score range on the MMPI, with an average asymmetry protocol score of

51.3.

The outcomes reported by Hammond (2005b) differed in 4 ways from the Baehr/Rosenfeld cases. First the sample was significantly more depressed. Second, the Fp1 and F3 protocol was different. Third, treatment duration was only approximately two-thirds the length. And finally, concurrent psychotherapy and relaxation training was purposely not provided to better determine treatment effects from purely the neurofeedback, without contamination from relaxation or cognitive therapy. Eight of the patients completed training, requiring an average of approximately 21 thirty-minute sessions (10+ hours) of neurofeedback, with no other psychotherapy provided. Seven of eight patients made very substantial improvements, and one other patient dropped out after 5 sessions because he was "too busy." The patient who prematurely terminated showed signs of questionable motivation from the beginning, seeming to be in treatment primarily to please his wife and daughter. Many of the patients were on medication at the time of initial testing, but were no longer on medication at the completion of treatment.

Pre-post changes on the MMPI may be seen in Figure 1. There was a mean decrease in the depression scale of 28.75 T-scores. One patient showed improvement from being classified as *severely* depressed to normal and two improved from being classified as *seriously* depressed to normal. Three showed improvement from severe to mild depression, and one showed improvement from moderately depressed to mildly depressed. One case who was severely depressed only showed mild improvement. This was an individual who had lost his wife to cancer a year earlier and issues surrounding this loss seemed likely to need to be addressed, and he was referred for psychotherapy for these issues. Excluding this last subject and the drop-out

failures, 77.8% of the subjects made significant improvements. The average length of follow-up for these cases was about 1 year, with a range from 2 years in two cases, to 4 months in the case of the individual who only mildly improved. It is fortunate that the MMPI was used as an outcome measure, rather than only a measure of depression. MMPI results (see Figure 1) have commonly shown not only decreases in depression, but also in other scales measuring anxiety, obsessive rumination, withdrawal, and introversion, while ego-strength has improved. The decreases in being withdrawn are congruent with what we would expect when an approach motivation area of the brain is being activated.

All total Hammond has treated at least 3 dozen depression cases using this depression protocol, with consistently positive results in an estimated 75-80% of cases. Anecdotal reports from other clinicians using this protocol have also been positive. In ongoing clinical practice, other psychotherapeutic techniques (self-hypnosis training, respiration biofeedback training, cognitive therapy, biblio-therapy) are often added to treatment, but by far the largest component of treatment has remained the use of the Hammond Depression Protocol. Occasionally the protocol has been done eyes closed when a patient was simply producing too much eye movement artifact, and in each case where this alteration has occurred, the outcomes remained positive. It should be added that clinical experience in the treatment of obsessive-compulsive disorder (Hammond, 2003, 2004), supported by research (Maihofner et al., 2007) implicating the left frontal area in Obsessional Compulsive Disorders (OCD), have also been suggestive that this protocol may prove helpful as one of the modules in OCD treatment with neurofeedback.

Other Neurofeedback Studies With Depression

A unique form of neurofeedback, the Low Energy Neurofeedback System (LENS)

(Hammond, 2007; Ochs, 2006) has also been used with mood disturbances. LENS training differs from other forms of neurofeedback in that it introduces a micro radio-wave electromagnetic signal which is only about the intensity of the output coming from a watch radio battery--far, far weaker than the input you receive from simply holding a cell phone to your ear. This very low intensity input is introduced down the electrode wires for only a few (e.g., often just 1-7) seconds. Its frequency varies depending on the dominant brainwave frequency from moment-to-moment and it is designed to gently nudge the brain become more flexible and self-regulating, reducing excess amplitude and variability of the brainwaves. Larsen, Harrington, and Hicks (2006) recently reported on a case series of 20 patients where over the course of 20 sessions there was a significant decrease in self-ratings of depression. Ratings decreased from an initial average of almost 8 (on a 0-10 scale) to less than 5 in just 6 sessions, and to less than 3½ at the end of 20 sessions.

Neurofeedback training to increase alpha and theta, while inhibiting faster beta frequencies has also been found to produce significant improvements in depression in alcoholic and post-traumatic stress disorder populations in randomized, control group studies (Peniston & Kulkosky, 1990; Peniston et al., 1993) as well as in a case series (Saxby & Peniston, 1995)—populations where one may expect an excess of fast beta activity to often be prominent and which is quite different from the EEG patterns usually seen in depression.

Research in Europe (Hardman et al., 1997; Kotchoubey et al., 1996; Rockstroh et al., 1990) using neurofeedback to alter slow cortical potentials has also demonstrated that it can be used to alter hemispheric asymmetries and thus may also hold potential for use in the treatment of depression.

Summary & Conclusions

A robust body of research has validated that there is a biological predisposition to depression (and to becoming withdrawn) which is associated with a frontal asymmetry wherein there is less arousal in the left frontal area. Although pharmacologic treatment for depression is widespread, reviews (e.g., Antonuccio et al., 1999; Greenberg et al., 1992; Hammond, 2007; Kirsch et al., 2002; Kirsch & Sapperstein, 1998; Moncrieff, 2001) have documented that antidepressants, on average, only have an 18% effect over and above placebo effects, and yet they are associated with significant side effects such as sexual dysfunction, insomnia, increased suicide risk, diarrhea, nausea, anorexia, bleeding, forgetfulness, and withdrawal syndromes.

Thus alternatives are needed to the invasive treatments commonly utilized by "biological psychiatry," namely medication, electroconvulsive therapy, transcranial magnetic stimulation, and neurosurgery, which are commonly associated with side effects. Studies (e.g., Elkin, Shea, Watkins, Imber, Sotsky, Collins, et al., 1989; Hollon, DeRubeis, Evans, Wiemer, Garvey, Grove, et al., 1992; Hollon, Shelton, & Loosen, 1991) that have compared psychotherapy with medication have found that treatment outcomes are generally comparable or better than pharmacologic treatment, and when dropout rates are taken into account, drug treatment alone produces worse outcomes than psychotherapy.

However, the authors have found that neurofeedback offers an additional noninvasive treatment alternative with depression. While more controlled research is certainly needed, neurofeedback that is targeted to altering the frontal asymmetry found in depression has consistently produced favorable results in a majority of clinical cases. LENS neurofeedback, slow cortical potentials training, and the use of alpha/theta neurofeedback training with an

alcoholic or PTSD population also appear promising in the treatment of depression.

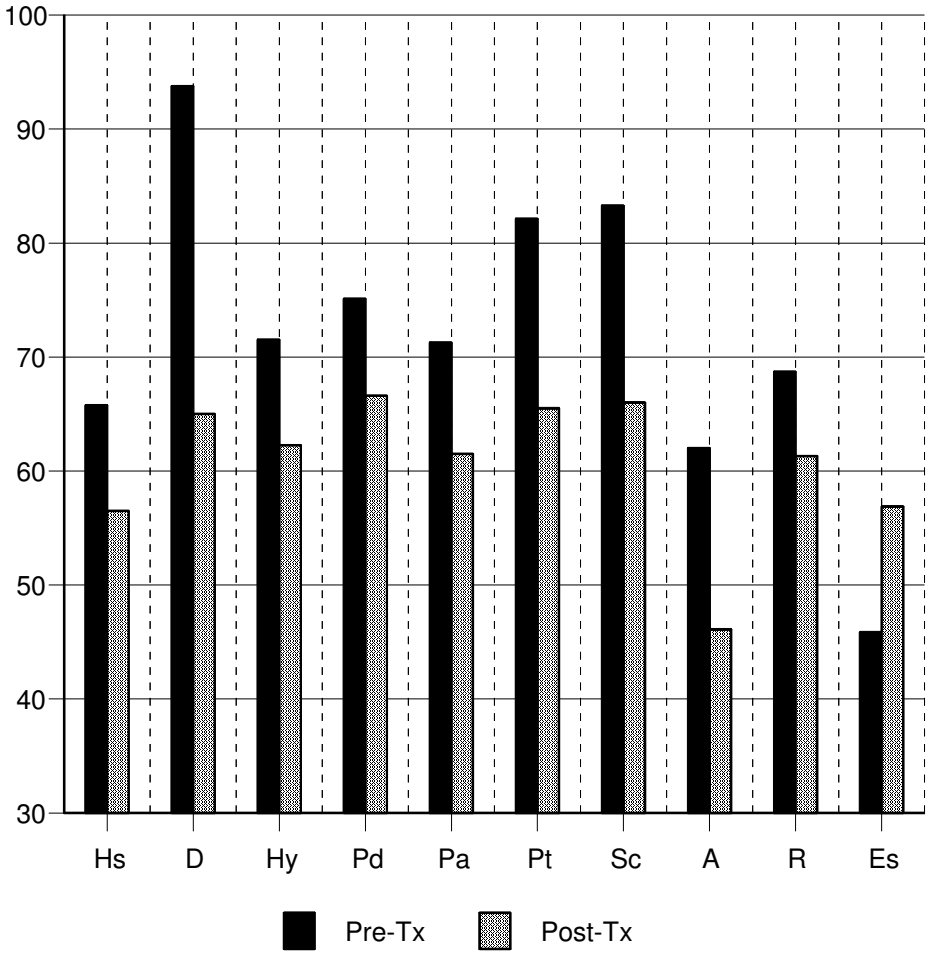

Figure 1. Hammond Depression Protocol: Average MMPI Pre-Post Changes for 8 Cases

Table 1a. Pre- and Post-Alpha Asymmetry Training Measure of Depression for the MMPI-2 and BDI, and the Percent of Time Asymmetry Is Greater Than Zero *

	MMPI-2	MMPI-2	BDI	BDI	A%	A\%
Subjects	Pre-alpha	Post-alpha	Pre-alpha	Post-alpha	Pre-alpha	Post-alpha
Bob	76	54[a]	21	03	48	84
Celia	74	62[b]	40	04	57	80
Katy	n/a[c]	n/a	07	25	50	69

Ann Rose	**64**	**47[a]**	**n/a**	**01**	**49**	**69**
Catherine	**62**	**36[a]**	**11**	**01**	**59**	**64**
Diedre	**n/a**	**n/a**	**34**	**18**	**36**	**55**

Reprinted from Baehr. E., Rosenfeld,, J.P., Baehr, R., Earnest, C. (1999)" ***Introduction to Quantitative EEG and Neurofeedback (J.R. Evans, & A. Arbarbanel, Eds.) Academic Press, New York) 181-203.***

Table 2b. 5-Year Follow-Up: The Beck Depression Inventory (BDI) scores[*] and the Percent of Time Asymmetry Is Greater Than Zero (PTAA) Scores Are Shown for Three Subjects Before and Immediately After Termination of Therapy.

	BDI	BDI	BDI	PTAA	PTAA	PTAA
SUBJECT	**Before Therapy**	**After Therapy**	**Follow-Up One to Five Years Later**	**Before Therapy**	**After Therapy**	**Follow-Up One to Five Years Later**
Bob	**31**	**03**	**03 (1 year)**	**48%**	**84%**	**86% (1 year)**
Celia	**40**	**04**	**04 (3 years)**	**57%**	**86%**	**66% (3 years)**
Ann Rose	**n/a**	**02**	**03 (5 years)**	**49%**	**69%**	**69% (5 years)**

Reprinted from: Baehr, E.,Rosenfeld, J.P., Baehr, R. (2001)*Clinical use of an alpha asymmetry protocol in the treatment of mood disorders, follow-up study one to five years post therapy***. Journal of Neurotherapy, 4(4), 11-18**

Table 3c 10-Year Follow-Up: The Beck Depression Inventory (BDI) scores[*] and the Percent of Time Asymmetry Is Greater Than Zero (PTAA) Scores Are Shown for Three Subjects Before and Immediately After Termination of Therapy.

	BDI	BDI	BDI	PTAA	PTAA	PTAA
SUBJECT	**Before Therapy**	**After Therapy**	**Follow-Up Ten Years Later**	**Before Therapy**	**After Therapy**	**Follow-Up Ten Years Later**
Katy	**7**	**25**	**02**	**50%**	**69%**	**75%**
Catherine	**11**	**01**	**02**	**59%**	**64%**	**72%**
Ann Rose	**n/a**	**01**	**02**	**49%**	**69%**	**61%**

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[*] BDI score < 9, and PTAA scores > 58 are in the non-depressed range.

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[1] We thank Carolyn Earnest for providing us with data for one client in this study. This is a different subject than the one reported in a replication study (Earnest, 1999).
[2] The Baehr/Rosenfeld protocol utilized the index [(R-L)/(R+L)] X 100 as the asymmetry index or A-score, where R and L represent right and left frontal alpha magnitude (microvolts) respectively. The higher the value of this index, the less depressed the patient is assumed to be (see earlier parts of this paper and Rosenfeld, 1997).
[3] One patient who was initially diagnosed Bipolar remained in therapy for several years after the study terminated. She will be presented later in this paper.

[a] Two SEM p > 0.0005
[b] One SEM p > 0.0025
[c] N/A, tests were not administered

Table 2b 5-Year Follow-Up: The Beck Depression Inventory (BDI) scores* and the Percent of Time Asymmetry Is Greater Than Zero (PTAA) Scores Are Shown for Three Subjects Before and Immediately After Termination of Therapy.

	BDI	BDI	BDI	PTAA	PTAA	PTAA
SUBJECT	Before Therapy	After Therapy	Follow-Up One to Five Years Later	Before Therapy	After Therapy	Follow-Up One to Five Years Later
Bob	31	03	03 (1 year)	48%	84%	86% (1 year)
Celia	40	04	04 (3 years)	57%	86%	66% (3 years)
Ann Rose	n/a	02	03 (5 years)	49%	69%	69% (5 years)

Reprinted from: Baehr, E.,Rosenfeld, J.P., Baehr, R. (2001)*Clinical use of an alpha asymmetry protocol in the treatment of mood disorders, follow-up study one to five years post therapy*. Journal of Neurotherapy, 4(4), 11-18

∗ BDI score < 9, and PTAA scores > 58 are in the non-depressed range.

Table 3c **10-Year Follow-Up: The Beck Depression Inventory (BDI) scores* and the Percent of Time Asymmetry Is Greater Than Zero (PTAA) Scores Are Shown for Three Subjects Before and Immediately After Termination of Therapy.**

	BDI	BDI	BDI	PTAA	PTAA	PTAA
SUBJECT	Before Therapy	After Therapy	Follow-Up Ten Years Later	Before Therapy	After Therapy	Follow-Up Ten Years Later
Katy	7	25	02	50%	69%	75%
Catherine	11	01	02	59%	64%	72%
Ann Rose	n/a	01	02	49%	69%	61%

* BDI score < 9, and PTAA scores > 58 are in the non-depressed range.