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Clinical Use of an Alpha Asymmetry Neurofeedback Protocol in the Treatment of Mood Disorders

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I. INTRODUCTION

The relationship between mood disorders and cortical asymmetry was first described by Robinson *et al.* (1984) when they observed that damage to the left frontal lobe results in symptoms of depression. On the other hand, they found that patients who displayed manic symptoms following a lesion were much more likely to have sustained damage to the right frontal area. This led Davidson (1995) and others (cited in his 1995 work) to postulate that brain systems mediating positive or approach behavior are located in the left frontal area (so that lesions here would lead to negative affect), whereas systems mediating negative or withdrawal behavior are located in the right frontal area (so that lesions here would lead to mania). Davidson's group has, for the past decade, largely confirmed this hypothesis of localization of emotion using electrophysiological (EEG) methods (Davidson, 1995). Davidson's group utilized alpha magnitude or power as an inverse index of cortical activation, such that high alpha means low activation; that is, alpha activity may thus be viewed as a kind of lesion. High right frontal alpha, like a right frontal lesion, would correlate with

positive affect, whereas high left alpha power is comparable with a left frontal lesion.[1] (Tomarken *et al.,* 1990, 1992).

Davidson's early work (Henriques & Davidson, 1990, 1991; Davidson, 1995) confirmed his hypotheses by recording from scalp sites F4 and F3, both referenced to CZ, and developing an asymmetry measure based on the formula, $A = \log(R) - \log(L)$, where R and L are right (F4) and left (F3) alpha power, respectively. It may be seen that as this A score increases, there is relatively more right than left frontal alpha activity (less right than left cortical activation). His research group has shown that normals have higher A scores than currently depressed persons. In a separate study, they showed that normals have higher A scores than previously depressed (but now remitted) persons. One of us replicated and extended this study by comparing all three groups in one study, finding no differences between currently and previously depressed persons, but a difference between normals and the other two groups (Gotlib *et al.,* in press). The lack of difference between previously (remitted) and currently depressed persons has been interpreted as evidence that the low A score is a trait marker for vulnerability to depression, as well as a correlate of current state, as suggested by Rosenfeld *et al.* (1996). Davidson's group has provided much confirmatory evidence of the A score as both state and trait marker (Davidson, 1995; Wheeler *et al.,* 1993) using a variety of referencing methods.

This research led us to speculate about the possibility of developing a new EEG biofeedback modality for treatment of depression. The first thing we needed to show was that the A score was modifiable in normals using a simple operant conditioning program that presented reward tones for significant increases in A scores on an epoch-to-epoch basis. This we did in a study (Rosenfeld *et al.,* 1995) in which we reported that 9 of 13 normals learned after 3 days of training to double the number of EEG epochs containing increased A scores. This work was replicated by Allen and Cavendar (1996). It then seemed appropriate to try out this novel neurofeedback protocol in real clinical patients (Baehr *et al.,* 1995; Baehr & Baehr, 1997). This work, still ongoing, is described in the next section of this chapter with a review of five specific cases.

Two technical details need to be clarified: (1) In our work with patients we use a different algorithm, $A = [(R - L)/(R + L)]$, to define the A score, rather than the metric used in the aforementioned studies (Henriques & Davidson, 1990, 1991; Davidson, 1995). In this case R and L are the magnitudes of alpha activity (in microvolts) at F4 (R) and F3 (L). Charan Ranganath and Peter Rosenfeld, in unpublished pilot studies, took data from five

[1] Beta activity might be thought of as a preferable direct measure of activation, but since electromyographic activity (EMG) has sizeable harmonics in the Beta range, Beta could confound cortical and muscle activity unless considerable care is taken to remove EMG-contaminated data. It is quite straightforward to use alpha as an inverse index of activation, and virtually no EMG leaks into the alpha band.)

subjects run in the Gotlib *et al.* (in press) study and analyzed *A* scores as defined earlier in both ways. They found correlations of 0.996–0.999 between *A* scores defined in the two ways described earlier. (2) It must be noted that with a dependent variable such as *A,* which is defined and measured as a function of two other variables, *R* and *L,* one cannot attribute any observed change in *A* to either *R* or to *L.* All we can know is that the relationship of *R* and *L* has changed. Thus, when we report an increase in A with training, we do not know whether this involves an increase in *R,* a decrease in *L,* or both of these changes simultaneously. Other referencing schemes (of a complex nature for a clinical setting) or imaging methods may be utilized in future work to localize precisely the source(s) of change in *A* score with neurofeedback.[2]

II. CLINICAL USE OF THE ASYMMETRY PROTOCOL

Beginning in spring of 1994, a small group of depressed patients of Drs. Elsa and Rufus Baehr agreed to try an experimental treatment for mood disorders. The rationale for this treatment stems from research mentioned earlier in this chapter in which differences in frontal EEG asymmetry, characterized by apparent left frontal hypoactivity, have been linked with depression (Henriques & Davidson, 1991). Furthermore, such asymmetry was found to be present in infants who were separated from their mothers (Davidson & Fox, 1989), and was found to index a trait identified with depression-vulnerable individuals, even when they were not experiencing depression (Allen *et al.,* 1993; Henriques & Davidson, 1990; Gotlib *et al.,* in press). In addition, resting brain asymmetry was found in adolescent children whose mothers had a history of depression, as compared to a group of children whose mothers had no history of depression (Tomarken *et al.,* 1994).

We reasoned that depressed persons might benefit from training to increase differences in activation in the right and left frontal cortices. Other researchers such as Lubar (1991) and Sterman *et al.* (1972) had demonstrated that EEG biofeedback training can have stable, long-lasting effects on clinical conditions. We hypothesized that if this asymmetry training was successful, then tests designed to assess depression would reflect improvement in affect, and that the asymmetry changes would hold over time.

[2] A major new finding (Baehr *et al.,* 1998) demonstrates that the percent of the time of the recording session in which the positive A score is greater than zero, better discriminates depressed vs. control subjects than the A score itself, the latter being the only metric correlated with affective performance in earlier studies. On the basis of preliminary results in only 24 subjects, it may be suggested that PCT <55 suggests the presence of depression; a PCT score of >60 suggests no depression.

In the remainder of this chapter we review briefly the various major symptoms associated with a variety of mood disorders. This information is followed by a discussion of our initial clinical findings in this small sample of patients. We then discuss some of the factors believed to be associated with the successes and failures observed with the use of the asymmetry protocol.

III. THE CLASSIFICATION OF DEPRESSIVE DISORDERS

In the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994), diagnostic criteria for mood disorders are presented. This classification is widely used by clinicians to categorize the symptomology presented by depressed patients. The general category of depressive disorders consists of unipolar and bipolar disorders. These are distinguished from mood disorders categorized by specific etiology, such as depression due to generalized medical condition and substance-induced mood disorders.

A. UNIPOLAR DEPRESSIVE DISORDERS

Major Depressive Disorder (296.xx). Characterized by one or more major depressive episodes (at least 2 weeks of depressed mood or loss of pleasure or interest, plus four or more other symptoms of depression).

Dysthymic Disorder (300.4). Characterized by depressed mood for a minimum of 2 years, plus at least four or more symptoms of depression.

Depressive Disorder Not Otherwise Specified (311.0). Characterized by depressive features that do not meet the criteria for the preceding disorders, or an adjustment disorder with depression/and or depression and anxiety.

B. BIPOLAR DISORDERS

Bipolar I Disorder (296.xx). Characterized by one or more manic or mixed episodes. A major depression usually accompanies bipolar I disorders.

Bipolar II Disorder (296.xx). Characterized by at least one hypomanic episode and one or more major depressive episodes.

Cyclothymic Disorder (301.13). Characterized by numerous periods of hypomanic symptoms and numerous periods with depressive symptoms occurring over at least a two year period. These

symptoms do not meet the criteria for either a manic episode or a major depressive episode.

Bipolar Disorder Not Otherwise Specified (296.80). Characterized by bipolar features that do not meet the criteria related to a general medical condition.

Substance Induced Mood Disorders (29x.xx). Characterized by mood disturbance caused by a drug of abuse, a medication, exposure to a toxin, or another somatic treatment for depression.

The coding allows for the addition of specifiers that further describe the disorder; that is, mild, moderate, with psychotic features, etc.

During the last several decades depression has emerged as a central mental health issue in our society. Anxiety disorders are no longer viewed consistently as the most prevalent illness. It has been estimated that about 14% of the general population will experience depression at some point in their lives, and that about twice as many women as men will be depressed (Rosenfeld, 1997). It has become evident that the phenomenon of depression requires more understanding as to its nature, as well as the exploration of more effective treatment strategies.

In a new book, edited by Akiskal and Cassano, (1997) entitled *Dysthymia and the Spectrum of Chronic Depressions,* mood disorders are presented as being chronic and occurring on a continuum, rather than as discrete episodes separated by periods of remission. This conceptualization encompasses the unipolar and bipolar disorders, and the subclinical disorders, as well as major melancholia (p. 54). Mood disorders are seen as enduring illnesses with an endogenous etiology. Chronicity, in this view, does not mean that symptoms are ever-present, but that there is an underlying depressive temperament that could emerge as mild temperamental pathology or as a major depressive illness with manic episodes. This concept is consistent with the aforementioned fact that previously depressed individuals have the EEG alpha asymmetry trait, whether they are currently depressed or not (Henriques & Davidson, 1990; Allen *et al.,* 1993; Gotlib *et al.,* in press).

Roth and Mountjoy (1997) argue that there is a spectrum of depressive states that extends from bipolar states at one end, to neurotic depression at the other end, and that nonendogenous disorders should be distinguished from endogenous disorders, not only because of differences in etiology, but because of differences in the course of the illness and the prognosis. They argue that the neurotic depressions have a different clinical profile; they are characterized by "episodic attacks which are separated by relatively clear intermissions broken by no more than mild occasional symptoms." They frequently evolve after some traumatic event, such as loss, demotion, or failure, and lack a history of trauma in childhood or adolescence, whereas dysthymia often occurs without a clear reason for onset, as well as sometimes

being triggered by adverse life events. The category "neurotic or reactive depression," found in DSM-III, has been eliminated from DSM-IV. Roth and Mountjoy (1997) argue for its inclusion, based on the fact that both the etiology and the treatment of this disorder are different from the chronic dysthymic disorders. They argue that the neurotic depressions are most amenable to psychological treatments, while the endogenous disorders are not "curable," and are at best treated with long-term psychotropic medications. Arieti and Bemporad (1978), in their study of severe and mild depression, recognize the value of medication, but they claim that a psychotherapeutic approach is basic to the treatment of all types of depression, whether or not there is a hereditary predisposition. All of the depressed subjects who participated in the studies Davidson and his colleagues conducted (as mentioned earlier) were classified as endogenously depressed. Henriques and Davidson (1991) hypothesized that frontal alpha asymmetry is a "state-independent marker of vulnerability to depression." Gotlib *et al.* (in press), as noted, recognize the inherited nature of the depressive pattern when they cite studies showing that nondepressed children of depressed parents show this asymmetry pattern, but they also refer to studies that demonstrate that the pattern of EEG alpha asymmetry may be found in subjects with nonendogenous backgrounds as well.

The patients we have treated all have family histories of depression, and are thus classified as endogenous. This distinction is crucial, because as we attempt to change a pathological EEG brain wave asymmetry pattern, we are also challenging widely held assumptions regarding the stability of a biochemically maintained trait, whether inherited or acquired. In any event, even if it is possible with neurofeedback to modify the neural circuitry, we feel there is still a need to psychotherapeutically process the emotional factors that accompany mood disorders.

IV. TREATMENT OF DEPRESSION USING THE ASYMMETRY PROTOCOL[3]

A. SUBJECTS

Four depressed female patients and one depressed male patient who participated in the asymmetry training were patients seen by Drs. Elsa and Rufus Baehr in their private practice. They were classified as endogenously depressed. The sixth person was the client of a colleague.[4] She was classified as nonendogenously depressed.

[3] A patented asymmetry protocol was used under license in this study. For information, contact Dr. Peter Rosenfeld, Department of Psychology, Northwestern University, Evanston, Illinois 60208.

[4] The authors wish to thank Carolyn J. Earnest, MSN, RN, CS for the contribution of her client's material.

B. PROCEDURES

The Beck Depression Index (BDI) and the Minnesota Multiphasic Personality Inventory–2 (MMPI-2)[5] were administered to assess emotional functioning before and after a series of EEG asymmetry training sessions designed to increase the difference between right and left alpha magnitude.[6] Adult clinical interpretations of the MMPI-2 were computer generated by the National Computer Center.

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. Subjects were taught to meet a hand warming criterion of 95°F. 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 1-hr-long sessions which consisted of approximately 50% brain wave biofeedback followed by 50% psychotherapy. During biofeedback, scalp sites F3 and F4, referenced to CZ, were recorded. Impedances were 5 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 hypoactivity; therefore, an increase in left frontal activation corresponds to decreased alpha and a positive change in the asymmetry score.[7]

The EEG data for A-score training was recorded on either a four-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-sec 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

[5] The MMPI-2 is the most widely used clinical testing instrument in the United States. It was selected for use in this study because it provided an objective way of measuring ten basic personality factors, including depression.

[6] Our protocol utilized the index $[(R - L)/(R + L)] \times 100$ 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 chapter and Rosenfeld, 1997).

[7] We cannot know from the data whether changes in alpha asymmetry resulted from a decrease in alpha rhythm in the left frontal lead or an increase in alpha rhythm in the right frontal lead, or both changes simultaneously; see Rosenfeld (1997).

TABLE 8.1 Pre- and Post-Alpha Asymmetry Training Measures 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

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

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.

V. CASE STUDIES[8]

A. BOB

Bob is a 37-year-old professional man. He sought therapy several months ago when his marriage was breaking up. He was diagnosed as having a Dysthymic Disorder of moderate severity (DSM-IV: 300.4). His mood was depressed and irritable for most of the day, for more days than not. He frequently had problems with insomnia, and his self esteem was poor. His condition was chronic, first appearing in his adolescence. Bob's father also suffered from dysthymia. Neither Bob nor his father ever suffered from a Major Depressive Episode.

Bob started on a course of the antidepressant medication Zoloft, 75 mg, for depression at the time he began neurotherapy in May 1997. He gradually discontinued his medication after his 18th session using the asymmetry protocol. His proportion of A scores >0 during the first quarter of his treatment was 43%. His average proportion of A scores >0 during the fourth quarter of his treatment was 84% (based on 22 sessions). Post-treatment test scores on the BDI and the MMPI-2 indicate significant reduction in his depression (Table 8.1, Fig. 8.1). Subjectively, he now reports

[8] The names and occupations of the clients discussed throughout this section have been changed to ensure their privacy. The authors wish to thank those patients who allowed their data to be used in this chapter.

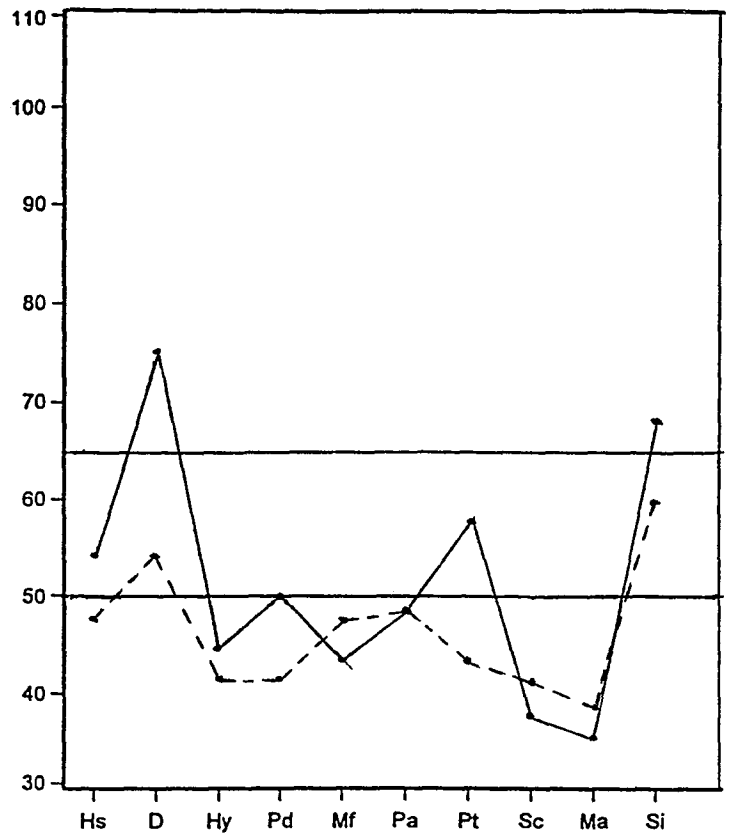

FIGURE 8.1 MMPI-2 basic scale profile pre- and post-asymmetry training for Bob. The clinical scales: Hs, hypochondriasis; D, depression; Hy, conversion hysteria; Pd, psychiatric deviate; Mf, masculinity-femininity; Pa, paranoia; Pt, psychasthenia; Sc, schizophrenia; Ma, hypomania; Si, social interaction. (———, before asymmetry training; --------, after asymmetry training).

feeling good, even though his marital problems are still unresolved. He has become more positively assertive and he is developing a sense of humor.

B. CELIA

Celia is a 34-year-old single teacher. She sought therapy 18 months ago when she was experiencing the onset of a major depressive episode (DSM-IV, 296.21). Her father had symptoms of dysthymia. Her mood was de-

pressed for most of the day, nearly every day. She was experiencing difficulty sleeping, felt a loss of energy, had feelings of worthlessness, and had recurrent suicidal thoughts. She had been taking Prozac, 20 mg daily, for 15 months prior to starting the alpha asymmetry neurofeedback sessions in January 1997. Because she felt less depressed after initiating the neurotherapy sessions, she abruptly stopped taking her medication at the end of February. Her average proportion of *A* scores >0 during the first quarter of her treatment was 57%. This level decreased to 48% during the second quarter, coincident with the discontinuation of her medication. Her proportion of *A* scores >0 during the fourth quarter of her treatment was 80% (based on 32 sessions). Post-treatment test scores on the BDI and the MMPI-2 indicate significant reduction in her depression (Table 8.1, Fig. 8.2). Subjectively she has been experiencing a range of feelings, but no depression. During the sessions she has recalled memories, both happy and sad, from her childhood. These associations were processed during her psychotherapy sessions. She feels more confident in her work and her self-esteem has improved.

C. CATHERINE

Catherine is a 40-year-old divorced woman. She has been a registered nurse for 12 years. She initially sought psychotherapy in the spring of 1993 when she was experiencing severe agitation and depression. She was diagnosed as has having a single episode of major depressive disorder (DSM IV, 296.2). Her symptoms included the presence of depression during most of the day every day, psychomotor agitation, insomnia, weight loss, obsessive thinking, and inability to concentrate. Catherine's mother has a history of depression. Catherine began using Paxil, 20 mg, per day at the onset of therapy. In spite of taking antidepressant medication, Catherine had another less serious episode of depression when one of her parents became depressed and a close friend became seriously ill. Her depression then was characterized by chronic, nonsevere depressive symptoms such as feeling sad and having low self-esteem and low energy. She also gained weight because of overeating. She became reclusive and socially isolated. Her diagnosis was changed to dysthymic depressive disorder (DSM-IV, 300.4).

She was offered neurofeedback treatment soon after the onset of this less severe episode. She initially rejected the offer, but because her feelings were unremitting after 2 years on medication, she agreed to try the alpha asymmetry protocol. She began the first of a series of 36 neurofeedback sessions in June 1996. Her proportion of *A* scores >0 during the first quarter of her treatment was 59%. Her average level of *A* scores >0 during the fourth quarter, which ended in June 1997, was 64%. Post-treatment test scores on the BDI and the MMPI-2 indicated significant improvement in her depression (Table 8.1, Fig. 8.3). During the last two quarters of

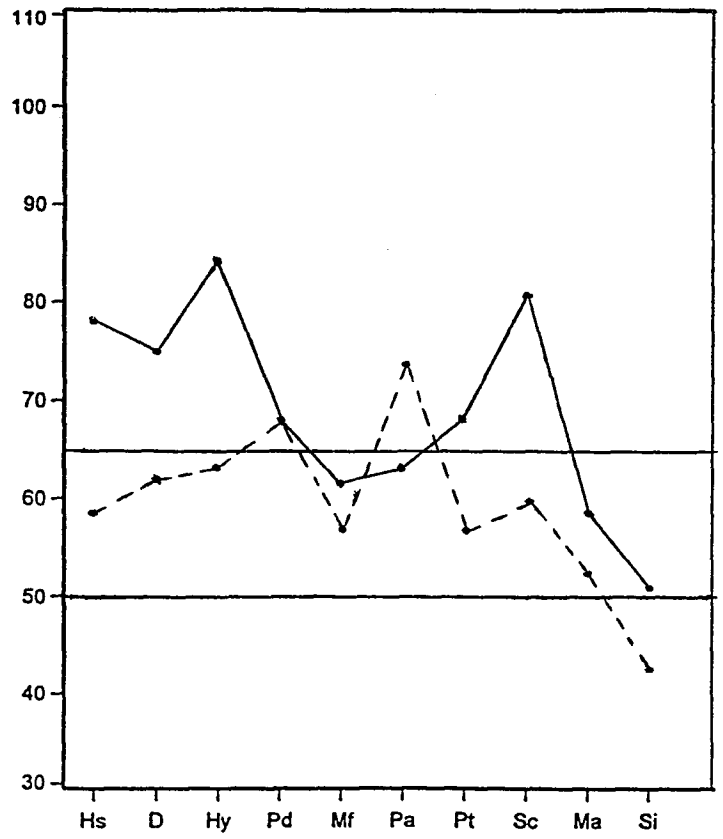

FIGURE 8.2 MMPI-2 basic scale profile pre- and post-asymmetry training for Celia. The clinical scales: Hs, hypochondriasis; D, depression; Hy, conversion hysteria; Pd, psychiatric deviate; Mf, masculinity-femininity; Pa, paranoia; Pt, psychasthenia; Sc, schizophrenia; Ma, hypomania; Si, social interaction. (———, before asymmetry training; --------, after asymmetry training). [Reprinted with permission from "The Clinical Use of an Alpha Asymmetry Protocol in the Neurofeedback Treatment of Depression: Two Case Studies," (1997), Fall/Winter.]

neurofeedback therapy she reported feeling better, and she became interested in increasing her sphere of activities with friends. She joined a dating service and began meeting men. She observed that she felt more flexible and less oppositional. She discontinued treatment when she became engaged and moved to another city. She elected to continue her medication during the transitional period of relocating.

D. ANN ROSE

Ann Rose is a 65-year-old semiretired librarian. She was referred for therapy 12 years ago by a psychiatrist who had been treating her for major

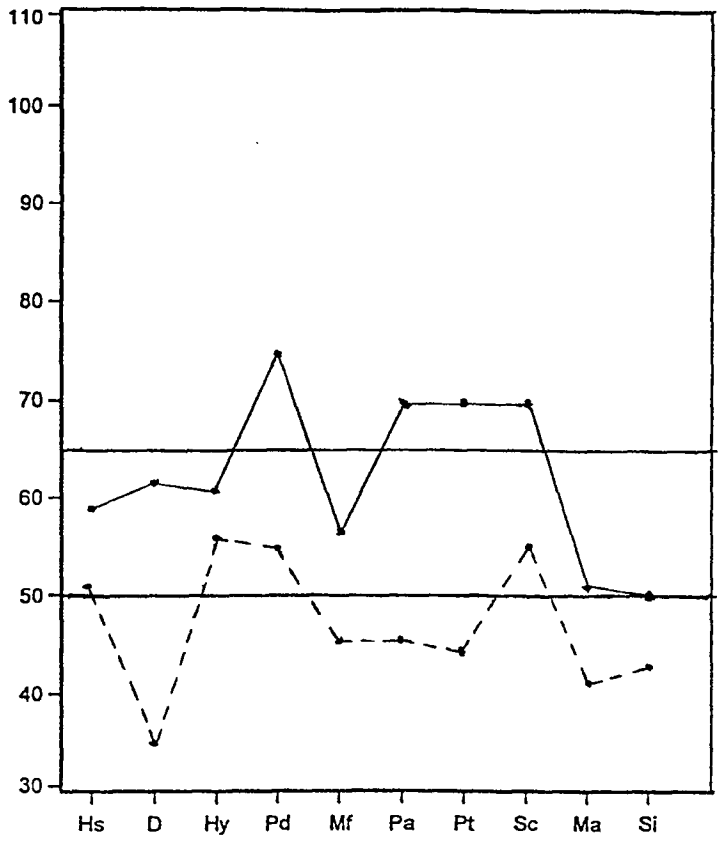

FIGURE 8.3 MMPI-2 basic scale profile pre- and post-asymmetry training for Catherine. The clinical scales: Hs, hypochondriasis; D, depression; Hy, conversion hysteria; Pd, psychiatric deviate; Mf, masculinity-femininity; Pa, paranoia; Pt, psychasthenia; Sc, schizophrenia; Ma, hypomania; Si, social interaction. (———, before asymmetry training; --------, after asymmetry training). [Reprinted with permission from "The Clinical Use of an Alpha Asymmetry Protocol in the Neurofeedback Treatment of Depression: Two Case Studies," (1997), Fall/Winter.]

depressive episodes over a period of 28 years. Her diagnosis at that time was recurrent major depressive disorder of moderate severity (DSM-IV, 296.32). She was seen periodically by Dr. Baehr when she was experiencing a major depressive episode, and she would typically stay in psychotherapy for a short period of time until her symptoms remitted. Her most recent episode occurred in spring of 1993. There was no precipitating event known to have caused this recurrence.

On a daily basis she presented six of the nine criteria for a major depressive episode listed in DSM-IV. Her symptoms included depressed mood during most of the day, a loss of interest and pleasure in activities, significant

weight loss, insomnia, fatigue and loss of energy, indecision, and diminished ability to concentrate. Obsessive thinking was a major personality trait. There was a family history of depression. After 6 months in therapy in which she failed to improve significantly, she was offered 32 neurotherapy sessions for the treatment of depression, using an alpha/theta protocol.[9] She reported some improvement in her feelings after this course of therapy, but her depression returned, particularly when she awoke in the morning. In June 1994 she began the first of 34 sessions using the alpha asymmetry protocol. At that time she also began taking 20 mg of Paxil per day.

Her progress was measured by her quarterly average proportion of *A* scores >0 (Table 8.1), based on a total of 34 sessions.[10] During the third quarter, stress in her life increased when her daughter developed lung cancer, her sister-in-law died, and a close family friend died. Her lowered *A* score in this period apparently reflected her reaction to life's vicissitudes, but she was not experiencing clinical depression. She could distinguish between the emotions generated by depression and those associated with appropriate worry or sadness evoked by situations in her life. Her fourth quarter *A* scores >0 of 67% demonstrated her ability to rebound in a healthier direction. While her sessions formally ended in April 1996, follow up visits in June, July, and November of that year indicated that she was maintaining a proportion of *A* score >0 of 67%. A follow-up visit 1 year later, in June 1997, yielded a proportion of *A* score >0 of 69%.

Ann Rose can no longer be considered depressed. She commented that at times she feels like she may be going into a depression again, but it does not materialize. She also commented that "her mind seems to be functioning better." She feels energetic and outgoing, and while she still tends to worry when things go wrong, she is not as obsessive in her thinking as she was in the past. Post-treatment scores on the BDI and the MMPI-2 indicated significant reduction in her depression (Table 8.1, Fig. 8.4).

E. KATY

Katy[11] was single and 40 years old when she began therapy in the fall of 1993. She worked as a salesperson in a boutique. She initially sought therapy because she experienced alternating manic and depressive symp-

[9] Alpha–theta treatment was selected because previous researchers using the Peniston protocol reported that depression (as measured by the MMPI-2) was alleviated during the course of alpha–theta training (Peniston & Kulkosky, 1990).

[10] The protocol used in this initial use of alpha asymmetry differed from the one used at a later date for the other subjects as it did not produce a score which indicated the percentage of right hemisphere alpha asymmetry. Progress was measured as the mean alpha asymmetry score over the entire session.

[11] Katy was an unusual case and was included in this chapter because of the many complications that occurred during her therapy.

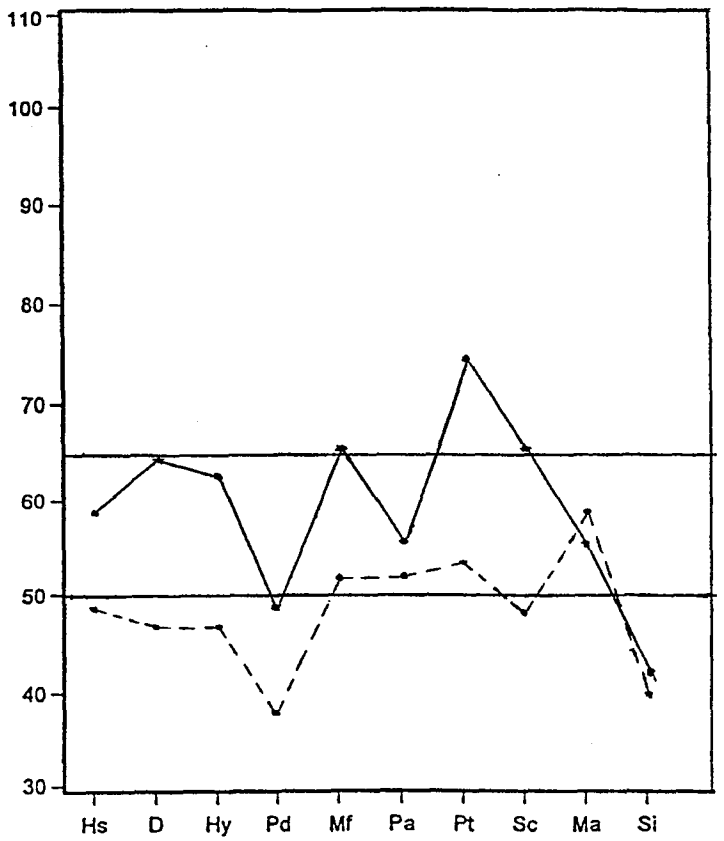

FIGURE 8.4 MMPI-2 basic scale profile pre- and post-asymmetry training for Ann Rose. The clinical scales: Hs, hypochondriasis; D, depression; Hy, conversion hysteria; Pd, psychiatric deviate; Mf, masculinity-femininity; Pa, paranoia; Pt, psychasthenia; Sc, schizophrenia; Ma, hypomania; Si, social interaction. (———, before asymmetry training; --------, after asymmetry training).

toms. She had been taking Prozac for a period of 6 years for depression. She claimed that for several years the medication made her feel "elated and alive," but she was no longer feeling the positive effects. In June 1994 her physician changed her medication to Zoloft. The manic and depressive symptoms emerged again as when she had initially taken Prozac. She was given a diagnosis of bipolar disorder, not otherwise specified (DSM-IV, 296.80) because her symptoms did not meet the minimal duration criteria for a manic episode or a major depressive episode. It was also unclear whether her bipolar symptoms may have been medically induced.

She began a series of 34 neurofeedback sessions in June 1994 that ended

in November 1995. Her progress was measured by dividing the sessions into four periods.[12] Her learning curve was excellent during the first three quarters. She reported feeling good after the sessions in which she had achieved a positive *A* score. While she was not experiencing depression, she missed the highs she had initially obtained with Prozac.

At the end of June 1995, she changed medication again to Effexor. She reacted badly to the new medication, developing flu-like symptoms and edema. During this period of time, which coincided with the fourth quarter, her average *A* score >0 declined. She also elected to stop the neurofeedback sessions. In January her medication was changed once more to a combination of lithium and Prozac. She could not tolerate these medications either, and by June 1996 she had totally discontinued all medications. Periodic assessment of her functioning when on the asymmetry protocol indicated that her level of alpha asymmetry in the right frontal cortices was generally 50% or less. Her BDI also reflected her regression. Her depression score of 25 was higher at the end of treatment than her score of 7 in the beginning of her neurotherapy sessions. In June 1997 she again tried medication (Wellbutrin). She again demonstrated that she could not physically tolerate antidepressants because she developed a skin rash and edema. By the end of July she had discontinued all medication. Clearly this was not a successful case. Her bipolar symptoms have disappeared, however, she remains in a chronic dysthymic condition, and is now considering trying another course of neurotherapy.

F. DEIDRE

Deidre is a single, 47-year-old woman. She has been a special education teacher for 20 years, but has been unable to work since May 1995. Her recent depression began insidiously in April 1994 following an acute viral infection in February 1994, complicated by mild hepatitis and post hepatitis C infection. Her current condition was diagnosed by her physician as post polio syndrome with major depressive episodes. She had been taking Nortriptyline, 50 mg daily, for 2 months prior to beginning neurofeedback treatment, and has continued the medication during the alpha asymmetry training . She met all the criteria for mood disorder due to a general medical condition (DSM-IV, 293.83). These criteria require that the disturbance be the direct physiological consequence of a general medical condition, the disturbance cannot be accounted for by another mental disorder, the disturbance does not occur exclusively during the course of a delirium, and the symptoms cause clinically significant distress or impairment in social,

[12] Katy's case used an early form of the asymmetry protocol that did not provide a percentage score. Instead, a score based on the mean alpha asymmetry score (ALAY) was used to evaluate progress.

occupational, or other important areas of functioning. She also met the full criteria for mood disorder with major depressive-like episodes: depressed mood most of the day, markedly diminished interest or pleasure in all or almost all activities most of the day, nearly every day, significant weight gain, hypersomnia nearly every day, psychomotor retardation nearly every day, fatigue every day, feelings of worthlessness and inappropriate guilt, and diminished ability to think or concentrate or indecisiveness. There is no previous history of depression for this client, and there is no immediate family history of depression.

Deirdre was initially seen for 24 alpha asymmetry training sessions. Her proportion of *A* scores >0 during the first quarter of training was 36%; her proportion of *A* scores >0 during the last quarter of training was 55%. Her BDI scores also showed improvement, decreasing from an initial score of 34 to a post-treatment score of 18 (Table 8.1). While her scores indicate that she was still functioning in the depressed range, she was beginning to feel better. At the completion of the 24 sessions Deirdre stated that she was "now more me than not, although a different me." She still had brief periods of 1–2 hr of feeling sad, but these periods are limited and not long lasting. Her overall energy improved and she believes she is thinking more clearly. Her self-esteem has improved, she is more optimistic about the future, and her guilt has diminished. There was a reemergence of a strong sense of humor. She has recently returned to continue the alpha asymmetry training.

VI. CLINICAL FACTORS ASSOCIATED WITH EEG NEUROFEEDBACK TREATMENT

Five of the six patients in the preceding study were seen in the private psychotherapy practices of Dr. Elsa Baehr and Dr. Rufus Baehr. One patient was the patient of Carolyn Earnest, MSN, RN, CS. Several factors were taken into consideration when EEG neurofeedback was introduced as an adjunct to ongoing psychotherapy. The nature of the relationship between the therapist and patient was, of necessity, altered; that is, a switch was made from a purely talking, "hands-off" therapy, to one where the therapist had to touch the patient to apply electrodes, and where at least 50% of the time, the therapist sat quietly beside the patient who was connected to the EEG machine and was silently responding to a feedback tone. The therapy sessions were conducted in a small lab room rather than in the more spacious therapist's office. Prior to beginning the EEG neurofeedback sessions, the changes in the relationship and the setting were discussed with the patient. The EEG neurofeedback treatment was initiated only after both the therapist and patient felt comfortable with the altered treatment structure.

Some changes likely occurred as a result of the changes in treatment. The more informal atmosphere in the lab, and the physical closeness of the chairs in which the patient and therapist were seated probably created an environment in which the patient felt secure and free to discuss feelings. Also a feeling of alliance likely was created because both patient and therapist were involved in the success of the EEG neurofeedback process. The environment is a nurturing one in which the unconscious dependency needs of the patient can be met in a nonthreatening way. These factors need to be taken into account when evaluating the progress of the patient being trained on the alpha asymmetry protocol. Perhaps the emotional environment reinforces and facilitates the EEG changes in alpha asymmetry as they are occurring. Studies need to be done to determine whether the same degree of improvement in mood would occur in a lab setting that was purely electrophysiological and the person applying the electrodes was an objective technician rather than a therapist.

VII. NEGATIVE FACTORS IN THE CLINICAL SITUATION

The patients who agreed to try the EEG neurofeedback also consented to allow their data to be used for research. This situation became an issue for one patient (not a participant in the present study) who was feeling stressed because of many outside demands on her time. She was attending graduate school part time, and was working as an administrator in a corporation. Her job required her to travel frequently, which interfered with the continuity of the EEG neurofeedback treatment. Although she had initially been successful in responding to the asymmetry protocol, she was unable to maintain positive scores. When it was suggested that she try two sessions in one day to "get back on track" she agreed; however, her scores consistently fell within the negative alpha asymmetry range. In a discussion following the sessions she admitted that she felt resentful in coming to therapy but did so to please the therapist. It was also revealed that she felt the therapist was more interested in collecting data than in her problems.

Two patients who had agreed to try the EEG neurofeedback became impatient after one or two sessions and expressed their need for "talking therapy" before engaging in further EEG treatments. It is of course crucial to the success of any therapy to be sensitive to the needs of the patient. In the EEG neurofeedback treatment this can be accomplished very easily by watching for *A*-score changes in the negative direction during a therapy session,[13] and then processing the thoughts that have occurred during the

[13] In this study the "trend" display in the Biolex program was useful because it allows the therapist to observe changes in asymmetry as they occur.

treatment. This can be done by interrupting a session when the asymmetry becomes negative, or by doing a briefing after the session.

VIII. MEDICATION AND THE ASYMMETRY PROTOCOL

Most patients were using antidepressant medication at the beginning of the EEG neurotherapy and continued it for varying amounts of time.[14] The effect of the medication on the asymmetry protocol is unknown and needs to be studied. It is apparent that the medication did not prevent the *A* score from slipping into the negative range when the patient was responding to an emotionally disturbing thought or situation. One patient, Katy, a long-time Prozac user who had achieved a consistently positive asymmetry score, began to develop a severe reaction to her medication. She developed edema and complained of flu-like symptoms. Changes in medication made her situation worse. Her *A* score was erratic and declined overall in the fourth quarter. She agreed to slowly terminate all medication, and after a month had passed she began to feel better physically, but her mood remained dysphoric.

Another patient, Celia, reported on earlier, had been on Prozac for more than 2 years when she began EEG neurotherapy. She abruptly stopped taking medication. Her asymmetry score, which had been consistently in the positive range, dropped into the negative range, and remained low for several weeks. She rebounded and has maintained positive scores since that time. She is no longer depressed.

Ann Rose, also reported on earlier in this chapter, gradually decreased her medication (Paxil) after 6 months of EEG neurotherapy. After 1 month she was completely off her antidepressant.

Bob, who started a low dose of Zoloft at the beginning of asymmetry treatment, has totally cut out his medication after 22 EEG neurofeedback sessions. While he experienced slight withdrawal feelings, he maintained his level of asymmetry percentage and did not become depressed.

In summary, three of the six patients reported on in this chapter successfully discontinued their medication before the end of the fourth quarter of their treatment. Their proportion of *A* scores remained stable.

IX. DISCUSSION

In this chapter we have described how theories of emotion and anterior cerebral asymmetry led to an investigation of an alternative way to treat

[14] The authors wish to thank Dr. Miepje DeVryer for her cooperation and assistance.

depression. A striking finding was that differences in frontal brain asymmetry discriminated populations of depressed and nondepressed subjects (Henriques & Davidson, 1990; Gotlib *et al.,* in press). It was also learned that normal subjects could be trained to modify their brain waves by changing their frontal alpha asymmetry (Rosenfeld *et al.,* 1995). In addition, it was demonstrated that there was a relationship between daily changes in frontal alpha asymmetry and changes in mood (Rosenfeld *et al.,* 1996).

The transition was made from the theoretical foundations and experimental studies to the practical applications when we trained depressed persons to change their frontal alpha asymmetry to resemble the asymmetry pattern found in nondepressed persons. Some depressed patients presented earlier in this chapter appeared to benefit from this alpha asymmetry training, as measured not only by their subjective feelings, but also by their post-training scores on the MMPI-2 and the BDI. There appears to have been general improvement in the MMPI-2 personality profiles for four of the subjects on which we had MMPI-2 data (Figs. 8.1–8.4). Clinically we observed that our patients were generally less obsessive and more positive in their thinking. Finally, along with our colleague Carolyn Earnest, we found that they were displaying a sense of humor.

A comparison of the patients' pre- and post-MMPI-2 depression scales indicates 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 8.1).[15] The SEM is based on the standard deviation of the sampling distribution. The standard deviations used in this study were derived from a sample of 1184 white females and a sample of 933 white males (Hathaway & McKinley, 1989, p. 105). Interpretation of change in a patient's profile at the retest should not be made unless the differences exceed the standard error of measurement, and preferably are two times the SEM for conservation personality appraisal (Butcher, 1990, p. 11).

Five of the six subjects scored above 9 on the BDI in the pretest, while four of the six scored below 9 in the post-test. (Scores above 9 on the BDI commonly are considered to be in the depressed range.)

While we may wish to view this asymmetry protocol as a major innovative treatment for mood disorders, it is apparent that it does not work for everyone. For example, in the case of bipolar depression presented earlier, improvement occurred in terms of eliminating mood swings, but the patient remained in a dysphoric state at the end of the treatment. This case also

[15] The SEM is based on the standard deviation of the sampling distribution. The standard deviations used in this study were derived from a sample of 1184 while females and a sample of 933 white males. (Hathaway and McKinley, 1989, pg. 105. Interpretation of change in a patient's profile at the retest should not be made unless the differences exceed the standard error of measurement, and preferably are two times the SEM for conservation personality appraisal. (Butcher, 1990, p. 11).

was complicated by reactions to psychotropic medications. Further study is needed to determine the type of mood disorders that are most amenable to treatment. At this time we also view neurofeedback as an adjunct to ongoing psychotherapy, and not as a "stand-alone" treatment. The lab setting where the neurofeedback treatment occurs, and the alliance with the therapist also may be important factors in the treatment situation. Some question is raised as to whether the positive effects we have observed would also occur in a lab setting where a therapist was not present.

Taking these factors into account, we feel that the crucial next step for research is to demonstrate that appropriate control cases do not improve clinically as much as cases given the specific asymmetry protocol. We have preliminary evidence along this line, noting a case in which the asymmetry protocol, but not the alpha–theta protocol was helpful. In another case, not reported in this chapter, a patient of Dr. Elsa Baehr was seen for 50 neurofeedback sessions with a different protocol to improve cognitive functioning after a head injury. Although the treatment was successful, the patient was still depressed at the end of the neurotherapy sessions as measured by the MMPI-2 and the BDI. She has returned for treatment of her depression using the asymmetry protocol and is currently showing progress.

This novel approach to the treatment of depression is in its infancy. We have reported here on the first clinical uses of the alpha asymmetry protocol for treatment of depression. Based on our initial findings we feel that alpha asymmetry neurofeedback is a promising alternative adjunctive treatment for mood disorders.

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