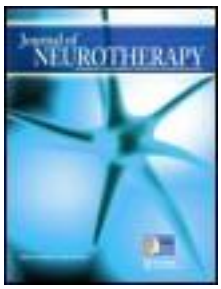


# Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience


# TOVA Results Following Inter-Hemispheric Bipolar EEG Training


J. A. Putman MA and MS [a] , S. F. Othmer BA [b] , S. Othmer PhD [b c] & V. E. Pollock PhD [b]

[a] EEG Institute , Suite 305, 22020 Clarendon Street, Woodland Hills, CA, 91367

[b] EEG Institute

[c] The Brian Othmer Foundation



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# TOVA Results Following Inter-Hemispheric Bipolar EEG Training

J. A. Putman, MA, MS
S. F. Othmer, BA
S. Othmer, PhD
V. E. Pollock, PhD

**ABSTRACT.** *Introduction.* This study examines recovery of attentional measures among a heterogeneous group of clients in a pre-and post-comparison using inter-hemispheric EEG training at homologous sites. A continuous performance test was used as an outcome measure. The client population was divided into three categories: (a) primarily attentional deficits, (b) primarily psychological complaints, and (c) both.

*Method.* Neurofeedback protocols included T3-T4, Fp1-Fp2, F3-F4, C3-C4 and P3-P4. A wide range of reward frequencies was used, and these were individually selected to optimize the subjective experience of the training. Participants were 44 males and females, 7 to 62 years old, who underwent treatment for a variety of clinical complaints. Dependent variables were derived from a continuous performance test, the Test of Variables of Attention (TOVA), which was administered prior to EEG training and 20 to 25 sessions thereafter.

*Results.* After EEG training a clear trend towards improvement on the

J. A. Putman is a neurofeedback practitioner and researcher at the EEG Institute, Suite 305, 22020 Clarendon Street, Woodland Hills, CA 91367.

S. F. Othmer is Clinical Director of the EEG Institute where she is a neurofeedback practitioner and instructor in neurofeedback courses for professionals.

S. Othmer is Chief Scientist of the EEG Institute and President of The Brian Othmer Foundation, a non-profit organization for research, education and clinical services in neurofeedback.

V. E. Pollock is a psychologist and Director of Research at the EEG Institute.

Address correspondence to: J. A. Putman at the above address (E-mail: JPutman@eeginstitute.com).

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impulsivity, inattention, and variability scales of the TOVA was evident. Participants with normal pre-training scores showed no deterioration in their performance, indicating that homologous site inter-hemispheric EEG training had no deleterious effect on attention. In addition reaction time was predominately in the normal range for this population and remained unchanged following training.

*Conclusion*. Normalization of attentional variables was observed following training irrespective of the primary clinical complaint. These results suggest that inter-hemispheric training at homologous sites provides another "generic" EEG biofeedback protocol option for addressing attentional deficits. Inter-hemispheric training likely serves as a general challenge to the regulation of cerebral timing, phase, and coherence relationships. Such a challenge may result in more effective regulation of cerebral networks, irrespective of whether these are involved in attentional or affective regulation.



**KEYWORDS.** EEG, neurofeedback, attention, inter-hemispheric, bipolar, TOVA

## *INTRODUCTION*

Compared to other neurofeedback protocols, evidence of the efficacy of inter-hemispheric training is not abundant. Results of neurofeedback attesting to the potential for inter-hemispheric training were first reported on incarcerated violent offenders (Quirk, 1996). Quirk used a composite EEG neurofeedback and galvanic skin response biofeedback training procedure. The EEG was trained inter-hemispherically, with electrode placements at C3-C4, using a 12 to 15 Hz reward band. Over the 18-month follow up period, those who received more than 33 half-hour training sessions had recidivism rates of 20%, as compared to those who received no treatment, who exhibited recidivism rates of 65%.

The context of this work has emerged from years of clinical impressions that inter-hemispheric training enhances cortical stability generally, and that it normalizes regulation of central arousal, attention, and affect. Specifically, we propose as a testable hypothesis that inter-hemispheric training normalizes (or optimizes) function on measures of attention irrespective of diagnostic presentation. To assess the hypothesis, standardized measures from a test of sustained attention, the Test of Variables of Attention

(TOVA, Leark, Dupuy, Greenberg, Corman, and Kindschi, 2000) were obtained before and after a series of treatment sessions in which individuals underwent inter-hemispheric neurofeedback training in a single-channel approach. This study describes the overall group results and individual attentional performance before and after neurofeedback treatment.

Because remediation of attention deficits was not specifically targeted in each case, the evaluation of the present data form a reasonable basis for assessing the hypothesis that inter-hemispheric training might prove generally efficacious in the attentional domain. Our *a priori* hypothesis was that attention deficits may serve as a reasonably good index of the quality of self-regulation. Such deficits accompany many conditions, and they tend to remediate along with the resolution of other clinical symptoms.

We were interested in the possibility that subgroups with different presenting complaints might differ in the degree to which attentional functions were altered after inter-hemispheric training, as compared to before neurofeedback. Therefore, for descriptive purposes we present results that compare three subgroups on the basis of attentional measures before and after inter-hemispheric training: (a) a subgroup characterized by prominent attentional deficits, (b) a subgroup characterized by both attentional deficits as well as mood dysregulation, and (c) a subgroup characterized by primarily mood dysregulation.

## *METHOD*

### *Participants*

Over 200 cases, consisting of individuals who sought treatment for a variety of symptoms were reviewed to identify those that satisfied three inclusion criteria: (a) interhemispheric training was the sole neurofeedback treatment administered, (b) a minimum of 20 treatment sessions were completed, and (c) pre- and post-treatment measures derived from the TOVA were obtained.

A total of 44 individuals (17 females, 27 males) aged 6 to 62 years ($M$ = 30.5, $SD$ = 20.5) fulfilled all three criteria for inclusion in the study. Subgroup 1 (n = 12) consists of participants who identified attentional deficits (AD) as their primary symptom. Subgroup 2 (n = 20) consists of participants who reported attentional deficits plus various types of mood dysregulations (MD) such as depression, anxiety, compulsive overeating, panic, PTSD, anger, autism, bipolar disorder, and head injury. Subgroup 3 (n = 12) consists of participants who did not report attentional deficits, but who did report various mood dysregulations. Table 1 shows relevant case symptomatology.

TABLE 1. Symptoms, Medication Status and Neurofeedback Protocols.

| Symptoms | Meds., Initial | Meds., Changes | Sites | Reward Freq.(Hz) |
|---|---|---|---|---|
| 1. AD, depression, anger | None | Same | T3-T4, Fp1-Fp2 | 11-14, 9-12 |
| 2. Anxiety | None | Same | T3-T4 | 12-15 |
| 3. Depression | Atavan, Beta Blkr | Same | T3-T4, Fp1-Fp2, F3-F4 | 10.5-13.5, 8.5-11.5, 8.5-11.5 |
| 4. AD | | Same | T3-T4, Fp1-Fp2 | 12-15Hz, 10-13 |
| 5. AD, depression | None | Same | P3-P4, T3-T4 | 3-6, 6-9 |
| 6. AD | None | Same | T3-T4, Fp1-Fp2 | 9.5-12.5, 7.5-10.5 |
| 7. Anxiety | Celexa | Same | T3-T4, Fp1-Fp2 | 9-12, 7-10 |
| 8. Autism, AD, anxiety | Xyprexa | Added neurontin | T3-T4, F7-F8, Fp1-Fp2 | 7-10, 5-8, 5-8 |
| 9. AD, hyperactivity | None | Same | T3-T4 | 12-15 |
| 10. Anxiety, AD | None | Same | T3-T4 | 12-15 |
| 11. AD, anxiety | Remeron, Restoril | Same | T3-T4 | 12-15 |
| 12. Depression | None | Same | T3-T4, Fp1-Fp2 | 11.5-14.5, 9.5-12.5 |
| 13. Bipolar disorder | Atavan | Same | C4-C3, T3-T4, F3-F4 | 9-12, 9-12, 7-10 |
| 14. Depression, AD | GH suppressant | Same | T3-T4, Fp1-Fp2 | 8.5-11.5, 6.5-9.5 |
| 15. AD, depression | None | Same | T3-T4, Fp1-Fp2 | 6-9, 5-8 |
| 16. Anxiety | None | Same | T3-T4, F3-F4 | 15-18, 13-16 |
| 17. Anxiety, AD | None | Same | T3-T4 | 7-10 |
| 18. AD, depression | Wellbutrin | Same | T3-T4 | 13-16 |
| 19. AD | None | Same | T3-T4, F3-F4 | 9.5-12.5, 7.5-10.5 |
| 20. AD, anxiety | None | Same | T3-T4, Fp1-Fp2 | 18-21, 16-19 |
| 21. AD, overeating | None | Same | T3-T4, Fp1-Fp2 | 8-11, 6-9 |
| 22. Chronic pain, depression | None | Same | T3-T4, Fp1-Fp2, P3-P4 | 11.5-14.5, 9.5-12.5, 7.5-10.5 |
| 23. Depression | Synthroid | Same | T3-T4 | 12-15 |
| 24. Anxiety | None | Same | T3-T4, Fp1-Fp2 | 4.5-7.5, 2.5-5.5 |
| 25. AD, head injury | None | Same | T3-T4 | 12.5-15.5 |
| 26. Depression | None | Same | T3-T4 | 4-7 |
| 27. AD | Aderall | Same | T3-T4, Fp1-Fp2 | 10-13, 8-11 |
| 28. AD, overeating | Lipitor | Same | C3-C4, Fp1-Fp2 | 2.5-5.5, 0.5- 3.5 |
| 29. Anxiety | None | Same | P3-P4, O1-O2, F3-F4 | 7.5-10.5, 7.5-10.5, 9.5-12.5 |
| 30. ADD | None | Same | T3-T4 | 12-15 |
| 31. Depression, AD | Vioxx | Same | P3-P4, Fp1-Fp2 | 0-3, 0-3 |
| 32. Anxiety, AD depression | None | Same | T3-T4, F3-F4, Fp1-Fp2 | 10.5-13.5, 8.5-11.5, 8.5-11.5 |
| 33. AD | None | Same | T3-T4 | 12.5-15.5 |
| 34. AD | None | Same | T3-T4, F3-F4 | 7-10, 5-8 |
| 35. AD, hyperactivity | Concerta | Same | T3-T4, Fp1-Fp2 | 6-9, 4-7 |

| Symptoms | Meds., Initial | Meds., Changes | Sites | Reward Freq.(Hz) |
|---|---|---|---|---|
| 36. AD, hyperactivity | None | Same | T3-T4 | 9-12 |
| 37. Chronic pain, depression | Prozac, Elavil | Reduced Prozac & Elavil | T3-T4, P3-P4, Fp1-Fp2 | 3.5-6.5, 0-3, 2-5 |
| 38. AD, Bipolar disorder | None | Same | T3-T4, F3-F4 | 6.5-9.5, 4.5-7.5 |
| 39. Anxiety | None | Same | T3-T4, Fp1-Fp2 | 9.5-12.5, 7.5-10.5 |
| 40. Hyperactivity | None | Same | T3-T4, Fp1-Fp2 | 7.5-10.5, 5.5-8.5 |
| 41. AD | None | Same | T3-T4, Fp1-Fp2, P3-P4 | 1-4, 0-3, 0-3 |
| 42. AD, depression | Neurontin, Lexapro | Same | T3-T4, P3-P4, Fp1-Fp2 | 7-10, 3-7, 5-8 |
| 43. AD, anxiety, PTSD | None | Same | C3-C4, Fp1-Fp2, F3-F4 | 0-3, 0-3, 0-3 |
| 44. AD, anxiety | None | Same | T3-T4, Fp1-Fp2 | 8-11, 6-9 |

### *Instrumentation*

NeuroCybernetics instrumentation was used in all of the selected cases. This system uses infinite impulse response (IIR) digital filtering with elliptic filters of two poles, with analog signal gain set at 10,000 followed by digital conversion with 12-bit resolution. Instrument input impedance for each of the two channels was nominally one million meg-ohms. Sampling rate was 160 per second. The raw EEG trace and the three filtered waveforms were displayed in a continuous scrolling fashion for monitoring by the therapist. Upon digital filtering, the signal was then sent to a second computer where it was mapped into different features of a video game for viewing by the participant. The second screen displayed variations on a "box lights" game wherein each filtered trace was represented by a box-like image. Size or movement of each of the three box images varied in direct proportion to the amplitude generated in each frequency band. When threshold criterion was met in all three bands simultaneously and sustained for more than 0.5 seconds, the participant heard a tone. The visual box light display provides reinforcement that relates to the ebb and flow of EEG amplitudes in the bands around their respective thresholds, and the tone serves as an additional reinforcement.

### *Attention Measures*

The computerized version of the TOVA involves a brief (100 msec) visual presentation of one of two patterns every two seconds. One pattern

is designated as the "target" and the other as the "non-target," and the distinction relies merely on up-down discrimination. The participant is instructed to press a micro-switch as quickly as possible when presented with the target, and to refrain from pressing when viewing the non-target. Test duration is 22.5 minutes. The purpose of the TOVA is to assess sustained attention via impulse control, reaction time, variability of reaction time, omission errors and commission errors (Leark, Dupuy, Greenberg, Corman, & Kindschi, 2000).

In the present report, TOVAs were administered prior to neurofeedback training, and again after the first 20 to 25 treatment sessions. Many different factors can elicit transient attention deficits. These include but are not limited to sleep deprivation, situational stressors, diurnal effects, and low blood sugar. In order to minimize diurnal effects, the acquisition of TOVAs occurred prior to 1:00 p.m. in accordance with TOVA administration procedures. We used standard TOVA scores *(M* = 100, *SD* = 15) to index: (a) impulsivity (i.e., commission errors), (b) inattention (i.e., omission errors), (c) reaction time (in msec), and (d) reaction time variabilityto measure attention. Hence, these four measures serve as dependent variables in statistical analyses. Participants who were on prescription stimulants were asked to refrain from taking them the day the TOVA was tested and retested.

### *Neurofeedback*

All electrode sites were placed according to the International 10/20 system of electrode placement. The most frequently used montages were T3-T4 and/or Fp1-Fp2, but five other montages were also used selectively: F3-F4, P3-P4, C3-C4, F7-F8 and O1-O2. (See Table 1.) In addition, F7-F8 was used with one patient due to a rather prominent EEG abnormality detected via the QEEG.

A wide range of reward frequency bands was employed, each of 3 Hz width. The initial reward frequency settings were generally 12-15Hz, with adjustments made in-session to optimize the person's subjective response to the training. High frequency inhibition was in the 22-30 Hz band with low frequency inhibition in one of the following: 2-7 Hz, 4-7Hz, or 8-11 Hz. Low frequency inhibition coupled with mid range frequency reward has been used with success in treating both seizures (Sterman & Friar, 1972; Sterman & Macdonald, 1978) and ADD (Lubar & Shouse, 1976; Lubar & Lubar 1984; Kaiser & Othmer 2000). This early work utilized reinforcement at 12-15Hz with either left-hemisphere

or midline placements. Excess alpha activity (asymmetry in the anterior region) has been associated with mood disorders and, as such, was appropriately inhibited when training was preformed at frontal and pre-frontal locations (Baehr, Rosenfeld, & Baehr, 2001). High frequency inhibition (22-30 Hz) has traditionally been performed for the purpose of preventing the reward of EMG activity.

Electrode placement was chosen by the clinician based on the individual's symptoms. The goal was to ameliorate the attentional or psychological symptoms as quickly as possible for each individual case. While training location was assigned on the basis of the gross features of presenting symptoms (e.g., T3-T4 for mood stabilization, Fp1-Fp2 for impulse control, and P3-P4 for physical calming), reward frequency alterations were dictated by intra-and inter-session responses. For example, if a person became slightly more agitated or anxious (or exhibited increased restlessness, generalized fear, delayed sleep onset, or emotional coldness or disconnectedness) the reward frequency was reduced in 0.5 Hz increments. Conversely, if individuals responded with increased sedation or depressive symptoms (e.g., grogginess or sadness), the reward frequency was increased in 0.5 Hz increments. Within session, a state of optimally alert focus and euthymic mood was sought through frequency adjustments as well.

Generous reward criteria were used, with the threshold on the reward band set to achieve success some 75% of the time. Low frequency inhibit thresholds were engaged approximately 15 to 20% of the time, and high-frequency thresholds were exceeded at most 10% of the time. Thresholds were actively maintained near these criteria, a process referred to as dynamic thresholding. With such a choice of thresholds, the reward tone would be given nominally 50% of the time. As shown in Table 1, various protocols were used in succession within a single session depending on the patient's array of symptoms. Training periods were generally 30 minutes long. Frequency of training varied from two to five sessions per week.

There were few medication changes in this group of subjects. The reason for this is that medication changes typically do not occur within the first 20 sessions of training. Such changes, if any, are predicated on tangible evidence of improved functioning such as that provided by the post-training TOVA. It is, therefore, somewhat unusual for medication changes to occur during the initial stage of neurofeedback training. Such was the case with this set of subjects.

### *Statistical Analyses*

Standard parametric techniques were used based on analyses for paired samples. Dependent variables consisted of TOVA measures, quantified in standard score units. Over 25% of the cases had scores in the normal range, based upon initial evaluation by the TOVA. The initial analyses of theoretical interest consist of four separate analyses of variance (ANOVAs). To provide conservative statistical tests of our theoretical hypotheses, Bonferroni correction was used in these initial analyses. Specifically, the Type 1 error rate for each ANOVA was $p = .0125$, so that the overall Type 1 error did not exceed $p = .05$. *Post hoc* and additional statistical tests conform to procedures routinely used throughout the scientific community (Abelson, 1995).

## *RESULTS*

The means and standard deviations for all four dependent variables before and after inter-hemispheric neurofeedback training are shown in Figure 1 for all 44 participants. Analysis of variance (ANOVAs) for paired samples wherein each participant served as his or her own control yielded statistically significant changes for inattention, impulsivity and variability (Table 2). There were no significant changes in reaction time for the group.

Those with the greatest degree of initial impairment exhibited the most substantial change (Figures 2, 3 and 5). Reaction time was largely in the normal range on initial testing, and those whose standard score was well above 100 generally maintained their scores following the neurofeedback (Figure 4).

Generally, the attentional problems indexed by the TOVA were resolved following neurofeedback in all three subgroups (Figures 6 and 7 and Tables 3 and 4). Subjective reports indicated that among participants in the mood dysregulation subgroup, symptoms improved after neurofeedback treatment also. It could not be unambiguously established whether changes in mood and attentional status were independent or concurrent.

## *DISCUSSION*

These results suggest that inter-hemispheric training using bipolar placement at homologous sites is an effective treatment protocol for im-

FIGURE 1. Standard scores on the TOVA before and after neurofeedback (n = 44). The scales are: Omission (inattention), Commission (impulsivity), Response Time and Variability (variability of response time).

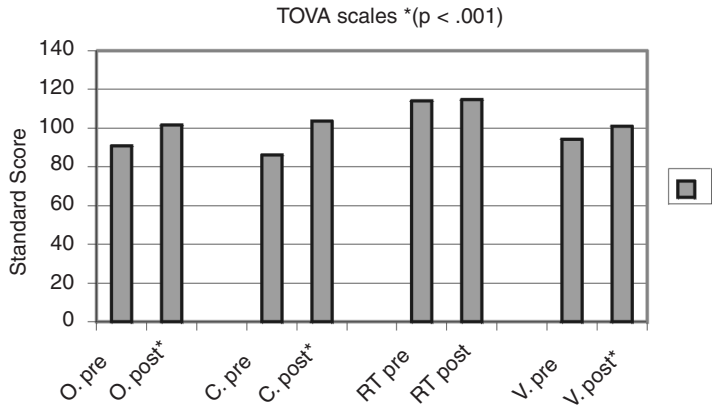



TABLE 2. Means (M) and Standard Deviations (SD) on the TOVA before and after neurofeedback (n = 44)

| Scale | *M* (pre) | *M* (post) | *SD* (pre) | *SD* (post) | *F* (143) | *p* |
|---|---|---|---|---|---|---|
| Omission | 90.7 | 101.5 | 20.3 | 11.6 | 16.81 | < .001 |
| Commission | 86.0 | 103.7 | 20.6 | 10.3 | 29.81 | < .001 |
| Response Time | 114.0 | 114.6 | 21.8 | 16.5 | .08 | N S |
| Variability | 94.3 | 101 | 17.4 | 16.8 | 11.56 | < .001 |

proving attention-related deficits. This training did not seem to impact individuals with normal TOVAs in a negative or adverse way. Since attention deficits are frequently co-morbid with other forms of symptomatology (e.g., head injury, autism, depression and anxiety), addressing the primary symptom(s) will often lead to a resolution of attention-related problems as a corollary effect. Training at T3-T4 appeared to be effective in alleviating or reducing instabilities in mood state and physiological regulation, whereas training at Fp1-Fp2 appeared to improve executive function, attention and impulse control, and reduce obsessive and compulsive symptoms. Other protocol locations were used on a case-by-case basis to address particular individual issues (e.g., P3-P4 appeared to be generally helpful in reducing hyperactivity and enhancing body and spatial awareness; F3-F4 appeared to be helpful for increasing motiva-

FIGURE 2. Omission errors (inattention) before and after neurofeedback (n = 44). Subjects were rank-ordered in the plot according to initial score.

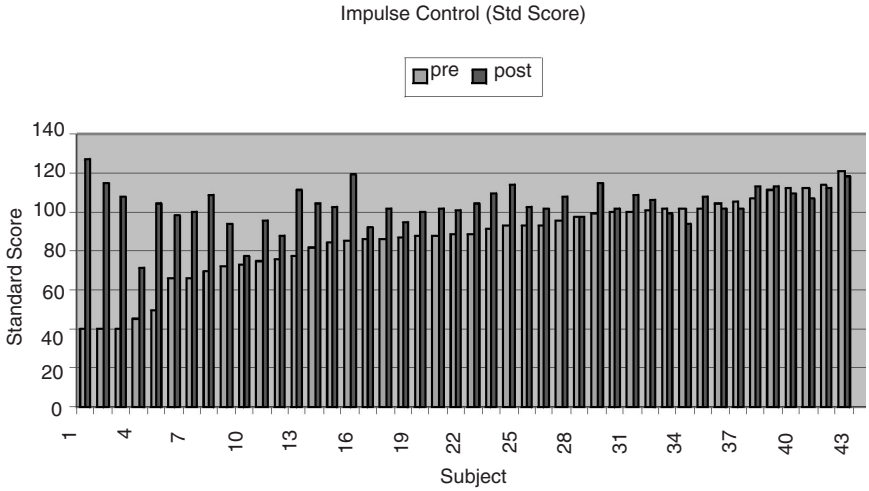
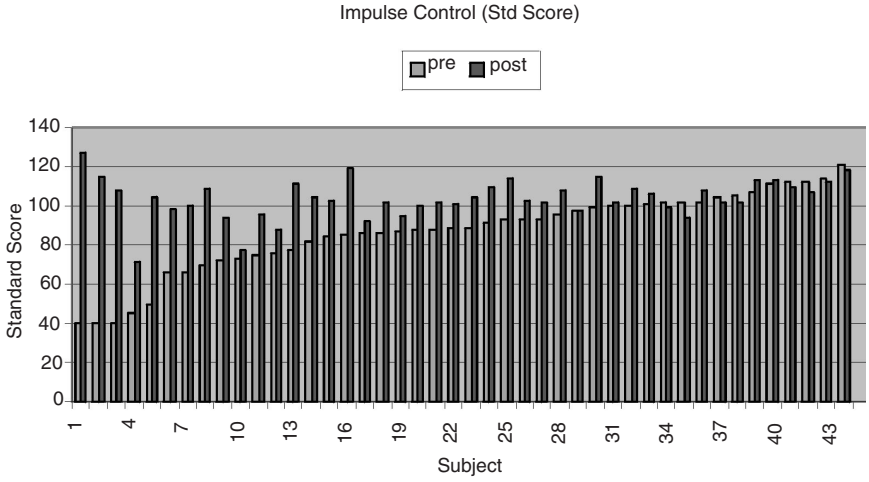



FIGURE 3. Commission errors (impulsivity) before and after neurofeedback (n = 44) from Figure 2 subjects. The figure illustrates a trend toward normalization.




tion and alleviating some forms of depression). Figures 2 and 3 show a trend towards normalization on measures of inattention and impulsivity where those with the poorest scores at initial evaluation showed the greatest degree of improvement at retest. The results on the impulsivity scale in (Figure 7 and Table 4) suggest that the severity of impairment before

FIGURE 4. Reaction time before and after neurofeedback (n = 44).

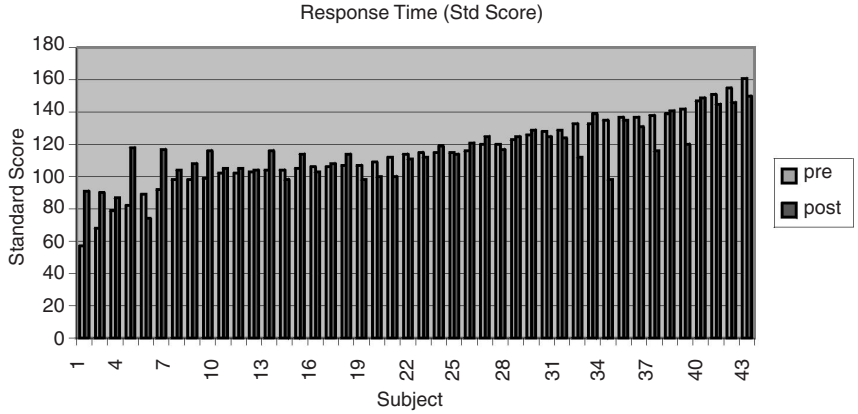
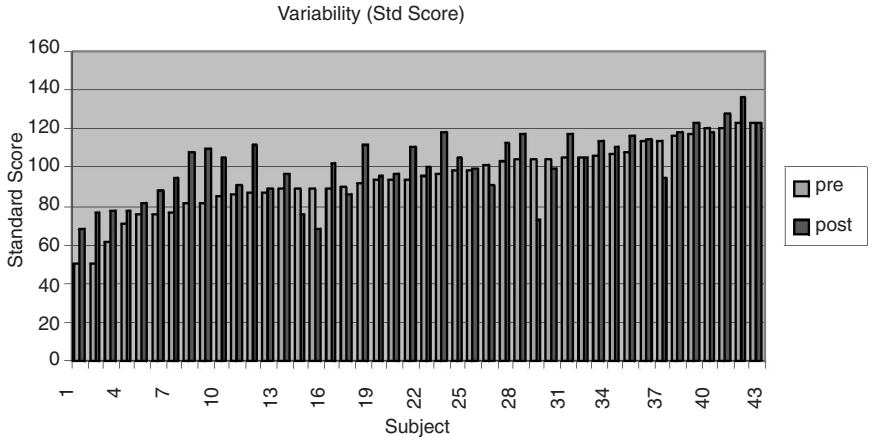



FIGURE 5. Variability before and after neurofeedback (n = 44).




treatment is greater when attentional deficits and affective disturbances are co-morbid.

These results suggest that interhemispheric neurofeedback intervention changes scores on a measure of attention in individuals whose treatment goal was to elicit mood stability and euthymia. Whether or not that implies a causal connection or common mechanism is not clear. There

FIGURE 6. Pre- and post-standard scores on omission errors (inattention) in the three subgroups. Means and standard deviation are given in Table 3.

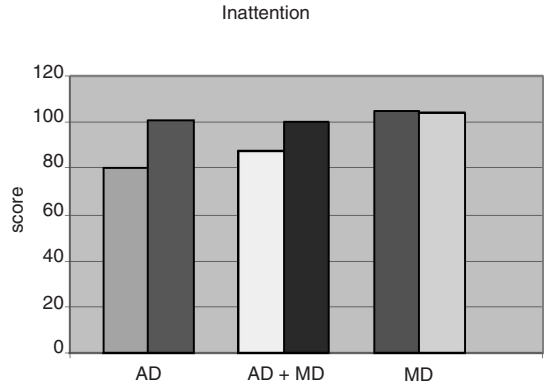



FIGURE 7. Pre- and post-standard scores on commission errors (impulsivity) in the three subgroups. Means and standard deviations are given in Table 4.

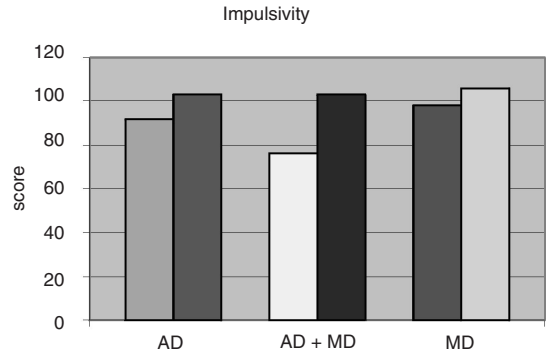



TABLE 3

| Group | *M* (pre) | *M* (post) | *SD* (pre) | *SD* (post) | *F* | *p* |
|---|---|---|---|---|---|---|
| 1. AD | 79.9 | 100.8 | 21.3 | 13.6 | F(1,11) = 14.73 | < .003 |
| 2. AD+MD | 87.2 | 100.1 | 20.4 | 12.7 | F(1,19) = 12.6 | < .002 |
| 3. MD | 105 | 103.8 | 5.9 | 8.1 | F(1,11 ) = .652 | N S |

TABLE 4

| Group | *M* (pre) | *M* (post) | *SD* (pre) | *SD* (post) | *F* | *p* |
|---|---|---|---|---|---|---|
| 1. AD | 91.8 | 103.0 | 15.7 | 9.7 | F(1,11) = 7.53 | < .019 |
| 2. AD+MD | 76.8 | 103.0 | 24.0 | 12.7 | F(1,19) = 19.67 | < .001 |
| 3. MD | 98.1 | 106.1 | 9.6 | 5.5 | F(1,11) = 12.21 | < .005 |

were substantial differences between the protocols for the three subgroups, but examination shows that the mean central frequency of the reward band at T3-T4 (the site common to all but three subjects) was approximately the same for all three subgroups: AD, Mean Central Reward Frequency (CRF) = 10 Hz, SD = 3.4; AD+MD, mean CRF = 10.5 Hz, SD = 3.8; MD, mean CRF = 11 Hz, SD = 3.1.

All of the displayed data met time of day TOVA test criterion-specifically, early a.m. to early p.m. (Leark et al., 2000). Generally, the pre-test was given in the morning hours and the post-test was given later in the morning or in the early afternoon. Research has demonstrated diurnal effects on relevant EEG variables, which may reflect changes in vigilance. Low-frequency activity (5-11 Hz) can increase dramatically in the parietal cortex between the hours of 10 a.m. and 12 noon and remain somewhat elevated throughout most of the afternoon (Kaiser & Sterman, 1994). These alpha and theta excesses presumably correlate negatively with attentional capability. The reaction time plot in Figure 4 shows that scores in the normal range at the start of training remained essentially unchanged following training. Since standard score is sensitive to small changes in reaction time, this stability in the pre-and post-reaction time data argues against a pronounced time-of-day effect within the allotted time window.

Individuals with an initial RT score at least one standard deviation below the mean (N = 4) showed significant improvement ($p < .03$). Moreover, all significant increases in RT standard score occurred in those persons whose initial score was below 100. On the other hand, some of those whose initial scores were greater than 100 showed a significant (> 0.5 SD) reduction in standard score. In all such cases, the initial standard score was considerably greater than 100. Further inspection found these individuals were impulsive in the pre-test and may have become more deliberate in the post-test, at the expense of some reaction time. However, in our experience individuals who are highly impulsive tend to have a number of anticipatory responses as well.

The response time data show stability in the population and no regression to the mean. It is possible there are two populations: (a) those who are fast and accurate on the TOVA, and who appear to remain so with the training (i.e., no substantial change is found outside of test/retest uncertainty); and (b) those who are fast by virtue of a tendency toward impulsivity. These might well show a decrement in RT standard score upon functional normalization. However, responses on the TOVA that occur 200 ms after the presentation of the target are arbitrarily considered anticipatory, in that the individual is assumed to be responding faster than is possible for a deliberate choice response. This assignment is mandated in the TOVA irrespective of whether the response was correct or not. In some individuals, the responses could have been deliberate. The 200 msec cut off may have affected the data in that an individual's best results are rejected as anticipatory, and very short reaction time is penalized as a functional deficit since anticipatory errors are excluded from the determination of reaction time (Leark et al., 2000).

EEG changes were not examined in this study. In the past, EEG data was recorded during sessions, but this policy has been abandoned recently as being of limited utility because of the lack of consistency in training protocol from session-to-session. Reward frequencies are shifted within sessions; multiple sites are trained within a session; or the time of day is not consistent and we cannot report EEG changes that occurred during or following neurofeedback.

The general rationale for using bipolar protocols stems from the intrinsic differences between long versus short range neural connections as stated in the "Two Compartmental" model of coherence. In this model (Braitenberg, 1978) there are short distance and long distance neural connections in the brain. The short distance system typically involves connections on the order of millimeters to a few centimeters. The long distance system involves interactions that occur over several centimeters. The critical difference between these two systems is that the long distance communication tends to require reciprocal feedback loops while the short distance networks tend to transmit their signal by the process of diffusion (Thatcher, 1998; Thatcher, Krause, & Hrybyk, 1986; Pasqual-Marqui, Valdes-Sosa, & Alvarez-Amador, 1988; Braitenberg, 1978; Braitenberg & Schuz, 1991). In single site training, a more localized form of coherence is facilitated, while in interhemispheric training communication between cortical sites via sub-cortical linkages (including the brainstem), cortical timing coordination at large distances, and thalamic regulatory networks is facilitated. Signal extraction suppresses the common-mode signal in electrodes placed on homologous sites in inter-

hemispheric training and electrode sites reward differential activity. As such, the relative phase within the reward band and amplitude enter into the feedback signal and this procedure rewards differentiation of function between the hemispheres through a change in coherence and comodulation. In contrast, single site amplitude "up-training" typically involves rewarding an increase in the common activity in the vicinity of the "active" sensor with respect to the neutral reference sensor, and phase is a secondary issue except to the extent that the reference is active.

Interhemispheric training can seem confusing and even counter-intuitive because it is a fundamentally different challenge to the brain than single-site training (Fehmi, 2002; Putman, 2002). However, the interhemispheric training approach to self-regulation may reduce coherence across hemispheres. It is possible that the brain organizes timing and sequencing between the hemispheres and that neurofeedback training is a challenge to the mechanisms governing this timing. From this perspective, inter-hemispheric EEG training may be a very subtle and specific challenge to the brain's regulatory networks that manage global timing relationships. The repetition of this action-reaction dynamic may serve to strengthen these regulatory loops.

Our clinical impression over years of training with this protocol is that interhemispheric training enhances the subjective experience of neurofeedback, increases the scope of efficacy across a greater range of conditions, enlarges the effect size, and improves the probability of good clinical outcome in the individual case. The above data represent a first attempt to firm up these clinical impressions quantitatively, in particular the observation that attentional variables normalize with successful neurofeedback irrespective of diagnostic presentation. Our observations would obviously be strengthened if they were derived from a research design that provided for relevant controls, and we hope that these preliminary findings will in time be subjected to such scrutiny.

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