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Short communication

Preliminary evidence that daily changes in frontal alpha asymmetry correlate with changes in affect in therapy sessions

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Abstract

Frontal EEG alpha asymmetry was recorded from five depressed outpatients during early EEG biofeedback sessions. Mood was assessed prior to and after each session, and affect change scores were also derived by subtracting pre-session from post-session scores. Alpha magnitude was obtained via Fast Fourier Transforms. All scores (EEG alpha asymmetry and affect) were converted to deviation scores by subtracting each patient's daily score from that patient's mean across all available sessions for that patient. Pearson correlations were then computed between asymmetry and affect scores using the deviation scores combined over patients. There was little evidence of correlation between day-to-day asymmetry score and any single affect score. Strong correlations were obtained, however, between asymmetry score and affect change score and, in particular, between asymmetry score and change in positive affect.

Keywords: Alpha asymmetry; Cortical activation asymmetry; Emotion; Electroencephalogram

Davidson (1992), Tomarken et al. (1990) and Wheeler et al. (1993) have provided evidence that frontal cortical EEG activation asymmetry predicts responsivity to affective manipulations. In this work, EEG is typically recorded on one or two occasions, and an EEG index is derived, based either on data from one session, or from the mean of two sessions for those subjects who show stable EEG asymmetry patterns over the two sessions (cf. Tomarken et al., 1990; Wheeler et al., 1993). Thus, although Tomarken et al. (1992, Tomarken et al., 1994) have suggested that both stable and phasic aspects of EEG asymmetry may be related to affective phenomena, the recent emphasis in this research has been on the functional significance of the stable, trait-like, asymmetry pattern, rather than on more phasic patterns based on daily changes in EEG asymmetry.

In the present study, we utilize a clinical population in a therapy situation to track, for the first time, day-to-day fluctuations in frontal activation asymmetry; moreover, we examine the covariation of these fluctuations with changes in affect. In previous work, a particular pattern of EEG activation asymmetry has been found to predict reactivity to specifically valanced stimuli in a laboratory situation (e.g. Tomarken et al., 1990; Wheeler et al., 1993). In the present study we examined the possibility that EEG activation asymmetry patterns are associated with affective changes within a consistent therapy situation.

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Table 1
Patient demographic, affective, EEG, and clinical characteristics

Patient	Sex	Therapist	Age	BDI score	Sessions	A-score range
1	f	EB	29	25	8	−8.2 to 11.0
2	f	EB	48	37	5	0.8 to 9.5
3	f	EB	42	7	7	−1.7 to 8.8
4	f	EB	34	23	11	1.5 to 14.3
5	m	RB	22	26	19	−3.7 to 22.8

BDI = Beck Depression Inventory; A-score = frontal EEG asymmetry score.

Subjects in this study were five right-handed outpatients from a private practice in Evanston, IL. The diagnosis was made in an informal, loosely structured clinical interview, using DSM-IV criteria. Beck Depression Inventories were also used. There were two DSM-IV diagnoses of 300.4 (dysthymic disorder; early onset, cases 2 and 3 in Table 1), two diagnoses of 309.0 (adjustment disorder with depression, cases 1 and 5 in Table 1), and one diagnosis of 291.8 (substance disorder with depression, case 2 in Table 1). All sessions yielding data presented here, on all patients, involved alpha asymmetry (ALAY) training through EEG biofeedback. Because our emphasis here is not on the effects of this training per se, we describe the biofeedback training only briefly below. Four females were treated by EB, a female therapist, and one male was treated by RB, a male therapist. One female patient was taking Prozac at a constant dose from the beginning to the end of the present observations. All patients gave informed consent to procedures that were described to them as experimental. In Table 1 we present demographics and scores on the Beck Depression Inventory (BDI; Beck et al., 1961) for the five patients. As is also shown in Table 1, the patients had relatively few ALAY sessions. Indeed, only one patient (R5) had a sufficient number of training sessions (19) to allow a clear inference of successful ALAY training; data on training effects will be presented elsewhere.

The patients typically had one 50-min eclectic verbal psychotherapy session per week and one session involving ALAY training. In this latter session, the first half-hour involved no therapy and minimal verbal interaction with the therapist during electrode attachment. After the ALAY training session and after completion of the post-session affect measures, the therapists explored the patients' feelings, ideation, etc., during and following removal of the electrodes. During the ALAY training, patients had their eyes closed and knew that the therapists were present, though silent. The therapists also knew that it was important for them to be neutral and silent during ALAY training sessions, and endeavoured to do so consistently.

Before and after each ALAY session, patients completed a visual analog mood scale (VAS) by marking a vertical slash across a 100 mm horizontal line, anchored on the left with 'as emotionally bad as you have ever felt', and on the right with 'as emotionally good as you have ever felt'. Patients also completed the Positive and Negative Affect Scale (PANAS; Watson et al., 1988) at the beginning and end of each ALAY training session. This measure yields scores on two orthogonal subscales: Positive Affect (PA) and Negative Affect (NA). Thus, the affect variables that were available for correlation with the EEG data were VAS, PA, and NA scores at the beginning and end of each session. We also derived a number of additional variables: AB (PA-NA; conceptually similar to the 'affective bias' measure utilized by Tomarken et al., 1990); GR (PA + NA; conceptually similar to the 'generalized reactivity' measure used by Tomarken et al., 1990); D-VAS, D-PA; D-NA; D-AB; and D-GR, which represent change in VAS, PA, NA, AB, and GR, respectively, from the beginning to the end of the ALAY session; i.e. post-session minus pre-session scores. NA scores (uniquely) were given minus signs so that a positive D-NA represented a reduction in negative affect. For the four female patients, data collection was begun in the first ALAY session; for the single male patient, affect data were inadvertently not collected until the seventh training day.

EEG was recorded from F3 and F4 referenced to Cz with the forehead grounded, utilizing a commercial EEG analysis system (Lexicor Neurosearch Model 2A). EEG was sampled at 256 Hz, after amplification at 32 000. Signals between 2 and 32 Hz were passed; alpha band was defined as 8–13 Hz. The system performs Fast Fourier Transforms on artifact-free 1-s epochs, overlapping by 50%, utilizing Blackman–Harris windows (Harris, 1978). The output is an ALAY or alpha asymmetry score $A = 100 \times (R - L)/R + L)$, where R and L are right- and left-alpha magnitude (square root of power),

respectively. (Ranges of these scores for each patient are shown in Table 1.) Artifacts included both EEG range errors (> 90 μV) and EOG excursions > 50 μV recorded from electrodes above and below the right eye. Mean A-scores were obtained for the first half of a session, A1, for the final part of the session, A2, and for the entire session. Thus, D-A scores (A2-A1) were also available.

In the ALAY training sessions the subjects were instructed to try to increase the pitch of a ubiquitous tone from a speaker in front of them, using whatever mental maneuvers they could summon. The pitch was proportional to the current A-score, updated every second, based on the most recent 1-s epoch. Additionally, noticeable, constant-tone volume increases accompanied 'hit' trials, defined as criterion-reaching A-scores. The criterion was set in the first session to be equal to the mean A-score for the first 5 min of the 30-min session. Patients were also told to try to generate these 'hits'.

The major question examined here did not concern the possible effects of ALAY training on affect (for which one should have more than 30 training sessions per patient; Lubar, 1991, and on which we will report later). Rather, our interest here is in the day-to-day covariations of affect and EEG measures. Indeed, at asymptotic training levels, the variance of A-scores would be expected to decrease as scores remain in the asymptotic ranges, thus reducing the likelihood of obtaining significant correlations between affect and EEG asymmetry. For that reason, we decided a priori to use only early training data in this study (i.e. fewer than 20 sessions per patient). We also decided that the available data would be organized for analysis when a total of 50 sessions from all five subjects were available, including at least 30 from the four females, collectively.

The approach we adopted was to convert all raw scores to deviation scores within each patient by subtracting each score from its mean over sessions for that patient. Thus, pre-session scores on the VAS (VAS-E), PA (PA-E), NA (NA-E), AB (AB-E), and GR (GR-E), and post-session scores on the VAS (VAS-O), PA (PA-O), NA (NA-O), AB (AB-O), and GR (GR-O), as well as change scores (D-A, D-VAS, D-PA, D-NA, D-AB, and D-GR), and A-scores were all converted to deviation scores within each patient. Thus, the deviation score for D-A will be indicated as D'-A; D'-AB will denote the deviation score on D-AB, and so on. This procedure made it possible to develop a within-subject correlation matrix in which data from (different) multiple numbers of sessions from multiple patients could be meaningfully combined (i.e. pooled) to yield adequate degrees of freedom for analysis. This method, which is unusual, is considered in detail by Pedhazur (1982, pp. 530–540), and by McNemar (1949; Chapter 15, pp. 320–322), where the degrees of freedom are derived (p. 321) as N-k-1, where N in our case is the number of total sessions from all patients, and k is the number of patients. The method involves pooling all deviation scores from all patients into one set, and then taking the Pearson correlations on relevant pairs of scores from the multi-patient, multi-session set.

We reasoned that there were three different rational ways to group sessions from the patients in this study: Grouping 1, all five patients combined ($N = 50$); Grouping 2, the four female patients treated by EB ($N = 31$); and Grouping 3, the three female patients treated by EB, excluding the one who was taking Prozac all through training ($N = 26$). In all three groupings, the absolute correlations of A-scores with all but one of the non-change affect scores were less than 0.3 (i.e. there were low correlations of the

Table 2
Correlations of asymmetry and affect change scores

Grouping	No. of patients	No. of sessions	df	A'-D'-VAS	A'-D'-PA	A'-D'-NA	A'-D'-AB
1	5	50	44	0.274	0.332 *	0.174	0.319 *
2	4	31	26	0.327	0.473 **	0.218	0.449 **
3	3	26	22	0.426 *	0.515 ***	0.449 *	0.554 ***

Note: Within-subject (all positive) correlation coefficients of individual deviation scores, pooled across sessions. Groupings are explained in the text. A' = frontal EEG asymmetry score; D'-VAS = change in Visual Analog Scale scores; D'-PA = change in postive affect; D'-NA = change in negative affect; D'-AB = change in affective bias. * = $p < 0.05$; ** = $p < 0.02$; *** = $p < 0.01$.

variables VAS′-E, PA′-E, NA′-E, AB′-E, GR′-E, VAS′-O, PA′-O, AB′-O, and GR′-O, with baseline EEG asymmetry). The single exceptional non-change affect variable, NA′-O, correlated 0.34 with A-score ($p < 0.05$) in Grouping 2, 0.31 with A-score ($p > 0.1$) in Grouping 3, and < 0.2 ($p > 0.1$) in Grouping 1.

In contrast, the results of the correlations of the affect change scores with baseline EEG asymmetry, with the exception of D′-GR, were quite different. As indicated in Table 2, whether the patients are examined in groups of three, four, or five, strong, positive correlations are obtained between baseline EEG asymmetry scores and changes in affect from the beginning to the end of sessions. The correlations reach a $p < 0.02$ or $p < 0.01$ level of statistical significance for the two groupings of female patients for D′-PA and D′-AB (change in PANAS positive affect and in affective bias, respectively). Not shown in Table 2, the correlations between EEG asymmetry and changes in generalized reactivity were all < 0.3 (ns).

Table 2 also suggests that as the groupings become more homogeneous (i.e. looking down each column), the correlations increase. Adding the single male subject to form Grouping 1 reduces the correlations so that, although still relatively high, none reaches the $p < 0.02$ level of significance. This could be a function of this subject's and/or his therapist's gender, but it could also involve the fact that the 19 training sessions' worth of data included for this subject probably included sessions when an ALAY training effect was evident. As already noted, learned asymmetry changes could reduce the range of the asymmetry variable. Therefore, the Grouping 1 correlation matrix was re-computed using only this male patient's first eight sessions (8 being the average number of sessions from the other patients). In these reanalyses, the positive correlations were 0.33 ($p < 0.05$), 0.39 ($p < 0.02$), 0.20 (ns), and 0.37 ($p < 0.05$) between baseline EEG asymmetry score and D′-VAS, D′-PA, D′-NA, and D′-AB, respectively.

Because the early and late A-scores, A1 and A2, correlated > 0.95 with each other and with the mean A-score for the entire session, we did not examine the correlations of affect with A1 and A2. Finally, as would be expected from the high correlation of A1 and A2, the D′-A score did not correlate > 0.20 with any affect or affect change variable. Thus, although one might have expected D′-A to correlate with change in affect scores, the narrow range of variation in asymmetry over the course of the session apparently precluded such a relationship.

The present data extend previous trait models of the functional significance of cortical activation asymmetry (Davidson, 1992; Tomarken et al., 1990; Wheeler et al., 1993; Henriques and Davidson, 1990, 1991; Tomarken et al., 1992) by demonstrating that day-to-day fluctuations in EEG asymmetry, heretofore unstudied, predict the direction of change in affective responses to EEG training over the course of the training sessions. Previous studies have examined EEG asymmetry in only one or two sessions. For example, Wheeler et al. (1993) computed correlations of EEG asymmetry with affect and affective reactivity only for subjects who demonstrated high test-retest reliability of asymmetry scores across two sessions. This procedure excluded more than half of their subjects from analyses. Similarly, Tomarken et al. (1992) found that correlations between EEG asymmetry and trait affect were relatively large in the 21 subjects who showed 'stable' EEG asymmetry across two occasions, but not in the 51 subjects with 'unstable' asymmetry. It is possible that the patients in our study, whose asymmetry scores showed considerable daily fluctuation, were similar to the EEG-unstable subjects who were excluded from the study of Wheeler et al. (1993). This lability of asymmetry scores may be related to: (1) their patient status, which could reasonably be expected to be associated with greater fluctuations in mood than those seen in the student samples studied by Wheeler et al. (1993); (2) the normative asymmetry instability demonstrated by the majority of subjects in these studies; and/or (3) the fact that we collected data over a considerably greater number of sessions than has been the case in previous studies.

A distinguishing result of the present study is that virtually all of the statistically significant correlations that were obtained involved affect change scores, rather than simple affect scores. However, it is important to note that we analyzed data from multiple sessions from multiple subjects, whereas Wheeler et al. (1993) and Tomarken et al. (1992) utilized single asymmetry scores or single means of two sessions' worth of scores from each subject. We

actually also calculated correlations in this way (not yet reported here), and, although the limited degrees of freedom of our small groupings temper the inferences that we can draw from these data, we should note here that for our Grouping 2 ($n = 4$), the correlations between EEG asymmetry and D'-VAS, D'-PA, D'-NA, and D'-AB were, 0.91 ($p < 0.05$), 0.58 (ns), 0.83 ($p < 0.1$), and 0.95 ($p < 0.05$), respectively. For our Grouping 1 ($n = 5$), the values were 0.78, 0.58, 0.24, and 0.56 (all ns), respectively.

We believe it is reasonable to assume that the actual conditions of the EEG training sessions in the present study, were relatively constant from day to day. The therapist–experimenters knew of the importance of such consistency; moreover the patients' eyes were closed during the session, with the therapists silent and out of sight. Nevertheless, as might be expected, patient affect improved from the beginning to the end of session on some days, and worsened on other days. Interestingly, the pattern of frontal EEG asymmetry correlated with the direction of the affective change. If the assumption of a constant external situation of affective evocativeness (i.e. the constant therapeutic situation) is correct, then the source of the affective variance must be internal. Thus, a novel aspect of frontal activation asymmetry, namely its daily fluctuation, may reflect patients' phasic endogenous bias to respond in a particular manner, i.e. positively or negatively, to a constant situation. This hypothesis is clearly speculative, and it remains for further research to examine this possibility more explicitly and systematically.

In conclusion, the present data set provides suggestive preliminary evidence that at least in a patient or other group showing considerable daily fluctuation of frontal activation asymmetry, the mean asymmetry score for the session predicts whether the change in subject affect over the session will be positive or negative. This conclusion must be tempered by the fact that only a small number of subjects were run in this study, and, indeed, these five persons did not have identical depressive psychopathology; i.e. a different patient group may have yielded a different pattern of correlations.

References

Beck, A.T., Ward, C.H., Mendelson, M., Mock, J. and Erbaugh, J. (1961) An inventory for measuring depression. Arch. Gen. Psychiatry, 4: 561–571.

Davidson, R.J. (1992) Anterior cerebral asymmetry and the value of emotion. Brain Cognit., 20: 125–151.

Harris, F.J. (1978) On the use of windows for harmonic analysis with the discrete Fourier transform. Proc. IEEC, 16: 51–84.

Henriques, J.B. and Davidson, R.J. (1990) Regional brain electrical asymmetries discriminate between previously depressed and healthy control subject. J. Abnorm. Psychol., 99: 22–31.

Henriques, J.B. and Davidson, R.J. (1991) Left frontal hypoactivation in depression. J. Abnorm. Psychol., 100: 535–545.

Lubar, J.F. (1991) Discourse on the development of EEG diagnostics and biofeedback for attention deficit/hyperactivity disorders. Biofeed. Self Reg., 16: 201–225.

McNemar, Q. (1949) Psychological Statistics, John Wiley and Sons, New York, pp. 320–322.

Pedhazur, E.J. (1982) Multiple Regression in Behavioral Research: Explanaton and Prediction, 2nd edn., Holt, Rienhart, and Winston, New York, pp. 530–540.

Tomarken, A.J., Davidson, T.J. and Henriques, J.B. (1990) Resting frontal brain asymmetry predicts affective responses to films. J. Personal. Soc. Psychol., 59: 791–801.

Tomarken, A.J., Davidson, R.J., Wheeler, R.E. and Doss, R. (1992) Individual differences in anterior brain asymmetry and fundamental dimensions of emotion. J. Personal. Soc. Psychol., 62: 676–687.

Tomarken, A.J., Keener, A. and Neubauer, D.L. (1994) Long-term stability of frontal brain asymmetry: It helps to aggregate. Poster presented at 34th Ann. Meet., Soc. Psychophysiol. Research, Atlanta.

Watson, D., Clark, L.A. and Tellegen, A. (1988) Development and validation of brief measures of positive and negative affect: The PANAS scales. J. Personal. Soc. Psychol., 54: 1063–1070.

Wheeler, R.E., Davidson, R.J. and Tomarken, A.J. (1993) Frontal brain asymmetry and emotional reactivity: A biological substrate of affective style. Psychophysiology, 30, 82–89.