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A study on effective of increasing right frontal alpha and decreasing left frontal alpha on treatment of major depressive disorder

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A B S T R A C T

Various studies have shown some relationship between brain wave abnormalities and depression. The current study aimed to examine the effectiveness of the real neurofeedback treatment compared with mock neurofeedback in decreasing major depression severity of symptoms and change on α waves into a desirable pattern among some patients who suffer from major depression disorder. The study chooses six patients who were suffering from major depression sufferers and they were randomly placed in two groups called real neurofeedback and mock neurofeedback group (placebo). The two groups were treated for a twenty sessions twice a week. The two groups were examined before, during and after the treatment by Beck Depression Inventory II, Hamilton Depression Scale. The research data were examined through the analysis of the size effect, improvement percentage and charts. The data resulting from the size effect and the improvement percentage suggested that the real neurofeedback was more effective in regulating brain waves and in decreasing major depression disorder symptoms in comparison with the mock neuro-feedback and the groups were significantly different from the clinical point of view. The effectiveness of the real neurofeedback was not from the changes in placebo and it can be used as a complementary treatment in treating major depression disorder. The findings of the current research were congruent with those of the related studies.

1. Introduction

Alternating treatments design is considered as one strategy for comparing the effects of two treatments in a single subject (Barlow & Hayes, 1979; Sadock, 2007). Baehr et al. (1997) investigated the clinical use of an alpha asymmetry protocol in the neurofeedback treatment of depression (Hammond, 2007, 2011; Sterman, 1996) by considering two different case studies. There has been a convergence trend in lesion and neuroimaging information in the determination of circuits

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underlying positive and negative emotion in the human brain. Davidson and Irwin (1999), in a novel work, put emphasis on the prefrontal cortex (PFC) and the amygdala as two important components of this circuitry. The organism must have some means of representing effect in the absence of immediate elicitors. They claimed that the PFC plays essential role in affective working memory. The ventromedial sector of the PFC is involved in the representation of elementary positive and negative emotional states while the dorsolateral PFC could be integrated in the representation of the objective states towards which these elementary positive and negative states would be directed. The amygdala has been consistently detected as playing essential role in both the perception of emotional cues and the production of emotional responses, with some evidence recommending that it could be involved with fear-related negative effect.

Debener et al. (2000) made an assessment on additional characteristics of resting electroencephalographic (EEG) alpha (8–13 Hz) asymmetry in 15 clinically depressed patients and 22 healthy adults by collecting EEG activity on two separate occasions, 2–4 weeks apart. Across both sessions, group differences in anterior EEG asymmetry were consistent with the original hypothesis. However, groups varied in temporal stability of anterior EEG asymmetry, which was retest reliable in controls but not depressed patients. Rosenfeld (1997) investigated EEG biofeedback of frontal alpha asymmetry in affective disorders. Raymond et al. (2005) investigated the effects of alpha/theta neurofeedback on personality and mood. Storch et al. (2004) investigated factor structure, concurrent validity, and internal consistency of the Beck Depression Inventory—Second Edition (Beck et al., 1993; Williams, 1998; Walker, 2007; Tenke et al., 2011) in a sample of college students.

According to Finniss et al. (2010), placebo impacts are genuine psychobiological events attributable to the overall therapeutic context, and that these influences can be robust in both laboratory and clinical settings. There is also events where placebo influences exist in clinical practice, even if no placebo is provided. According to Hagemann (2004) reviewed concentrated on the reliability and validity of measures of anterior resting EEG asymmetry, which could serve as a proxy for trait-like asymmetries of cortical activity. These issues incorporated the treatment of ocular and muscle artifacts, the choice of the EEG reference, the implementation of current source density (CSD) measures, the state–trait nature of resting asymmetry, and the treatment of state-like fluctuations of the measures. Hammond (2000) provided some support on Henriques and Davidson's (1991) belief that hypoactivation of the left hemisphere results in an “approach deficit” and more withdrawal behavior.

Hammond (2005a) reported that there are biological predispositions that often exist for depression, anxiety, and obsessive–compulsive disorder but new research has demonstrated that medication was only mildly more effective than placebo in the treatment of these problems. Hammond (2005b), in another work, reviewed neurophysiologic research on functional brain abnormalities related to depression, anxiety, and obsessive-compulsive disorder. The review disclosed that pharmacologic treatment could not be as effective as previously believed. A more recent neuroscience technology, EEG biofeedback (neurofeedback), seems to become a technique for keeping abnormal brain wave patterns.

2. The proposed model

The paper examines the effectiveness of the real neurofeedback treatment compared with mock neurofeedback in decreasing major depression severity of symptoms and change on α waves into a desirable pattern among some patients who suffer from major depression disorder. The study chooses six patients who were suffering from major depression sufferers and they were randomly placed in two groups called real neurofeedback and mock neurofeedback group (placebo). The two groups were treated for a twenty sessions twice a week. The two groups were examined before, during and after the treatment by Beck Depression Inventory II, Hamilton Depression Scale. The research data were examined through the analysis of the size effect, improvement percentage and charts.

3. The results

Table 1 demonstrates details of Kuhn d test on left and right of pre-test left α, post-test α. Table 2 show the results of BDI-II and Table 3 demonstrates the results of HRSD for two groups.

Table 1
The results of Kuhn d on left and right of pre-test left α, post-test α

Group	Experiment	Pre-test left α	SD	Post-test left α	SD	effect	Pre-test right α	SD	Post-test right α	SD	effect
real neurofeedback	1	5.70	0.56	8.22	0.66	4.13	5.44	0.21	8.94	1.18	5
	2	4.52	0.13	4.58	0.19	0.37	4.57	0.39	4.62	0.17	0.17
	3	5.46	0.57	5.56	0.21	0.25	5.27	0.26	5.68	3.00	1.46
		Objective=reduce				1.58		Objective-increase			2.21
mock neurofeedback group	4	8.43	0.98	8.86	0.62	0.53	9.16	0.69	9.03	0.55	0.20
	5	6.4	0.48	5.29	0.23	1.97	4.47	0.44	5.1	0.72	1.8
	6	7.56	0.74	7.41	0.28	0.29	7.39	2.49	7.65	0.39	0.18
						0.93					0.48

Table 2
The results of BDI-II for two groups

Group	Test	Base-mean	Std. dev.	Mean treatment	Std. dev. treatment	Recovery	Effect
real neurofeedback	Experiment 1	33.31	3.21	15.5	6.85	50.52%	3
	Experiment 2	34.66	1.52	12	5.29	65.37%	6.66
	Experiment 3	33	1	23.25	7.88	29.54%	2.19
	Mean	32.99		16.91		48.47%	3.95
mock neurofeedback group	Experiment 4	31	3.6	24.25	2.36	21.77	2.26
	Experiment 5	30	1	23.25	4.11	22.5	2.64
	Experiment 6	37.66	2.88	28	2.16	25.65	3.83
	Mean	32.88		25.16		23.30	2.91

Table 3
The results of HRSD for two groups

Participants	real neurofeedback			group mock neurofeedback		
Treatment stages	Experiment 1	Experiment 2	Experiment 3	Experiment 4	Experiment 5	Experiment 6
Basic	36	42	40	43	35.33	44
20^{th} session	15	14	16	30	24	36
Recovery	58.33%	66.66%	60%	30.23%	32.06%	18.18%
Total recovery		61.66%			26.82%	

4. Discussion and conclusion

The data resulting from the size effect and the improvement percentage have suggested that the real neurofeedback was more effective in regulating brain waves and in decreasing major depression disorder symptoms compared with the mock neuro-feedback and the groups were significantly different from the clinical point of view. The effectiveness of the real neurofeedback was not from the changes in placebo and it can be used as a complementary treatment in treating major depression disorder. The findings of the current research are consistent with results of Debener et al. (2009) but they are in contradiction with Hammond (2005a), Baehr et al. (1997) and Rosenfeld (1997).

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