

Comparative Study of the Efficacy of Biofeedback-Assisted Jacobsons Progressive Muscle Relaxation (Jpmr) for Managing mild/Moderate Depression

Swayamprava prava baral (✉ swayamprava123@gmail.com)

Central Institute of Psychiatry <https://orcid.org/0000-0003-1801-9431>

Gyanendra Raghuvanshi

DMC: Datia Medical College

Abhay paliwal

MGM Medical College: Mahatma Gandhi Memorial Medical College

Research

Keywords: Biofeedback, JPMR, Depression, Relaxation

Posted Date: August 30th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-744964/v1>

License:  This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Biofeedback is the way of gaining greater awareness of physiological functions with a goal of self-regulation. JPMR (Jacobsons progressive muscle relaxation) causes release of tension in the skeletal muscles, neuro-muscular system is thus seen as a mediator in the relief of depressive symptoms. This study aimed to see the Comparative efficacy of Biofeedback assisted JPMR, Escitalopram and Bimodal use of both in management in mild/moderate depression. The study was conducted at Mental hospital, Indore, with a Sample Size of 30 [**Group A** 10 ; biofeedback ,**Group B** 10 ; Escitalopram ,**Group C** 10;both]. 8 sessions of biofeedback assisted JPMR was given to group A and C .Escitalopram was given group B and C. HAM-D and BDI was applied at baseline , 4 weeks and 8 weeks. **As per BDI scale scores, Biofeedback assisted JPMR combined with escitalopram has significantly better response than only biofeedback or only Escitalopram in patients of mild to moderate depression. As per HAM-D scale scores, Biofeedback assisted JPMR combined with escitalopram has significantly better response than only biofeedback or only Escitalopram in patients of mild to moderate depression.** Thus Biofeedback appears to be a useful adjunctive treatment for mild to moderate depressive episode.

Summary Points

- Biofeedback is the way of gaining greater awareness of physiological functions with a goal of self-regulation.
- JPMR (Jacobsons progressive muscle relaxation) causes release of tension in the skeletal muscles, neuro-muscular system is thus seen as a mediator in the relief of depressive symptoms
- As per HAM-D scale scores, Biofeedback assisted JPMR combined with escitalopram has significantly better response than only biofeedback or only Escitalopram in patients of mild to moderate depression
- Thus Biofeedback appears to be a useful adjunctive treatment for mild to moderate depressive episode.

1. Introduction

Biofeedback is the process of gaining greater awareness of many physiological functions by using instruments that provide information on the activity of those same systems((**EEG, EMG, GSR, PR, TEMP, RESP**), with a goal of being able to change them at will (Barlow et al.,2016). **A growing body of research indicates that autonomic nervous systemdysfunction in depression** (Veith et al., 1994; Carney et al., 2005).The Bio-feedback method aims to counteract the effects of SympatheticNervous System by promoting the action of the Parasympathetic Nervous System(Benson et al.,1974).

Before Biofeedback: Sympathetic arousal,Beta activity in EEG, Muscular constriction in EMG, Shallow and rapid respiratory curves in Pneumograph, Increased resistance in GSR, Vasoconstriction in thermal feedback, Increased Noradrenalin secretion **After Biofeedback:** Parasympathetic dominance, Alpha

activity in EEG, Muscular relaxation in EMG, Deep and regular respiratory curves in Pneumograph, Decreased skin resistance in GSR, Vasodilatation in Thermal feedback, Acetylcholine secretion.

Most patients are trained to relax and modify their behaviour in biofeedback. Stressful events produce strong emotions, which arouse certain physiological responses. Many experts believe that these individual responses to stress can become habitual. When the body is repeatedly aroused, one or more functions may become permanently overactive. Actual damage to bodily tissues may eventually result (Lazarus and Folkman, 1984). Biofeedback is often aimed at changing the habitual reactions to stress that can cause pain or disease. Many clinicians believe that some of their patients have forgotten how to relax. Feedback of physical responses such as skin temperature and muscle tension provides information to help patients recognize a relaxed state. The feedback signal may also act as a kind of reward for reducing tension.

In a health care environment that where cost containment and evidence-based practice are important, biofeedback provides an effective way of non-pharmacological management in neurotic disorders like mild-moderate depression that comprises of maximum percentage of depressive disorders. Moreover it is not associated with any side effects or pain and has long term effect. Yucha and Montgomery's (2008) ratings are listed for the five levels of efficacy recommended by a joint task force and adopted by the Boards of Directors of the Association for Applied Psychophysiology (AAPB) and the International Society for Neuronal Regulation (ISNR) (Vaque et al., 2002). For depression it was Level 2 (Possibly Efficacious). This study aims to demonstrate that biofeedback achieves comparable efficacy as that of pharmacological methods.

2. Methods

2.1: *Study Objectives*

Efficacy of Biofeedback assisted JPMR in management of patients with mild/moderate depression.

Comparative efficacy of Biofeedback assisted JPMR, Escitalopram and bimodal use of both in management in mild/moderate depression

2.2 Subjects And Design

It was a comparative longitudinal study conducted at mental hospital, dept of psychiatry, mgmmc, indore, biofeedback unit. Randomized sampling technique was used to recruit 30 subjects divided into Group A 10 Depression patients on biofeedback, Group B 10 Depression patients on antidepressant (Escitalopram), Group C 10 Depression patients on biofeedback + antidepressant (Escitalopram)

2.3 Inclusion criteria: Diagnosis of Depression (F32 Depressive Episode or F33 Recurrent Depressive Episode, mild and moderate, except severe depression, depression with psychotic symptoms, depression with suicidality) according to ICD 10 (DCR). Patient aged between 18-60 yrs, either sex, who were drug naïve or drug free for 3 months. Patients giving written, informed consent.

2.4 Exclusion criteria: Any co-morbid psychiatric illness, h/o substance dependence, Head injury, epilepsy, SOL, any medical co-morbidity like .hypertension, endocrinological disorder (hypothyroidism, hyperthyroidism, cushing syndrome, diabetes mellitus,), Pregnancy and lactation, Current use of anti-hypertensive drugs, steroid hormones, growth hormone, anabolic steroids, retinoids, antipsychotics, Sedatives, immunosuppressants and immunomodulatory agents.

2.5 Tools: Informed Consent Form, Socio Demographic and Clinical Data Sheet, General Health Questionnaire 12, Hamilton depression rating scale, Beck Depression Inventory BIOFEEDBACK MACHINE –RELAX 701, Biofeedback workbook

2.6 PROCEDURE:

Subjects were recruited from mental hospital indore, fulfilling the inclusion and exclusion criteria. Written informed consent was taken after explaining the objectives and procedure of study in detail. Detailed physical examination was done to rule out any medical or neurological abnormality. The diagnosis of depression was made using the ICD -10. 1st session was introductory session which involved explaining the patients details of the study procedure. Group B and C patients were given escitalopram in optimum dosage. For group A and C, Next Sessions involved 20-25 minutes of abdominal breathing and biofeedback guided JPMR and parameters (alpha-EEG, EMG, GSR, PR ,RR, TEMPERATURE) were recorded using the biofeedback machine. Recorded audio was used for guided JPMR. Sessions were repeated once a week and continued upto two months. Rest 6 days of the week patients had to practice the techniques at home without biofeedback. Records of changes of all the parameters of all patients (all the 3 groups) through subsequent weeks was maintained in biofeedback computer. HAM-D was applied to all patients at baseline, 4weeks and 8weeks.

3. Results And Discussion

The mean age, in years, of patients in group A was 31.34 ± 11.21 years. The mean age, in years, of patients in group B was 33.1 ± 11.33 years. The mean age, in years, of patients in group C was 31.52 ± 11.11 years.(table 2) Patients were more likely to have low socioeconomic status (table 2) , an urban background, and be educated up to primary school and mostly Hindu, married, and from joint family. There was no statistically significant difference among the groups in gender, habitat, education or marital status (table 1).

The mean age of onset of depression in patient group A was 28.64 ± 8.76 years. The mean age of onset of depression in patient group B was 27.66 ± 9.20 years. The mean age of onset of depression in patient group C was 29.66 ± 9.44 years. The mean duration of illness in patient group A was 45.48 ± 46.08 months. The mean duration of illness in patient group B was 53.64 ± 45.49 months. The mean duration of illness in patient group C was 48.44 ± 40.55 months (table 3). Most patients had precipitating factors, had no past history, had no family history and had acute onset of illness (table 4).

For group A, the HAM-D score was 11 at baseline, 7 at 1 month, and 4 at 2 months. For group B, the mean HAM-D score was 11 at baseline, 8 at 1 month, and 4 at 2 months. For group C, the mean HAM-D score was 11 at baseline, 7 at 1 month, and 3 at 2 months. (table 5) For group A, the mean BDI score was 15 at baseline, 12 at 1 month, and 10 at 2 months. For group B, the mean BDI score was 15 at baseline, 12 at 1 month, and 10 at 2 months. For group C, the mean BDI score was 15 at baseline, 12 at 1 month, and 8 at 2 months 8 (table 6).

Significant improvements were noted in the Hamilton Depression Scale (HAM-D) and the Beck Depression Inventory (BDI) by Session 4, and further significant improvement was noted between session 4 and session 8 in patients in all groups.

The difference in BDI score (baseline vs 8th session) **was significantly greater** in group C (biofeedback +escitalopram) than in groups A (only biofeedback) and B (only escitalopram). The difference in BDI score (baseline vs 8th session) was equal for group A (only biofeedback) and group B (only escitalopram). The difference in BDI score (baseline vs 4th session) was **significantly greater** in group C (biofeedback +escitalopram) than in groups A (only biofeedback) and B (only escitalopram). The difference in BDI score (baseline vs 4th session) **was significantly greater** for group B (only escitalopram) than for group A (only biofeedback). The difference in BDI score (4th session vs 8th session) was **significantly greater** in group C (biofeedback +escitalopram) than in groups A (only biofeedback) and B (only escitalopram). The difference in BDI score (4th session vs 8th session) was equal for group A (only biofeedback) and group B (only escitalopram).

Therefore, according to BDI scale scores, biofeedback-assisted JPMR combined with escitalopram as a treatment modality produces a better response than biofeedback alone or SSRIs alone (escitalopram) in patients with mild to moderate depression.

Biofeedback-assisted JPMR produces an equal response compared to escitalopram in patients with mild to moderate depression.

The difference in HAM-D score (baseline vs 8th session) was greater in group C (biofeedback +escitalopram) than in groups A (only biofeedback) and B (only escitalopram). The difference in HAM-D score (baseline vs 8th session) was greater for group B (only escitalopram) than for group A (only biofeedback). The difference in HAM-D score (baseline vs 4th session) was greater in group C (biofeedback +escitalopram) than in groups A (only biofeedback) and B (only escitalopram). The difference in HAM-D score (baseline vs 4th session) was greater in group A (only biofeedback) than in group B (only escitalopram). The difference in HAM-D score (4th session vs 8th session) was **significantly greater** in group C (biofeedback +escitalopram) than in groups A (only biofeedback) and B (only escitalopram). The difference in HAM-D score (4th session vs 8th session) was **significantly greater** for group B (only escitalopram) than for group A (only biofeedback).

Therefore, according to HAM-D scale scores, biofeedback-assisted JPMR combined with escitalopram as a treatment modality produces a better response than biofeedback alone or SSRIs alone (escitalopram) in patients with mild to moderate depression.

According to HAM-D scale scores, biofeedback-assisted JPMR produces more response than escitalopram in patients with mild to moderate depression after 1 month (4th session), but produces less of a response than escitalopram between 1 to 2 months (between 4th and 8th session).

This finding can be explained by the fact that antidepressant action needs 2 to 3 weeks, but biofeedback-assisted JPMR acts immediately by inducing relaxation and reducing sympathetic tone.

Therefore, considering the overall improvement in symptoms for patients assessed using both HAM-D and BDI, biofeedback-assisted JPMR combined with SSRIs (escitalopram) as a treatment modality produces a better response than biofeedback alone or SSRIs alone (escitalopram) in patients with mild to moderate depression.

Only biofeedback is also a successful treatment for mild-moderate depression.

Moreover, it is not associated with any side effects or pain and has long-term effects. It improves overall relaxation for all parameters (i.e., EEG, EMG, GSR, PR, TEMP, RESP) over subsequent sessions.

The findings of this study are substantiated by the findings of previous studies. 'Preliminary case studies (Kumano et al., 1996; Rosenfeld, 2000) and pilot studies (Waldkoetter & Sanders, 1997) show neurofeedback decreases depressive symptoms. One study compared biofeedback-assisted relaxation to a wait-list control on depression in chronic pain patients and improved scores on the Beck Depression Index was found (Corrado & Gottlieb, 1999).

Physiological arousal is governed by the ANS. When the organism is under threat the SNS (Sympathetic Nervous System) increases arousal on the other hand the PNS (Parasympathetic Nervous System) restores the body to a resting state. These actions are involuntary and enable the organism to survive. When the activity of SNS is prolonged and the organism is exposed to constant threat the organs concerned can become fatigued. The Bio-feedback method aims to counteract the effects of SNS by promoting the action of the PNS (Basmajian, 1979).

Neuro-therapists have used EEG biofeedback when treating addiction, attention deficit hyperactivity disorder (ADHD), learning disabilities, anxiety disorders (including worry, obsessive-compulsive disorder and posttraumatic stress disorder), depression, migraines, and generalized seizures (Yucha & Montgomery, 2008).

HRV biofeedback may be useful for reducing loss of energy, lack of motivation, sleep disturbances or any of the other neuro-vegetative features of MDD. As an inexpensive, safe, and noninvasive technique, it may prove to be a useful alternative to some medical or surgical interventions (Karavidas et al., 2007)

Conclusions

On the basis of the index study, which substantiate the earlier findings of previous studies, it can be concluded that:

Biofeedback is a useful adjunctive treatment for mild to moderate depressive episode.

Biofeedback assisted JPMR is a successful non-pharmacological modality for treatment of mild-moderate depression.

So, non-pharmacological methods like biofeedback should be added to pharmacological management of mild-moderate depression.

Advantages

This the only study of its kind that compared the response three groups (only biofeedback ,only escitalopram and both).

Previous studies had conducted fewer sessions of biofeedback.

LIMITATIONS

Sample size could have been larger.

FUTURE DIRECTIONS

Further studies with larger sample size and more sessions of biofeedback assisted JPMR should be conducted in patients of depression as well as other psychosomatic illness.

Biofeedback is applicable not only for people suffering from any psychological or physiological disorders, but also applied on normal healthy individuals as Peak Achievement Training for improving attention and concentration. So further studies should be done in this regard.

Declarations

Acknowledgment: Authors wish to thank department of psychiatry MGM Medical College Indore.

Competing interests: None

Funding: There was no funding for this article

There is no conflict of interests.

Ethical Approval : THE STUDY WAS APROVED BY INSTITUTIONAL ETHICAL COMMITTEE MGMMC INDORE.

Competing interests: None

Funding: There was no funding for this article. There is no conflict of interest.

Authors contribution: *dr S. P. baral and dr G. Raghuvanshi collected the data and analysed the data and compiled it. Dr A paliwal guided the project.*

Consent for publication was obtained from each author and the institution.

References

1. Basmajian, J. V. (1979). *Biofeedback: Principles and practice for clinicians*. Williams & Wilkins.
2. Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Beck depression inventory-II. *San Antonio*, 78(2), 490-498.
3. Benson, H., Beary, J. F., & Carol, M. P. (1974). The relaxation response. *Psychiatry*, 37(1), 37-46.
4. Carney, R. M., Freedland, K. E., & Veith, R. C. (2005). Depression, the autonomic nervous system, and coronary heart disease. *Psychosomatic medicine*, 67, S29-S33.
5. Corrado, P., & Gottlieb, H. (1999). The effect of biofeedback and relaxation training on depression in chronic pain patients. *American Journal of Pain Management*, 9, 18-21.
6. Damasio, A. (2012). Emotions create our preference: the somatic marker hypothesis. *Neurorelay*. Recuperado de: <http://neurorelay.com/2012/05/15/emotions-create-our-preferences-the-somatic-marker-hypothesis>.
7. Barlow, D. H., Durand, V. M., & Hofmann, S. G. (2016). *Abnormal psychology: An integrative approach*. Cengage learning.
8. Hamilton, M. *Journal of Neurology, Neurosurgery, and Psychiatry*. 23:56-62, 1960.
9. Jacobson, E. (1938). Progressive muscle relaxation. Interview Behaviour". *Journal of Abnormal Psychology*, University of Chicago Press, Chicago. *chology*, 75(1), 18.
10. Karavidas M. K., Lehrer P. M., Vaschillo E. G., Vaschillo B., Marin H., Buyske S. et al. (2007). "Preliminary results of an open-label study of heart rate variability biofeedback for the treatment of major depression". *Applied Psychophysiology and Biofeedback* 32 (1): 19–30.doi:10.1007/s10484-006-9029-z. PMID 17333315.
11. Kumano, H., Horie, H., Shidara, T., Kuboki, T., Suematsu, H., & Yasushi, M. (1996). Treatment of a depressive disorder patient with EEG-driven photic stimulation. *Biofeedback and self-regulation*, 21(4), 323-334.
12. Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. Springer publishing company.
13. Rosenfeld, J. P. (2000). An EEG biofeedback protocol for affective disorders. *Clinical Electroencephalography*, 31(1), 7-12.
14. Vaque, T. J. L., Hammond, D. C., Trudeau, D., Monastra, V., Perry, J., Lehrer, P., ...& Sherman, R. (2002). Template for developing guidelines for the evaluation of the clinical efficacy of psychophysiological

interventions. *Journal of Neurotherapy*, 6(4), 11-23.

15. Veith, R. C., Lewis, N., Linares, O. A., Barnes, R. F., Raskind, M. A., Villacres, E. C., ...& Pascualy, M. (1994). Sympathetic nervous system activity in major depression: basal and desipramine-induced alterations in plasma norepinephrine kinetics. *Archives of general psychiatry*, 51(5), 411-422.
16. Waldkoetter, R. O., & Sanders, G. O. (1997). Auditory brainwave stimulation in treating alcoholic depression. *Perceptual and Motor Skills*, 84(1), 226-226.
17. What is biofeedback? Association for Applied Psychophysiology and Biofeedback. 2008-05-18. Retrieved 2010-02-22.
18. Yucha, C., & Montgomery, D. (2008). Evidence-based practice in biofeedback and neurofeedback. Wheat Ridge, CO: AAPB.

Tables

TABLE 1: COMPARISON OF SOCIODEMOGRAPHIC VARIABLES BETWEEN THE GROUPS

		Biofeedback assisted JPMR (N=10)	Escitalopram (N=10)	BOTH (N=10)	χ^2	p
Gender	Male	6(60%)	6(60%)	5(50%)	0.083	0.99
	Female	4(40%)	4(40%)	5(50%)		
Religion	Hindu	7 (70%)	6(60%)	9(90%)	35.686	0.000
	Others	3(30%)	4(40%)	1(10%)		
Habitat	Rural	4(40%)	3(30%)	4(40%)	4.244	0.236
	Urban	6(60%)	7(70%)	6(60%)		
Family type	Joint	6(60%)	8(80%)	4(40%)	23.681	0.001
	Nuclear	4(40%)	2(20%)	6(60%)		
Education	Primary	4(40%)	4(40%)	5(50%)	35.046	0.768
	Secondary	2(20%)	4(40%)	1(10%)		
	Graduate+	4(40%)	2(20%)	4(40%)		
Marital status	Married	7(70%)	7(70%)	5(50%)	7.813	0.252
	Unmarried	3(30%)	3(30%)	5(50%)		

TABLE 2: COMPARISON OF SOCIO-DEMOGRAPHIC PROFILE BETWEEN THE GROUPS (CONTINUOUS VARIABLES)

Variables	Biofeedback assisted JPMR (N=10)	Escitalopram	BOTH	F ratio	P
	Mean±SD	Mean±SD	Mean±SD		
Age (in years)	31.34±11.21	33.1±11.33	31.52±11.11	0.312	0.817
Total Income	17520.00± 9006.21	15240.00± 6096.00	21990.00± 20268.22	2.159	0.094

TABLE 3: CLINICAL CHARACTERISTICS OF THE DEPRESSION PATIENTS (CONTINUOUS VARIABLES)

Variables	Biofeedback assisted JPMR (N=10)	Escitalopram (N=10)	BOTH (N=10)
	Mean± SD	Mean± SD	Mean± SD
Age of onset of illness (in years)	28.64±8.76	27.66±9.20	29.66±9.44
Duration of illness (in months)	45.48±46.08	53.64±45.49	48.44± 40.55

TABLE 4: CLINICAL CHARACTERISTICS OF THE DEPRESSION PATIENTS(CATEGORICAL VARIABLES)

Variables		Biofeedback assisted JPMR	Escitalopram	BOTH
Precipitating factor	Yes	6(60%)	5(50%)	6(60%)
	No	4(40%)	5(50%)	4(40%)
Family history	Not present	8(80%)	6(60%)	8(80%)
	Present	2(20%)	4(40%)	2(20%)
Onset	Insidious	3(30%)	2(20%)	4(40%)
	Acute	7(70%)	8(80%)	6(60%)
Past history	Not present	9(90%)	7(70%)	6(60%)
	Present	1(10%)	3(30%)	4(40%)

TABLE5: COMPARISON OF HAM-D SCORES

		Group			
		Biofeedback assisted JPMR	Escitalopram	Both	Total
HAM-D baseline	Mean	11.0000	11.1000	11.4000	11.1667
	Std. Deviation	2.44949	2.33095	2.36643	2.30567
HAM-D 4th session	Mean	7.0000	8.2000	7.8000	7.6667
	Std. Deviation	2.21108	2.25093	2.29976	2.23350
HAM-D 8th session	Mean	4.2000	4.4000	3.0000	3.8667
	Std. Deviation	1.98886	2.50333	1.41421	2.04658
Baseline-4th session	Mean	4.0000	2.9000	3.6000	3.5000
	Std. Deviation	1.63299	.73786	.51640	1.13715
4th session- 8th session	Mean	2.9000	4.1000	4.8000	3.9333
	Std. Deviation	.73786	1.44914	1.54919	1.48401
baseline-8th session	Mean	6.9000	7.0000	8.4000	7.4333
	Std. Deviation	1.37032	1.88562	1.57762	1.71572

ANOVA Table

			Mean Square	F	Sig.	Post-hoc
HAM-D baseline * group	Between Groups	(Combined)	.433	.076	.927	
	Within Groups		5.678			
HAM-D 4th session * group	Between Groups	(Combined)	3.733	.735	.489	
	Within Groups		5.081			
HAM-D 8th session * group	Between Groups	(Combined)	5.733	1.407	.262	
	Within Groups		4.074			

Baseline-4th session * group	Between Groups	(Combined)	3.100	2.674	.087	Both>E
	Within Groups		1.159			Both=B
						B > E
4th session-8th session * group	Between Groups	(Combined)	9.233	5.491	.010	<i>Both > B</i>
	Within Groups		1.681			Both>E
						E > B
baseline-8th session * group	Between Groups	(Combined)	7.033	2.663	.088	Both > B
	Within Groups		2.641			Both>E
						E > B

TABLE6: COMPARISON OF BDI SCORES

		Group (N=30)				
		Biofeedback assisted JPMR (N=10)	Escitalopram (N=10)	Both (N=10)	Total	
BDI baseline	Mean	15.4000	15.3000	15.4000	15.3667	
	Std. Deviation	3.94968	3.94546	3.94968	3.81000	
BDI 4th session	Mean	12.8000	12.6000	12.0000	12.4667	
	Std. Deviation	3.99444	3.50238	4.02768	3.72997	
BDI 8th session	Mean	10.3000	10.2000	8.2000	9.5667	
	Std. Deviation	4.13790	2.93636	3.11983	3.46095	
Baseline-4th session	Mean	2.6000	2.8000	3.4000	2.9333	
	Std. Deviation	.69921	.78881	.69921	.78492	
4th session- 8th session	Mean	2.7000	2.7000	4.3000	3.2333	
	Std. Deviation	.94868	.94868	1.76698	1.45468	
baseline-8th session	Mean	5.3000	5.5000	7.7000	6.1667	
	Std. Deviation	1.33749	1.43372	1.41814	1.74363	
ANOVA Table						
			Mean Square	F	Sig.	Post-hoc
BDI baseline * group	Between Groups	(Combined)	.033	.002	.998	
	Within Groups		15.589			
BDI 4th session * group	Between Groups	(Combined)	1.733	.117	.890	
	Within Groups		14.815			
BDI 8th session *	Between Groups	(Combined)	14.033	1.187	.321	

group	Within Groups		11.826			
Baseline-4th session * group	Between Groups	(Combined)	1.733	3.250	.054	Both>B
	Within Groups		.533			Both>E
						E>B
4th session-8th session * group	Between Groups	(Combined)	8.533	5.201	.012	Both>B
	Within Groups		1.641			Both>E
						E= B
baseline-8th session * group	Between Groups	(Combined)	17.733	9.085	.001	Both>B
	Within Groups		1.952			Both>E
						E= B