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Thank you for your interest in neurofeedback for anxiety symptoms!

Below you'll find four papers, **the first** is looking at the brain waves connected with anxiety and whether they change in a positive direction after training.

The second, a meta-analysis of research on neurofeedback for anxiety concludes that neurofeedback studies thus far are showing positive effects.

The third is more recent (presented at a neurofeedback conference in 2014) study conducted by psychiatrists using the NeurOptimal system on subjects with anxiety and depression. They also measured changes on a PTSD scale and found all three significantly reduced.

The last study is looking at anxiety in children and found significant positive results.

We have highlighted relevant sections if you only have time to scan the articles.

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An open label study of the use of EEG biofeedback using beta training to reduce anxiety for patients with cardiac events

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Objective: To establish the effectiveness of EEG biofeedback using beta training as a relaxation technique and ultimately reducing anxiety levels of patients with confirmed unstable angina or myocardial infarction.

Methodology: Patients with confirmed unstable angina or myocardial infarction referred by cardiologists were recruited 2–3 days after their cardiac event from the cardiology wards. Their initial anxiety scores were determined using the Hospital Anxiety and Depression Scale. Those that returned for therapy underwent instrument feedback training using EEG every two weeks for a total of five sessions. EEG frequencies were measured for all sessions. Dropouts who did not participate in the program agreed to return 3 months later for the second psychological assessment. The study design was uncontrolled.

Results: Subjects had significantly lower anxiety scores at the second screening ($p < 0.001$), while the dropouts had significantly higher scores ($p < 0.001$). Beta training was effective in increasing sensory motor rhythm (SMR) waves but no significant effect was present for the alpha waves.

Conclusions: The uncontrolled nature of the study limits firm conclusions. However, the significantly lowered anxiety scores for subjects and enhancing of SMR waves indicate the effectiveness of beta training as a promising approach to EEG biofeedback for anxiety reduction.

Keywords: EEG biofeedback, cardiac events, anxiety, alpha waves, beta waves

Introduction

Appropriate breathing and relaxation techniques decrease the morbidity and mortality of patients with cardiac events especially myocardial infarction (Dixhorn and Duivenvoordeen 1999). Incorporating these stress reduction therapies in the usage of EEG biofeedback proves to be an important entity in cardiac rehabilitation programs involving patients with ischemic heart diseases. EEG biofeedback might benefit patients with cardiac events as it is associated with the central nervous system and reduces anxiety. Anxiety aggravates the heart condition of patients with coronary heart disease through sympathetic nervous system hyperactivity, increased heart rate, diminished autonomic control of the heart, and increased blood pressure variability, which eventually affects coronary endothelium and plaque formation (King 1997). The present study aims to discover the effectiveness of EEG biofeedback in influencing brainwaves by looking at the EEG frequencies and the distribution of alpha and beta waves during therapy as well as its capability to decrease anxiety levels after five concurrent sessions.

EEG biofeedback, or as its more commonly known, neurofeedback, has shown much clinical value in treating neurobiological disorders such as epilepsy (Kotchoubey

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et al 1999), attention deficit hyperactivity disorder (Masterpasqua and Healey 2003), and substance abuse (Peniston and Kulkosky 1989). Research shows that a number of these neurological and psychological disorders can be characterized by distinctive EEG patterns and that neurofeedback may help clients change their patterns (Masterpasqua and Healey 2003). However, studies have also focused on healthy individuals as EEG biofeedback, like mental skills training and aerobic exercise, was able to improve the performance of musicians (Egner and Gruzelier 2003).

Neurofeedback refers to an operant conditioning paradigm where participants learn to influence the electrical activity of their brain by regulating specific AC frequencies and the slow cortical potentials (SCPs) of the EEG (Vernon et al 2003). The electrical brain activity is used to control a computer, and this type of communication is usually called a brain-computer interface (Neuper et al 2003). Theta training was traditionally viewed as forming part of a relaxation inducing technique and was originally associated with meditation (Kassamatao and Hirai 1969). However, this form of training requires participants to relax with their eyes closed, receiving only auditory feedback. The most common indicators of relaxation are an increase in alpha frequencies and less complexity in EEG signals, and this establishes the positive effect of a mind machine (Stolc et al 2003). This study utilizes the less frequent beta training and its capability to bring about a mental state of brain relaxation as effective as the alpha and theta bands.

Methodology

Participants

Patients with confirmed unstable angina or myocardial infarction referred by the cardiologists were recruited 2–3 days after their cardiac event from the cardiology wards, National University of Malaysia Hospital (HUKM) and the Institut Jantung Negara (IJN). A cardiac event was based on typical clinical symptomatology, identified through ECG evidence and elevated serum levels of myocardial enzymes. These patients were entered into the study irrespective of age, sex, or ethnicity. All patients were given full details of the study at recruitment and were invited to participate. Written consent was obtained.

Psychological assessment

The Hospital Anxiety and Depression Scale (HADS) was administered at initial diagnosis of cardiac events and during follow-up three months later. The cut-off point indicates the range from normal to severe. The normal rate is below 8, while 8–10 indicates mild symptoms, 11–14 moderate symptoms, and a range of 15–21 points to a severe state of anxiety. For this study, severity of anxiety was limited to three groups, normal and severe representing both extremes, while mild and moderate were categorized as one level. The questionnaire was interview-aided and not self-rated, and only one interviewer conducted the questionnaire for all subjects to avoid interviewer bias. It was not a single-blind study as the interviewer was aware of the treatment conditions. Anxiety levels were used in this study to determine the effectiveness of the therapy. The HADS was chosen due to its practicality and expediency (Roberts et al 2001). Its most important feature was its capability to produce a separate score to establish the presence and severity of anxiety and depression simultaneously. The HADS was also practical in this study as the sample size was small and the scale was validated for use in non-psychiatric units, thus making it an acceptable instrument for cardiac patients.

EEG biofeedback training

Participants who returned for therapy received instrument feedback involving beta/sensory motor rhythms (SMR) training using commercially available biofeedback equipment (Procomp+/Biograph programme-Thought Technology Ltd, Montreal, QC, Canada) every two weeks for a total of 5 sessions. The biofeedback instrumentation provided a media of transferring information from these brainwaves. It worked on a reward basis in which positive feedback resulted in accumulated points, and negative feedback resulted in these points being withheld. A computer monitor was placed 150 cm from the patient. Both the visual feedback received through a video game-like display portraying a maze animation and audio feedback with the inclusion of music with changes in frequency, volume, and rhythm enabled the individual to respond by moving toward a better, learned, and voluntary-controlled function as they consciously directed their brainwaves. For feedback control, there were 3 bargraphs; beta (16–40 Hz), theta (4–7 Hz), and EEG (1–40 Hz). Bars were dark when the signal was within acceptable parameters and turned a bright color when they were not, to rapidly indicate which condition was not satisfied. When the beta signal was above threshold, the animation started and a song played while the counter accumulated points. When any of the three

signals got out of condition (beta below threshold, theta, or EEG [1–40 Hz] were above threshold), the animation and music stopped. The animation instrument comprising a maze was linked to this beta instrument.

EEG was recorded from channel C3 according to the International 10–20 system for all training. The reference electrode was placed at the left ear lobe and ground electrode at the right ear lobe. The system applied a variety of digital filters to the recorded signal to extract frequency-domain information. The subsets of the whole EEG bandwidth were filtered and the real-time changes in the total power generated by all the frequencies in the subsets were measured. Frequencies above 40 Hz were interpreted as EMG noise from the neighboring muscles. The component brainwaves of the EEG were separated into individual bands with alpha band defined as 8–12 Hz. Beta waves were categorized into low beta or sensory motor rhythm, SMR (12–15 Hz), beta (16–20 Hz), and high beta (21–40 Hz). Minimal configuration for monochannel EEG measurement consisted of 1 active electrode.

Procedure

Subjects received continuous feedback involving a completion of five sessions of EEG biofeedback training in the three months duration. The workings of the feedback loop were explained to all participants before the start of sessions and they were required to relax without falling asleep. In the training sessions, they were instructed on ways of improving their positive feedback and taught simple breathing exercises to use. Subjects were also required to practice the relevant relaxation and breathing exercises that provided positive feedback daily, preferably at a fixed time of day. The subjects underwent the whole length of the experiment period in a quiet but not a soundproof area, with no one present except for the examiner, and these conditions were preserved for all subjects as described by Teplan (2002). Changes in the EEG frequencies were measured by use of a task that required participants to sustain the 3 bar graphs displayed within the expected parameters described in the above section titled *EEG biofeedback training*. The first session and the last session were assessed to compare the progress made by the participants at the end of the therapy with the time of recruitment. Mean frequencies were compared for the entire session for the first and last session each participant underwent. Those that dropped out earlier did not receive any therapy. Anxiety levels for all patients (subjects and dropouts) were determined twice, once at time of recruitment (first screening) and at the follow-up assessment three months later (second screening). Within-group and between-group statistical comparisons were made using Student's t-test.

Results

Characteristics of the sample population

All 115 patients recruited into the study went through an initial psychological assessment in the first screening. Fifty (43%) of them returned to participate in the EEG biofeedback therapy. Four percent completed only 3 sessions, and 20% completed 4. The remainder (n = 38, 76%) completed the required number of 5 sessions. Mean time of completion for sessions was 7.20 minutes, with times ranging from 3.01 to 14.27 minutes. Forty-three percent of the sessions were completed within 5 minutes, with 55% exceeding 5 minutes. Only 2 sessions were completed in less than 5 minutes, with one taking 3 minutes and the other 4.

The remaining 65 patients that declined to participate in the program agreed to be reevaluated for the follow-up psychological assessment during the second screening. For the purpose of this paper, the first group of 65 patients that dropped out without any EEG biofeedback intervention will be identified as dropouts. The patients that consented to the therapy will be considered as subjects, with those successfully completing all 5 sessions as subjects 1, and those that dropped out after going through at least 3 sessions of EEG biofeedback sessions as subjects 2 (n = 12). Most of the subjects (subjects 1 and subjects 2) were male (68%),

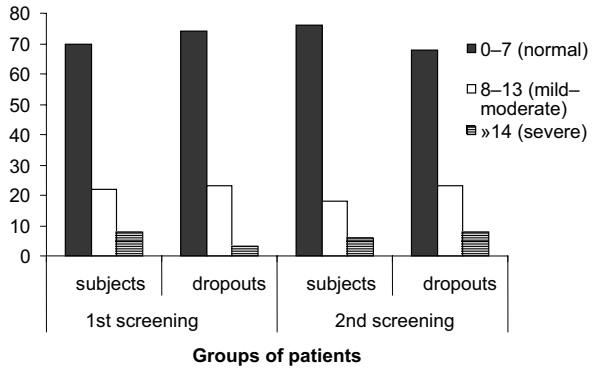

Figure 1 Percentage of patients with different levels of severity for anxiety. Levels of severity for anxiety based on Hospital Anxiety and Depression Scale. First screening is during recruitment; second screening is at follow-up assessment 3 months later. Subjects (n = 50) are those that consented to participate in the EEG biofeedback. Thirty-eight of the subjects completed all 5 sessions, and 12 of them dropped out after 3 or 4 sessions. Dropouts (n = 65) declined to participate in the therapy but agreed to be psychologically assessed.

Table 1 Changes in anxiety scores at second screening as compared with the first screening in subjects 1, subjects 2, and dropouts

	Patients recruited (n = 115)													
	Consented to treatment (n = 50)								Declined treatment Dropouts[c] (n = 65)					
	Completed 5 session Subjects 1[a] (n = 38)				Completed < 5 sessions Subjects 2[b] (n = 12)									
	Initial anxiety levels (based on anxiety scores at first screening)[d]													
	Normal (n = 27)		Mild to severe (n = 11)		Normal (n = 7)		Mild to severe (n = 5)		Normal (n = 48)			Mild to severe (n = 17)		
	Changes in anxiety scores at second screening as compared with first screening													
	↓	No	↓	No	↓	No	↓	No	↓	No	↑	↓	No	↑
n	16	11	5	6	3	4	4	1	5	15	28	4	4	9

[a] Subjects 1: patients recruited that returned to participate in the therapy and successfully completed all 5 required sessions.
[b] Subjects 2: patients recruited that returned to participate in the therapy and completed at least 3 sessions but not all of the 5 required sessions.
[c] Dropouts: patients recruited that did not return to participate in the therapy although agreed to be assessed for the follow-up psychological assessment at second screening.
[d] Anxiety levels: normal range (< 8) and mild to severe (≥ 8).
NOTE: ↓, those that decreased levels of anxiety scores; No, those with no changes in their anxiety scores; ↑, those that had increased levels of anxiety scores.

while dropouts had an almost equal distribution of both sexes; males (n = 30, 46%) and females (n = 35, 53%).

Anxiety levels

Most patients (subjects and dropouts) had normal anxiety levels (71%) at initial recruitment. Thirty-four (68%) of the subjects and 73% of the dropouts had normal levels of anxiety scores at first assessment (Figure 1). There was no difference for anxiety scores between subjects and dropouts for the first screening, but dropouts had significantly higher scores compared with subjects at the second screening ($p < 0.001$). At completion of therapy, subjects had significantly ($p < 0.001$) lower mean anxiety scores than at screening, while dropouts had significantly ($p < 0.001$) higher mean anxiety scores at second than at first screening.

The dropouts had more than half of those cases (n = 28, 58%) with initial normal range of scores and more than half of the initial mild to severe cases (n = 9, 53%) recording higher scores for the second screening, respectively (Table 1). A mere 14% of the dropouts decreased their anxiety scores. Neither of the subject groups (subjects 1 and subjects 2) had a single case that had increased anxiety scores for the second assessment. More than half of subjects 1 with normal anxiety levels in first screening (n = 16, 59%) decreased their scores while the remainder maintained their scores. Almost half of subjects 1 who initially scored in the mild to severe range lowered their scores (Table 1). Four of the 5 mild to severe cases in the subjects 2 decreased their scores. Mean and maximum value for subjects 2 with initial normal scores succeeded in decreasing their mean and maximum scores but not their maximum value (Table 2). Those who scored mild to severe levels of anxiety scores for dropouts and the subject groups all managed to decrease their minimum scores, but the most drastic change was for

Table 2 Anxiety scores for subjects 1, subjects 2, and dropouts for first and second screening

	Patients recruited (n = 115)											
	Initial anxiety levels (based on anxiety scores at first screening)											
	Normal (n = 82)						Mild to severe (n = 33)					
	Dropouts[a] (n = 48)		Subjects 1[b] (n = 27)		Subjects 2[c] (n = 7)		Dropouts (n = 17)		Subjects 1 (n = 11)		Subjects 2 (n = 5)	
Screening	1st	2nd	1st	2nd	1st	2nd	1st	2nd	1st	2nd	1st	2nd
Anxiety scores												
Mean	4	5	4	2	4	3	11	11	10	8	13	10
Minimum	0	1	0	0	1	0	8	5	8	0	8	6
Maximum	7	12	7	6	6	6	15	15	19	15	19	15

[a] Dropouts: patients recruited that did not return to participate in the therapy although agreed to be assessed for the follow-up psychological assessment at second screening.
[b] Subjects 1: patients recruited that returned to participate in the therapy and successfully completed all 5 required sessions.
[c] Subjects 2: patients recruited that returned to participate in the therapy and completed at least 3 sessions but not all of the 5 required sessions.

Table 3 EEG frequencies for alpha and beta waves in subjects 1 and subjects 2 for the first and last session

	Patients consented to treatment (n = 50)							
	Initial anxiety levels (based on anxiety scores at first screening)[a]							
	Normal (n = 34)				Mild to severe (n = 16)			
	Subjects 1[b] (n = 27)		Subjects 2[c] (n = 7)		Subjects 1 (n = 11)		Subjects 2 (n = 5)	
Session	1st	last	1st	last	1st	last	1st	last
Alpha frequencies (≥ 8 Hz), n	19	19	4	4	8	9	2	3
AMF, Hz								
Mean	12.8	15.5	10.3	14.2	16.1	21.5	7.8	9.0
Minimum	4.8	5.1	4.1	5.0	2.5	7.2	6.4	7.7
Maximum	27.2	28.9	24.5	26.8	27.3	28.9	8.7	11.0
Beta frequencies (16–40 Hz), n	6	11	1	2	5	8	–	–
BMF, Hz								
Mean	8.8	10.9	7.1	9.8	11.6	14.7	4.5	5.3
Minimum	3.5	3.7	2.3	4.1	2.2	4.2	4.1	4.5
Maximum	19.3	20.1	17.6	19.6	19.3	19.2	4.7	6.5

[a] Anxiety levels: normal ranges (< 8) and mild–severe (≥ 8).
[b] Subjects 1: patients recruited that returned to participate in the therapy and successfully completed all 5 required sessions.
[c] Subjects 2: patients recruited that returned to participate in the therapy and completed at least 3 sessions but not all of the 5 required sessions.
NOTE: Dashes denote data are not suitable for beta frequencies.
Abbreviations: AMF, mean alpha frequencies; BMF, mean beta frequencies.

subjects 1 that had a minimum score of 0 as compared with 8 in the first screening.

EEG frequencies for alpha and beta waves

To describe the performance over the training period, the online results of training sessions were shown as mean frequencies of both alpha and beta waves. Nineteen (70%) of the subjects that scored normal scores had high initial alpha waves for the first and last sessions. The normal initial scorers maintained their high alpha frequencies in the last session and those that did not have high initial frequencies were still unable to increase their frequencies. Nevertheless, 73% of those that scored mild to severe anxiety levels in the first screening also had high initial alpha waves. Furthermore, these participants had 1 person for subjects 1 and 2 persons for subjects 2 that succeeded in increasing their alpha frequencies by their final session (Table 3).

None of the subjects recorded high beta waves and instead most had very low beta frequencies of SMR. A mere 6 participants of subjects 1 with normal levels of anxiety scores recorded moderate levels of beta waves (16–40 Hz) at their first session. Nevertheless, 5 of the subjects 1 group and 1 of the subjects 2 group managed to increase beta waves from low frequency SMR waves to moderate beta frequencies. Three of the subjects 1 group with initial mild to severe anxiety scores increased their SMR waves to the preferred beta frequencies. All of these 3 lowered their anxiety scores to normal levels by the second assessment.

Discussion

As anxiety remains prevalent in patients after their cardiac events (Goodacre et al 2001), progress was indicated in the participants by their significantly reduced anxiety scores after five sessions of beta training. The ability to generate alpha brainwaves has been associated with the self-regulation of stress (Wacker 1996). Previous studies specifically linked anxiety and relaxation with EEG recordings (Isotani et al 2001) and found that an increase in alpha frequencies in the frontal scalp area is an indication of positive relaxation training effects of audiovisual stimulation (Teplan et al 2003) and is neuroprotective (Sterman et al 1970).

As a whole, most participants had normal anxiety levels and this probably had some effect on the results of the EEG recordings particularly the alpha waves. Although alpha changes are tightly linked to anxiety changes, this is only evident in high-anxiety subjects as discovered by Hardt and Kamiya (1978). Their study showed that some people with high levels of anxiety have low alpha waves, and EEG alpha increase is beneficial only for patients who exhibit this low amplitude alpha. Patients with anxiety who show abnormally

high levels of alpha at baseline readings do not respond as effectively to alpha increase biofeedback. Another important factor is that alpha waves attenuation is no longer evident with open eyes (Nakagome 2000), and our subjects were instructed to keep their eyes open throughout their sessions. Another study also found no stimulation on the alpha band for participants that were asked to remain awake but with their eyes closed (Schutter et al 2001).

Generally, patients undergoing beta training would have initial high beta frequencies of 22–40 Hz to be used for baseline comparisons. However, the beta bands for all our subjects during the recruitment stage were in the low SMR frequencies, which were below normal beta frequencies. This also suggests that these participants were not highly stressed at the initial diagnosis of their cardiac event during recruitment as excessive beta waves are associated with anxiety (Kiloh et al 1981).

The significant difference in the mean anxiety scores between the participants and the control group for the second assessment suggests that EEG biofeedback can effectively lower mild to moderate levels of anxiety, thus making it ideal to be used in nonpsychiatric units, ie, cardiac settings. Biofeedback as a low arousal training methodology has been previously efficient in treating anxiety disorders (Rice et al 1993). Inconsistencies in the different studies concerning anxiety reduction could be attributed to the way anxiety was assessed in individual patients and the actual length of treatment with longer periods of treatment reducing anxiety more effectively.

Detailed evaluation of each patient undergoing treatment would be a better evaluation of the characteristics and efficiency of the treatment. The Biograph program used for our study only required an electrode to be placed on only one active site in the scalp (C3) for EEG recordings. Most of the other similar studies recorded EEG from more than one active site according to the International 10–20 system. Furthermore, any increase in arousal affects the EEG frequencies in the entire region of the scalp and not just an isolated area (Barry et al 2004). Changes in arousal levels are linked with global activity while specific regional activity is linked with processing. A comprehensive EEG recording from the global activity might give more details on the effect of EEG beta waves training on anxiety levels.

Although we used an open and uncontrolled design, our study has a reasonably large sample size compared with some previous neurotherapy studies (Tebecis et al 1976; Bird et al 1978; Lubar JO and Lubar JF 1984; Wacker 1996; Miner et al 1998; Kim et al 2002), all of which had less than 20 participants. Nevertheless, criticisms of the small number of subjects have resulted in bigger sample sizes for more recent studies (Kaiser and Othmer 2000; Putnam 2000; Joyce and Siever 2001).

While the number of subjects participating was sufficient, the initial high dropout rate makes it difficult to conduct a double-blind study in which the one group of subjects are randomly assigned to EEG biofeedback and the other group to another type of biofeedback. Nevertheless, our study of 5 sessions of operant training of EEG activity was sufficient to produce significant changes in anxiety scores. Even in the subjects 2 group, those that dropped out by the third or fourth session were able to reduce their anxiety scores. A more detailed assessment would be to see the long-term effect of therapy, and evaluate whether a 3-month program could still have positive influence on anxiety 6 months or one year after completion of treatment.

Clinical implications

EEG biofeedback using beta training appeared useful in the treatment of anxiety based on the psychological assessment. Even so, the absence of significant associations between anxiety scores and EEG frequencies for either alpha or beta waves suggests that the improvement may be due to a placebo effect (Passini et al 1977). Although training involving EEG activation must be within a consistent therapy situation (Rosenfeld et al 1996), it is difficult to distinguish the effects of EEG training from confounding conditions like drowsiness, medications, caffeinated drinks, changes in emotional state/arousal, artifact from eye movements, time of day, and state of alertness. Therefore, these factors should be taken into consideration to eliminate the possibility of their influence on the positive effects resulting from intervention.

Nevertheless, EEG biofeedback provides accumulated benefits for some participants. The learning gained by the subjects during their training can be applied in their everyday life, as participants had their eyes open and learnt to stay relaxed while remaining alert and reducing their tendency to fall asleep. Biofeedback may also indirectly assist the participant to be better focused, more in control, and to feel clinically better.

The open and uncontrolled nature of our study militates against firm conclusions. However, the significant reduction in anxiety scores could lead to a big improvement in the morbidity and mortality rate of these patients as a further reduction was seen even in those scoring normal level scores

during initial recruitment. The increase in SMR waves by the end of the therapy suggested a higher level of concentration and alertness for participants as they underwent their relaxation and biofeedback therapy. This was evident in another study which showed that enhancing SMR frequencies into a relaxed state had been linked with significant behavioral and cognitive changes (Ramirez et al 2001). Further research, however, is warranted to evaluate beta training as an effective EEG biofeedback in producing positive results as an adjunct technique to improve the various relaxation techniques. In summary, beta training could be proposed as a promising approach.

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CHILD AND
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Neurofeedback with anxiety and affective disorders

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Compelling evidence exists for a neurophysiologic basis for obsessive-compulsive disorder (OCD). A large number of positron emission tomographic and single photon emission computed tomographic studies have found increased blood flow and metabolism in the mediofrontal, anterior cingulate, right frontal, or orbitofrontal areas [1–14], which implicates a cortico-striato-thalamocortical network. Functional abnormalities also have been documented in a large number of quantitative EEG (qEEG) studies [15–22] and evoked potential studies [23–27]. OCD seems to be somewhat heterogeneous, however, with at least two qEEG subtypes that have been found [17–21]. Prichep et al [20] and Kuskowski et al [15] found a group with excess alpha brain waves throughout most of the head, with frontal excess beta, whereas another subgroup has an excess of theta activity, particularly in frontal and posterior temporal areas. Clinical experience in conducting qEEG assessment with patients with OCD also has shown that excess beta activity is often found along the midline, in cortical areas approximately over the anterior cingulate.

Strong research evidence also indicates that there are functional brain abnormalities associated with anxiety and panic disorder [28–30] and post-traumatic stress disorder (PTSD) [31]. A particularly robust body of research, summarized by Davidson [32], has documented that depression is associated with an activation difference between the right and left prefrontal cortex. A large number of EEG studies, reviewed in earlier papers by Davidson [32–34], have established that the left frontal area is associated with more positive affect and memories, whereas the right hemisphere is more involved in negative emotion. A biologic predisposition to depression exists when there is a frontal asymmetry in

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brain wave activity, with more left frontal alpha activity. This imbalance with more left frontal alpha means that the left frontal area is less activated. Such persons may be anticipated to be less aware of positive emotions while at the same time being more in touch with the negative emotions that are associated with the right hemisphere. This asymmetry in EEG activity is best seen when the EEG is examined with an average reference or a reference on the vertex at Cz [32,35,36].

Researchers have observed for decades that individuals who are depressed are also typically withdrawn. We know that there is a neurophysiologic basis for such withdrawal. Henriques and Davidson [37] found that the frontal area in the left hemisphere is associated with approach motivation and behavior, whereas the right frontal area is involved in avoidance motivation and withdrawal behavior. Researchers found that when the left hemisphere is basically "stuck" in an alpha idling rhythm, there is not only a deficit in positive affect but also more withdrawal behavior. This biologic predisposition to depression is also firmly documented in research findings that have shown that infants of depressed mothers display this same reduced left frontal EEG activation [38,39], even as young as 3 to 6 months [40] and 1 month of age [41].

The belief has been expressed (J.H. Askew, unpublished data) [35] that this frontal alpha asymmetry may represent a state marker of depression, as well as reflecting a biological or trait marker of a vulnerability [37,42] to depression. This has been supported in a study (J.H. Askew, unpublished data) that found a strong correlation between alpha asymmetry scores and the Beck depression Inventory ($P < 0.0001$) and the Minnesota Multiphasic Personality Inventory (MMPI-II) depression scale ($P < 0.0001$).

Davidson [43], who has contributed more research in this area than any other individual, has expressed his belief that this asymmetry is not necessary or sufficient for the production of a specific type of affective style or psychopathology but that differences in prefrontal asymmetry are perhaps most appropriately perceived as diatheses that bias a person's affective style and then modulate someone's vulnerability to developing depression. He does not subscribe to a purely biologic model of depression, but he believes that the frontal alpha asymmetry does predict a vulnerability to depression so that when negative life events occur over a prolonged period of time to such an individual, there is an increased probability that he or she will become depressed. Based on his research, Davidson [43] believes that not everyone with this asymmetry will be depressed, despite being more vulnerable to becoming so, and someone can experience negative life events and still become depressed in the absence of this asymmetry.

The need for new treatments for depression, anxiety, and obsessive-compulsive disorder

Responding to these well-established biologic predispositions, there has come to be a strong reliance in psychiatry on the use of medication for the treatment of

depression and anxiety, although some evidence currently suggests that medication may not be as effective in treating these conditions as has often been believed [44–48]. Similarly, Greist [49] estimated the degree of symptomatic improvement in OCD from treatment with serotonin drugs to only be 30%. Goodman et al [44] similarly found that symptom amelioration in OCD treatment with serotonin uptake inhibitors is approximately 35% on average and that only 50% of patients experience this partial symptomatic improvement.

The Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) is widely regarded as the finest research measure of OCD. The mean from four separate samples [45,46] of patients with OCD on the Y-BOCS is 24.7 ± 6. A recent meta-analysis of 25 drug studies found that with the most effective pharmacologic treatment for OCD (clomipramine), the average drug treatment effect on the Y-BOCS was 10.64 (uncorrected for placebo effects; corrected for placebo effects it was 8.7), which is a 1.33 standard deviation improvement [50]. In fluoxetine (Prozac) studies, the mean Y-BOCS improvement was only 5.4 points (4.1 points when corrected for placebo effects). In OCD cases that are resistant to medication and therapy, psychosurgery has been the next treatment of choice. Even using a liberal criteria of 35% or more improvement on the Y-BOCS, however, cingulotomies have only benefited from one fourth to one third of patients [47,50], even with the confounding factor that most of the patients continued receiving pharmacotherapy after cingulotomy. Rauch [51] concluded that "For neurosurgical treatment of OCD, the overall rate of efficacy is quite modest, the costs are high, and the risks are considerable."

In light of this brief review and the fact that an increasing number of patients and parents seem interested in less invasive treatments, a need exists for nonmedication treatment alternatives that still address the underlying biologic factors associated with depression, anxiety, and OCD. We know that psychotherapy for depression compares favorably with medication in short-term follow-ups [52] and seems to be superior in long-term follow-ups [53,54]. With OCD, results from behavior therapy [55] are clearly superior to those found with medication, although there is a significant drop-out rate and behavior therapy methods are not well liked by patients. It would be desirable to find a treatment that also would help address the biologic aspects of mental health disorders. Neurofeedback holds promise for offering such an alternative.

What is neurofeedback?

Neurofeedback is EEG biofeedback or brain wave training. Neurofeedback training begins with a qEEG assessment. A tremendous body of research exists on the abnormal EEG and qEEG patterns associated with various medical and psychiatric disorders [48]. The assessment for neurofeedback training may consist of anywhere from 2 to 19 electrodes being placed on the head at standardized electrode sites to gather EEG data. These data then may be compared statistically to a normative database, which provides scientifically objective

information on how a patient's brain activity differs from age-appropriate norms. These data then guide the neurofeedback training process. During neurofeedback training, there usually are two electrodes placed on the scalp at locations where the EEG activity diverges most from norms. Reference and ground electrodes are placed on the earlobes. Nothing intrusive is introduced into the brain. The electrodes simply measure the ongoing brain wave activity. Ordinarily we are unable to reliably influence our brain wave activity because we lack awareness of it. When we are able to see representations of our brain wave activity on a computer screen a few thousandths of a second after it occurs, however, it allows us to modify our brain wave patterns through operant conditioning.

The patient is placed in front of a computer screen. The computer display may be as complex as a computer/video game type of display. It also may be as simple as two bar graphs, one representing slow and inefficient brain wave activity and the other representing efficient, beta brain wave activity. The patient concentrates on the screen. When the inappropriate activity decreases slightly and the appropriate activity increases slightly, a pleasant tone is heard. At first, changes in brain wave activity are transient. As sessions are repeated, however, and the therapist gradually modifies the thresholds for inhibiting inappropriate activity and reinforcing healthier brain wave activity, enduring changes are gradually conditioned. Research with uncontrolled epilepsy [56], for example, in which researchers have used pre- and post-sleep laboratory evaluations, has documented that positive changes in EEG activity after neurofeedback training are not just a voluntary waking change associated with learning a certain mode of concentration on a computer screen. The positive changes in reduced epileptogenic activity were seen even during sleep.

As is seen in other articles in this issue, EEG biofeedback (neurofeedback) has been found to be effective in modifying brain function and producing significant improvements in clinical symptoms in children, adolescents, and adults who have several different biologic brain disorders. These conditions include such things as epilepsy, attention deficit disorder and attention deficit hyperactivity disorder (ADHD), and learning disabilities and have included up to 10-year follow-ups of patients [57].

Neurofeedback treatment of depression, anxiety, and obsessive-compulsive disorder

Neurofeedback for anxiety and obsessive-compulsive disorder

A review of the literature on the neurofeedback treatment of anxiety disorders was conducted by Moore [58]. He was able to identify eight studies of generalized anxiety disorder, three studies with phobic anxiety disorder, two studies of OCD, and one report of using neurofeedback with PTSD. He noted several problems with this literature. One problem was that most of the research studies only used brief neurofeedback training in comparison with what clinicians tend to

do. For example, in the generalized anxiety disorder studies, treatment only averaged 3.2 hours, whereas clinicians often anticipate needing 7 to 12 hours of neurofeedback training with anxiety problems. The eight studies of generalized anxiety disorder also only averaged 6.25 subjects per study, but seven of the eight studies that he reviewed produced positive changes in clinical outcome.

The best studies of neurofeedback with anxiety were three outcome studies [59] with phobic (test) anxiety. These studies included random assignment, four alternative treatment control groups, and a wait-list control group. In one study, the group that received alpha EEG enhancement training produced 33% more alpha after treatment, and all three feedback groups (who received alpha enhancement biofeedback, electromyography [EMG] [muscle] biofeedback, and alpha plus EMG biofeedback) demonstrated significant reductions in test anxiety. In comparison, the untreated control group and the relaxation training group experienced no significant reduction. In another study, subjects received phases of alpha enhancement training and EMG biofeedback training. The alpha training was found to increase alpha production from 64% to 78%, and anxiety scores dropped significantly ($P < 0.001$) for this combined treatment group compared with a nontreatment group. Moore [58] concluded in his review that a placebo effect was present in these neurofeedback studies but that alpha and theta enhancement training provided additional effects beyond placebo and are effective treatments for anxiety disorders. When these results are compared with the American Psychological Association Clinical Psychology Division criteria [60,61] and comparable biofeedback specialty criteria [62] for evaluating the status of efficacious treatments, neurofeedback for phobic anxiety qualifies for the status of a probably efficacious treatment.

Before proceeding further, an outline of these guidelines for evidence-based support is reviewed. According to the biofeedback efficacy guidelines [62], the status of ''possibly efficacious'' is accorded for treatments that have been investigated in at least one study and had sufficient statistical power and well-identified outcome measures but lacked randomized assignment to a control condition internal to the study. For the last two decades, randomized, controlled trials have been emphasized as the scientific gold standard by the pharmaceutical industry, in medicine, and in the recent clinical psychology guidelines for defining empirically supported therapies. Recently, however, this academic ''gold standard'' has been challenged by two research reports in the scientifically prestigious *New England Journal of Medicine* [63,64] and another study [65]. The three studies discovered that results from nonrandomized observational studies were similar to randomized, controlled trials. To attain the lower evidence-based status of ''possibly efficacious,'' a randomized, controlled trial was deemed unnecessary.

The biofeedback efficacy guidelines define a treatment as meriting the status of ''probably efficacious'' when multiple observational studies, clinical studies, wait-list controlled studies, and intrasubject or within-subject replication studies demonstrate efficacy. A biofeedback treatment is considered to have reached the higher ''efficacious'' status when research by at least two independent research

groups (which has included comparison with a no-treatment control group, alternative treatment group, or sham/placebo control group with randomized assignment) has found that the experimental treatment is significantly superior statistically to control conditions or equivalent to a treatment of established efficacy. Finally, a biofeedback treatment is considered as having reached the status of ''efficacious and specific'' if, in addition to the previous criteria, the treatment has been demonstrated to be statistically superior to a credible sham therapy, pill, or bona fide treatment in at least two independent studies. With regard to requiring placebo-controlled studies to establish efficacy for psychological treatments, however, in which a known effective treatment is already available, this has been deemed unethical by medical ethicists [66,67] and by the Declaration of Helsinki of the World Medical Association [68]. Supporting the Declaration of Helsinki, a university Institutional Review Board (IRB) committee deemed that a study proposal to include a placebo control condition compared with neurofeedback to treat attention deficit disorder and ADHD would be considered unethical because a medication treatment with known effectiveness existed already for this condition [69].

Returning to the literature review, two relevant studies of neurofeedback for the treatment of anxiety were not reviewed by Moore [58]. Passini et al [70] used 10 hours of alpha neurofeedback training, comparing 25 anxious patients (23 of whom were alcoholics) with a control group of 25 anxious patients (22 of whom were also alcoholics), most of whom were seeking treatment at a Veterans Administration hospital brief treatment unit. While most subjects were assigned to one group or the other randomly, deliberate placement of younger patients in the control sample occurred toward the end of data collection and was implemented to offset an age difference that had developed earlier between the groups. Thus, this would be considered to be a matched control group study. Although they did not evaluate drinking status, the alpha neurofeedback training produced significant ($P < 0.001$) changes in state and trait anxiety compared with controls. This was accompanied by an increase in eyes-closed alpha production from 38% to 55%, whereas controls dropped slightly. An 18-month follow-up of those patients was published, with virtually identical results of lower anxiety still found, which validated that the anxiety changes from alpha neurofeedback were enduring [71]. A recent randomized, blinded, controlled study was conducted at London's Royal College of Music to evaluate the ability of alpha-theta neurofeedback to enhance musical performance in high talent level musicians when they were performing under stressful conditions in which their performance was being evaluated [72]. When compared with five alternative treatment groups, only the neurofeedback group that received training to increase alpha and theta resulted in enhancement of real-life musical performance under stress. These results qualify under the guidelines reviewed earlier as meeting probably efficacious status for neurofeedback treatment of anxiety.

Two neurofeedback outcome studies have focused on chronic PTSD, only the first of which was reviewed by Moore [58]. In a randomized, controlled group study [73], 30 30-minute sessions of alpha-theta EEG biofeedback training were

added to the traditional Veterans Administration hospital treatment that was provided to a group of 15 Vietnam combat veterans with PTSD. The study compared them after treatment and at follow-up with a contrast group of 14 veterans who only received traditional treatment. One strength of this study is that in addition to the posttreatment testing, on a monthly basis, patients and informers were contacted for a full 30-month follow-up period to determine if there had been PTSD symptoms (eg, flashbacks, nightmares, anxiety attacks, depression). At follow-up, all 14 traditional treatment patients had experienced relapse, whereas only 3 of 15 neurofeedback training patients had experienced relapse. Another outcome measure involved psychotropic medication requirements. Medications were equivalent at the onset of treatment, with 14 of the neurofeedback group receiving medication and 13 of the 14 standard Veterans Administration hospital treatment group on medication. All 14 patients who were treated with neurofeedback had decreased their medication requirements at follow-up, whereas in contrast, only 1 traditional treatment patient had decreased medication needs, 2 reported no change, and 10 required more medications. Changes on the MMPI may be seen in Figs. 1 and 2. Neurofeedback training patients improved significantly on all ten MMPI clinical scales—in many in-

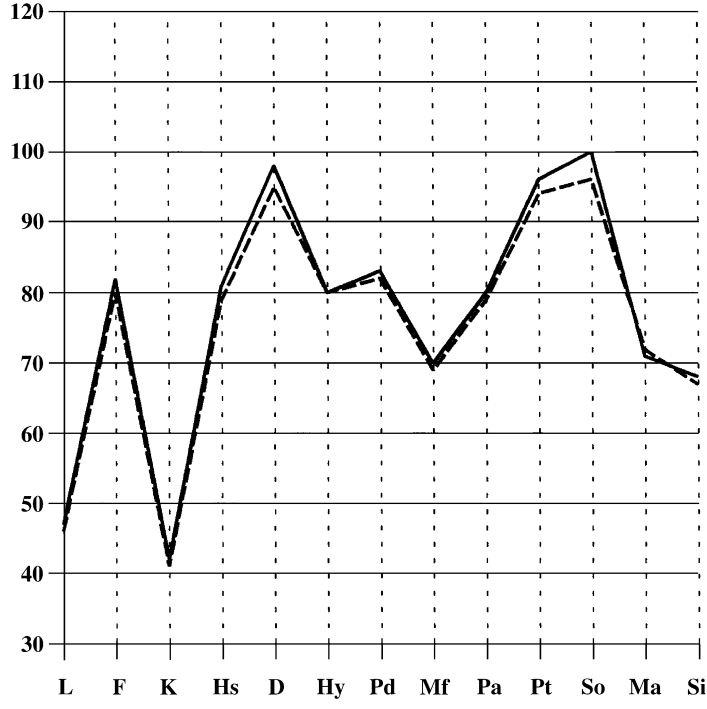

Fig. 1. Peniston-Kulkosky 1991 PTSD study. Pre- and post-MMPI changes from traditional treatment. Solid line indicates pretreatment. Dotted line indicates posttreatment.

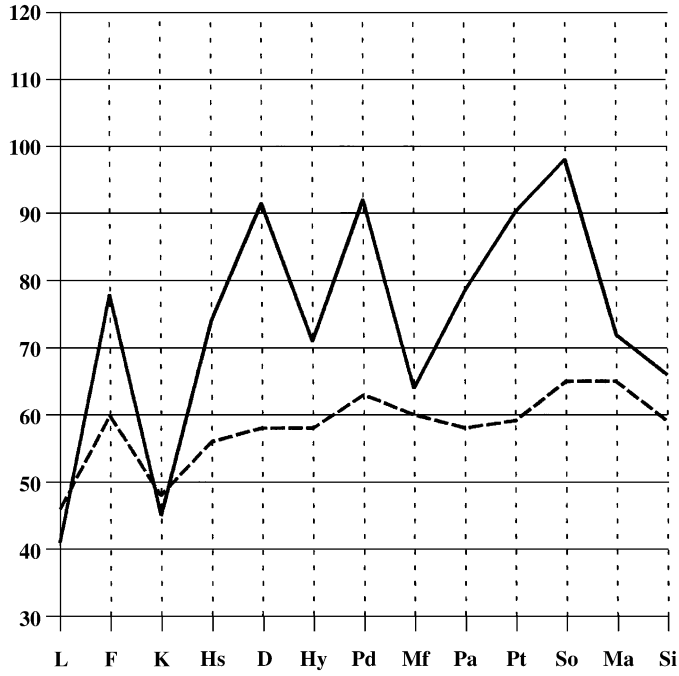

Fig. 2. Peniston-Kulkosky 1991 PTSD study. Pre- and post-MMPI changes after neurofeedback treatment. Solid line indicates pretreatment. Dotted line indicates posttreatment.

stances dramatically—but there were no significant improvements on any scales in the traditional treatment group.

In examining the figures, T-scores may be seen down the left hand side of each figure. A T-score of 50 represents the mean average of a ''normal'' population, and only 2.5% of normals score higher than the heavy line that goes across the figures at T-score 70. For readers unfamiliar with the MMPI, a brief overview of what the clinical scales measure is helpful. The first three scales (L, F, and K) are validity scales. When the F scale is elevated, as it is in these two samples, it is associated with an endorsement of more problematic symptoms. Scale 1 measures somatic symptoms. Scale 2 is the depression scale, and both treatment groups showed a severe level of depression before treatment. Scale 3 is associated with over-emotionality and repression. Scoring high on scale 4 indicates tendencies to be nonconforming, resentful of rules and authority, manipulative, and self-centered. Scale 5 measures traditionally masculine versus more feminine or more passive interest patterns. Elevations on scale 6 suggest that a patient is more paranoid, suspicious, hostile, and prone to project blame and responsibility. Scale 7 is associated with obsessive-compulsive symptoms, anxiety, and feelings of inferiority or inadequacy. Higher scores on scale 8 tend to be associated with being withdrawn, having odd or peculiar (thought disorder) thinking patterns, and feeling alienated from self and others. Scale 9, when it is elevated, can be

associated with impulsiveness, high energy level, or manic tendencies. Scale 0 is an introversion/extroversion scale, with elevations associated with being introverted and having a deficit in social skills.

In another Veterans Administration hospital uncontrolled study [74], 20 Vietnam veterans with chronic PTSD, all with comorbid alcohol abuse, were randomly selected. All patients showed frequent (eg, two to three times per week) episodes of PTSD symptomatology and had been hospitalized for PTSD an average of five times. They were treated with 30 30-minute sessions of alpha-theta neurofeedback training. Follow-up interviews occurred with the patients and their wives or family members on a monthly basis for 26 months. In that time, only 4 of the 20 patients reported a few (one to three) instances of recurrence of nightmares or flashbacks, and the other 16 patients had no recurrence of PTSD symptoms. The status of alcohol symptoms was not reported. According to the biofeedback efficacy criteria [62], neurofeedback treatment of PTSD qualifies for the status of probably efficacious.

Two published studies of OCD were reviewed by Moore [58]. Both studies used alpha enhancement training, without positive results. Viewed from a modern perspective, these studies, which were published in the mid-1970s, used a naïve and simplistic treatment approach of only doing alpha enhancement training. Literature since that time [17–21] has shown that there are at least two subtypes of EEG patterns that are found in OCD, neither of which would be anticipated to benefit from alpha enhancement training.

Recent reports are available on the successful treatment, with lengthy follow-ups, of three consecutive cases of OCD. In each of these cases, neurofeedback protocols were individualized to the unique neurophysiologic characteristics of each patient through using a qEEG assessment. In the first report [75], scores on the Y-BOCS and the Padua Inventory normalized after treatment, with the two patients improving on the Y-BOCS from scores of 26 and 25 to scores of 4 and 7 (showing 3.7 and 3 standard deviation improvements, respectively). This should be considered particularly significant because a meta-analysis of 25 drug studies found that even the most effective pharmacologic treatment for OCD only produced an average treatment effect on the Y-BOCS of a 1.33 standard deviations improvement (uncorrected for placebo effects) and approximately one half that much improvement across studies with fluoxetine (Prozac) [50]. Improvements also were documented with an MMPI, and follow-ups of the two cases at 15 and 13 months after treatment (which included interviews with relatives) found that changes were maintained.

A third case of neurofeedback treatment of OCD with a college student also has been reported [76]. The individual suffered with obsessional OCD, which is the type of OCD that has proven most resistant to cognitive-behavioral treatment [58]. He proved resistant to improvement with trials of eight previous medications. On his pretreatment MMPI he scored 115 T-scores on the Pt (7) scale. After treatment, his Pt scale decreased to 60 T-scores. Before treatment he scored 16 on the Y-BOCS, which is the cut-off score generally used for inclusion in OCD medication trials. On the obsessions subscale he scored 10; the mean for

patients with OCD is 10.7. At the completion of neurofeedback treatment, his Y-BOCS score had improved to 3 (a 2.2 standard deviation improvement) and his obsessions subscale score decreased to 0. Changes were maintained at 10 months, with external validation of improvements with his family.

All three of these cases had been treated unsuccessfully with various medications. In addition to these published cases, there are many clinical reports of comorbid OCD and ADHD improving with neurofeedback. Although these are uncontrolled case reports and do not yet even meet criteria for the status of a possibly efficacious treatment, the outcomes from treatment with neurofeedback in these preliminary reports are encouraging. The father of one of these patients, after having completed 21.5 hours of neurofeedback, said, "This week my daughter told me, 'Dad, for the first time in my life, I feel normal.'" The patient has been followed for more than 2 years and she has maintained her improvements.

Neurofeedback for depression

In relation to the research reviewed earlier on the presence of a frontal alpha asymmetry in depression, Rosenfeld [77] developed a neurofeedback protocol for modifying this asymmetry. This ALAY protocol (which stands for alpha asymmetry; F4 − F3/F3 + F4, with a reference electrode at Cz) has been used in case studies [35,36,78] with encouraging preliminary results, but no controlled research has been conducted. Baehr et al [78] did 1- to 5-year follow-ups on patients treated with the ALAY protocol and documented that the changes in depression were enduring and that the frontal alpha asymmetry not only had changed at the end of treatment but that this physiologic asymmetry continued to be reversed on long-term follow-ups. This is of particular relevance because several studies [42,79–81] have found that after pharmacologic treatment that produced a remission of depression, the frontal alpha asymmetry remained unchanged, which suggests that patients in drug treatment continue to have a biologic vulnerability to future depression.

A different protocol for modifying the frontal alpha asymmetry also was developed in association with a successful case report with an 8.5-month follow-up [82]. In this protocol electrodes are placed at Fp1 (on the left forehead) and F3 (approximately 2.5–3 inches straight above Fp1). During the training, slow brain wave activity is inhibited in the alpha and theta frequency bands during reinforcement of 15- to 18-Hz beta for the first 20 to 22 minutes of each training session, after which the reinforcement frequency band is decreased to 12 to 15 Hz for the final 8 to 10 minutes of each session. A 2-year follow-up of the initial case found that the depression remained in remission.

This second protocol has continued to be used clinically in the treatment of depression during the past 5 years, and there is a new report with a sample of nine consecutive patients who were treated with it [83]. All the patients in this series were relatively medication resistant and had been diagnosed with dysthymic disorder. They were all administered the MMPI and screened with the ALAY

protocol to verify the presence of the frontal alpha asymmetry associated with a biologic predisposition to depression. This screening takes approximately 15 minutes, and researchers have found that percentage scores of more than 60 indicate that there is no predisposition to depression, whereas scores of 58 or less indicate the presence of a predisposition [80]. The mean percentage score in the recent sample was 40.1, and their mean on the MMPI Depression scale (scale 2) was 93.8 T-scores. From the beginning, one patient seemed to have questionable motivation and dropped out after five sessions. The other eight patients received an average of 10.4 hours of training (20.8 30-minute sessions). No other psychotherapy was provided. After treatment, there was a mean decrease in the depression scale of 28.8 T-scores.

Improvement was categorized using the following criteria. Less than 60 T-scores on the depression scale was considered as representing normal, 60 to 70 T-scores represented mild depression, 71 to 80 T-scores represented moderate depression, 81 to 90 T-scores represented serious depression, and 91 T-scores and above represented severe depression. According to these criteria, overall this was a severely depressed patient sample. One patient was judged to have improved from being severely depressed to being normal, and two improved from being seriously depressed to normal. Three of the patients were judged to have improved from a severe to a mild level of depression, and one improved from moderately depressed to mildly depressed. In one case, a severely depressed individual only manifested mild improvement. He had lost his wife to cancer a year earlier, and this loss seemed to need further attention. He was referred for more traditional psychotherapy. All the patients had been treated with several antidepressant medications without substantive effect, and most of the patients were on medication at the beginning of neurofeedback training but not at the conclusion. The average length of individual follow-up of the eight patients was 1 year (range, 4 months to 2 years), at which time improvements had been maintained. Classifying the patient who only mildly improved as a failure, 87.5% of the cases improved, and if the drop-out is included as a failure, then 77.8% of the case series made significant improvements.

Patients in many of the published medication studies are moderately depressed, whereas in this case series, seven of the eight patients were classified as seriously to severely depressed, and only one patient was moderately depressed. The cases in the ALAY protocol studies [83] were only in the mild range of depression, with scores in the 62 to 64 T-score range on the MMPI, which also is reflected in their ALAY scores, which averaged 51.3, whereas the case series reported by Hammond [83] had a mean ALAY score of 40.1.

Although reports to date on the application of neurofeedback to depression only represent uncontrolled case reports that are not sufficiently rigorous to receive one of the levels of evidence-based support, they provide encouragement that neurofeedback may hold potential for treating mildly to severely depressed patients and that unlike medication, it may enduringly modify the functional brain abnormality associated with a biologic predisposition to depression. Controlled research seems warranted.

Clinical experience and further case examples

Depression

A case example illustrates the use of this second neurofeedback protocol with depression. Dan was an engineer in his 30s. He had originally entered treatment for a circumscribed complaint of fear of public speaking, which had been successfully treated in five sessions with self-hypnosis training. A year later he returned and indicated that he had experienced depression for many years but that it had been getting worse. His ALAY score of 36.1% indicated an extreme frontal alpha asymmetry, and his MMPI depression scale of 92 T-scores confirmed his severe depression. After informed consent, neurofeedback was started with the depression protocol. After three sessions he said that despite having had a difficult week at work, "I have been feeling a lot better. It's hard to believe that it's working this quickly." He explained that he had been skeptical about the possibility of neurofeedback being successful and was particularly surprised that he already could feel a difference. In clinical experience with this protocol, most patients can begin to perceive a difference in their depression level after three to six 30-minute training sessions. Usually by 10 to 12 30-minute sessions they feel significant improvement, and by 20 to 22 sessions treatment is completed.

Dan indicated after five sessions that he was still feeling depressed but that it was improving. After seven sessions he reported sleeping better, and after eight sessions he said that several people at work had commented on seeing a difference in him and had said, "We were worried about you there for a while." He explained that previously he had attributed his depression to his work situation but that his work had not changed and his depression was much improved. He continued to improve steadily. His total treatment consisted of 19 30-minute neurofeedback training sessions. Fig. 3 displays his before and after MMPI changes. His depression (scale 2) had decreased from a severe level (92 T-scores) to a mild, perhaps subclinical level (63 T-scores). The rest of his MMPI profile reflects changes that have been found in most cases after using this treatment protocol. His anxiety, obsessional rumination, and feelings of inferiority and inadequacy (as reflected in scales 7 and A) decreased, whereas ego strength (Es scale) increased. His withdrawal and feelings of alienation from people (scale 8) decreased and he changed from being moderately introverted and quiet (scale 0) to being on the mean between introversion and extroversion. The MMPI has proved to be a much better outcome measure than using a depression scale alone because it has illuminated the many other dimensions on which change has occurred. On the MMPI, a decrease in withdrawal and introversion (scales 8 and 0) commonly accompany the decline in depression, which would be anticipated because an area of the brain is being activated that is also associated with approach motivation. Dan's changes were maintained at 6.5-month follow-up, at which time he took a new job in another state.

Based on clinical experience with more than 25 patients with dysthymia, in which most of them have been followed for between 6 and 24 months,

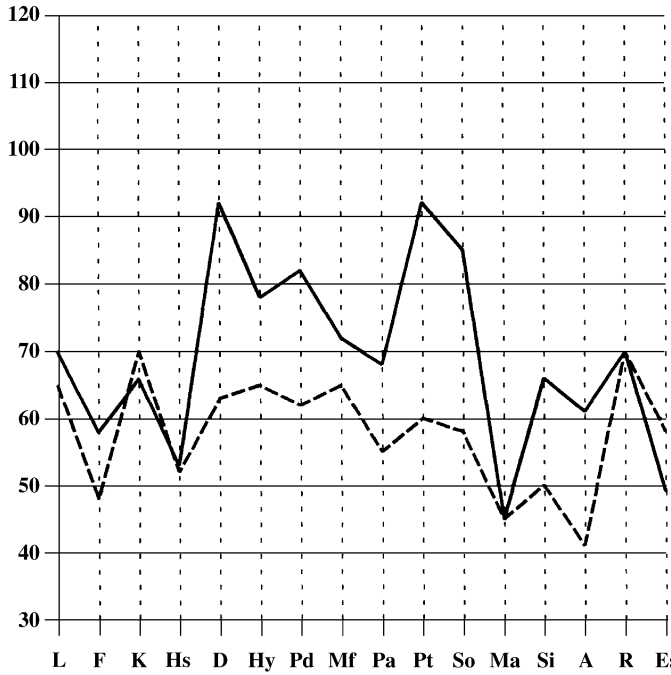

Fig. 3. Pre- and post-MMPI changes after neurofeedback for depression. Solid line indicates pre-treatment. Dotted line indicates posttreatment.

neurofeedback has seemed to be successful in producing significant and enduring change in approximately 80% of the patients. There have been no published research or clinical reports on the use of neurofeedback in a pediatric depression sample. Because the biologic marker of a frontal alpha asymmetry has been found in multiple studies with children and infants [38–41] of depressed mothers, and because there is abundant evidence that children respond to neurofeedback training for other conditions, it is reasonable to expect that this approach would be beneficial with depressed children. There are widespread clinical reports of improvements in mood among children treated with neurofeedback for ADHD, which further supports the expectation that neurofeedback may be effective with childhood depression. There also are anecdotal reports of improvements in bipolar disorder. Neurofeedback seems to involve minimal risk of side effects or adverse reactions [84], and it is less invasive than antidepressant medication or transcranial magnetic stimulation.

Anxiety and insomnia

In most cases, anxiety and insomnia are readily treated with neurofeedback [58,59,85–88]. Many children with ADHD are treated by inhibiting slow brain wave activity (eg, theta) while reinforcing the sensorimotor rhythm (12–15 Hz)

over the sensorimotor strip. In these cases, one of the first improvements that parents often notice is that the child falls asleep more easily and remains asleep. The reason may be that the sensorimotor rhythm overlaps in frequency with beta spindles, and when the sensorimotor rhythm is increased in a waking state, it also increases beta spindles that occur as one begins trying to go to sleep [87,88], which facilitates improvement in insomnia. Although anxiety often involves excess right frontal beta brain wave activity, clinical experience has shown that a qEEG assessment is often invaluable because the excess of fast beta activity may be in other locations. Someone who obsessively worries may have this beta excess along the midline or in the center of the top of the head at an electrode location known as Cz. In other cases, the excess beta may be in the parietal area. With anxiety patients, neurofeedback training often is done eyes closed while listening to auditory feedback, and in a sense it resembles high-technologic meditation training.

As a case example, a patient was referred by a physician who was a headache specialist, indicating that everything that could be done with medication seemed to have been done. The patient had a lengthy history of several migraines weekly, which had progressed to daily migraines. She had been given a self-hypnosis tape to use for anxiety management, but she complained that her mind was so busy that she was unable to obtain much relaxation from the tape. After 20 30-minute sessions of inhibiting fast beta and reinforcing alpha activity in the parietal area, she was off all her prescription medications. She sensed a migraine trying to begin approximately twice weekly but would take over-the-counter medication and could use the self-hypnosis tape successfully to abort the headache. She felt more relaxed in general and reported no longer feeling compelled to do two things at once.

Summary

As reviewed in other articles, the neuroscience technology known as EEG biofeedback (or neurofeedback) has considerable research support in areas such as uncontrolled epilepsy and attention deficit disorder and ADHD. In evaluating the studies in the overall broad area of the neurofeedback treatment of anxiety disorders, EEG biofeedback qualifies for the evidence-based designation of being an efficacious treatment [62]. When separate anxiety disorders are individually evaluated, the areas of phobic anxiety, generalized anxiety, and PTSD each qualify for designation as being a probably efficacious treatment. Currently there are only reports of cases and series of cases on the treatment of depression and OCD and no published reports thus far on treatment of bipolar disorder. Despite the lengthy follow-ups and use of objective measures, neurofeedback treatment for depression and OCD is not yet empirically supported.

EEG biofeedback is an exciting, cutting-edge technology that offers an additional treatment alternative for modifying dysfunctional, biologic brain patterns that are associated with various psychiatric conditions. It has the advantage of

not being as invasive as medication, transcranial magnetic stimulation, or electroconvulsive therapy, and it has been associated with few side effects or adverse reactions [84]. Frank H. Duffy, a professor and pediatric neurologist at Harvard Medical School, said that scholarly literature suggests that neurofeedback "should play a major therapeutic role in many difficult areas. In my opinion, if any medication had demonstrated such a wide spectrum of efficacy it would be universally accepted and widely used." "It is a field to be taken seriously by all" [89]. Duffy further pointed out that the field of neurofeedback also must produce more randomized, controlled studies. The efficacy of other biologic treatments (eg, medication and transcranial magnetic stimulation) for anxiety and affective disorders in children is not fully established, and many parents and patients increasingly seek less invasive treatment alternatives. It is desirable that more funded research be directed to providing further research evaluation of the potential of neurofeedback as a treatment with adults and children. Clinicians who are interested in learning more about neurofeedback training and qEEG may contact the International Society for Neuronal Regulation (www.isnr.org) or the Association for Applied Psychophysiology and Biofeedback (www.aapb.org).

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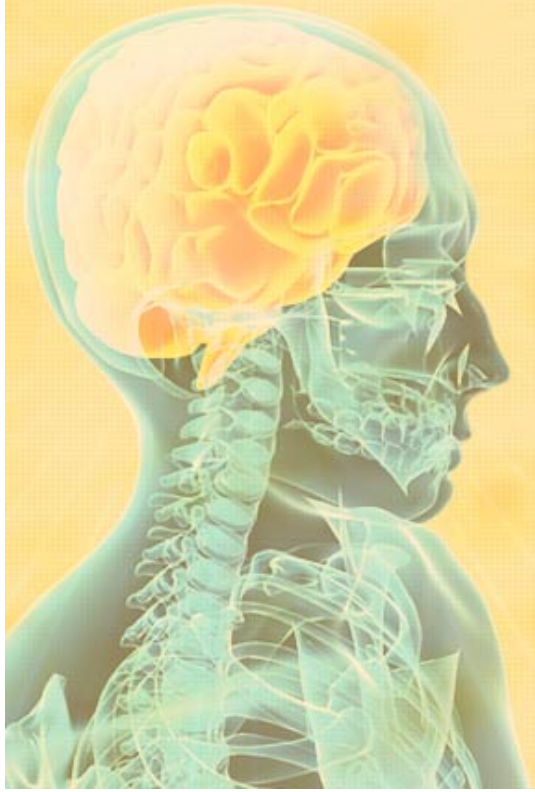
Effects of NeurOptimal Neurofeedback on Symptoms of Depression and Anxiety



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Kingston, Ontario, Canada

Kingston Institute of Psychotherapy & Neurofeedback

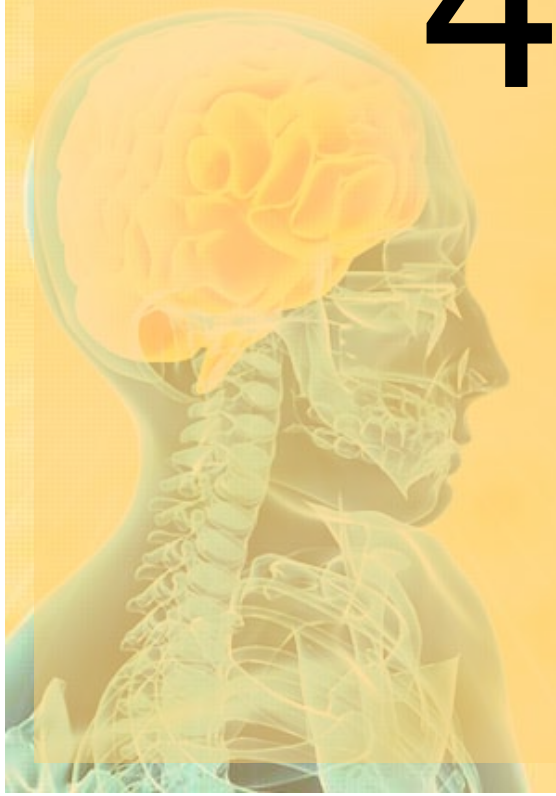
Janet McCulloch MD, FRCP
Linda Beckett MD, CCFP

Kingston Institute of Psychotherapy and Neurofeedback

- Roots in Psychotherapy
- NF Clinic Established 2010
- Initially 4 systems
- Currently 8 NF rooms with 10 NeurOptimal systems
- Training 350 to 400 people per week
- Staff: 2 Physicians, Nurse Specialist,
- 2 MSW therapists, 8 technicians
- Also: HRV, HEG, BAUD, QEEG & Targeted NF
- Volunteer run Yoga, Meditation, Cranial Sacral Therapy

45,000+

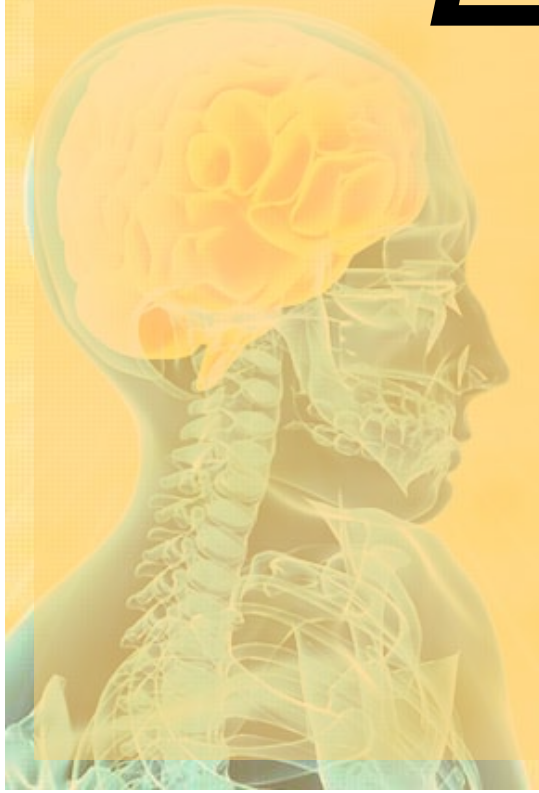
NeurOptimal Sessions



Data at Sessions 0 and 8

Who did we include?

Everyone



- ALL Adults 18+
- ALL diagnoses
- ALL length of illness

Beck Depression Inventory

- 21 groups of statements
  - (0) I do not feel sad.
  - (1) I feel sad much of the time
  - (2) I am sad all the time.
  - (3) I am so sad or unhappy that I can't stand it.

- Higher scores = more severe symptoms
  - 0–9: minimal depression
  - 10–18: mild depression
  - 19–29: moderate depression
  - 30–63: severe depression.

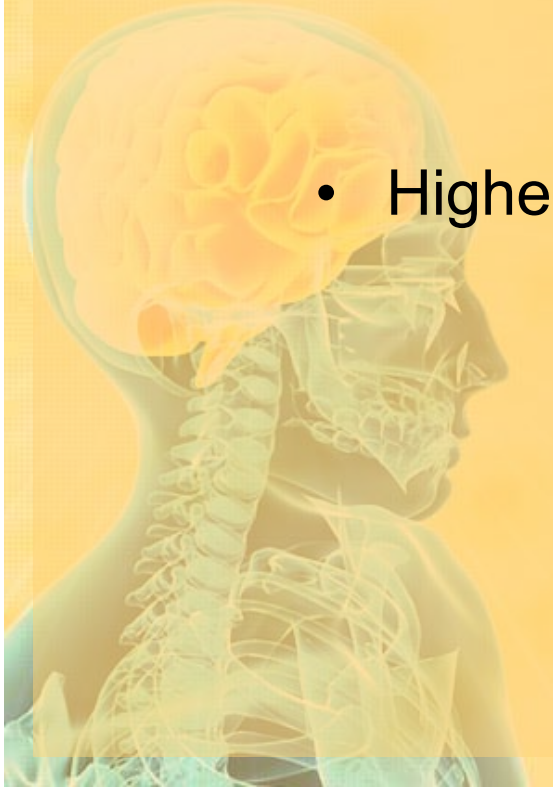
- .
-
- Beck Anxiety Inventory

Beck Anxiety Inventory

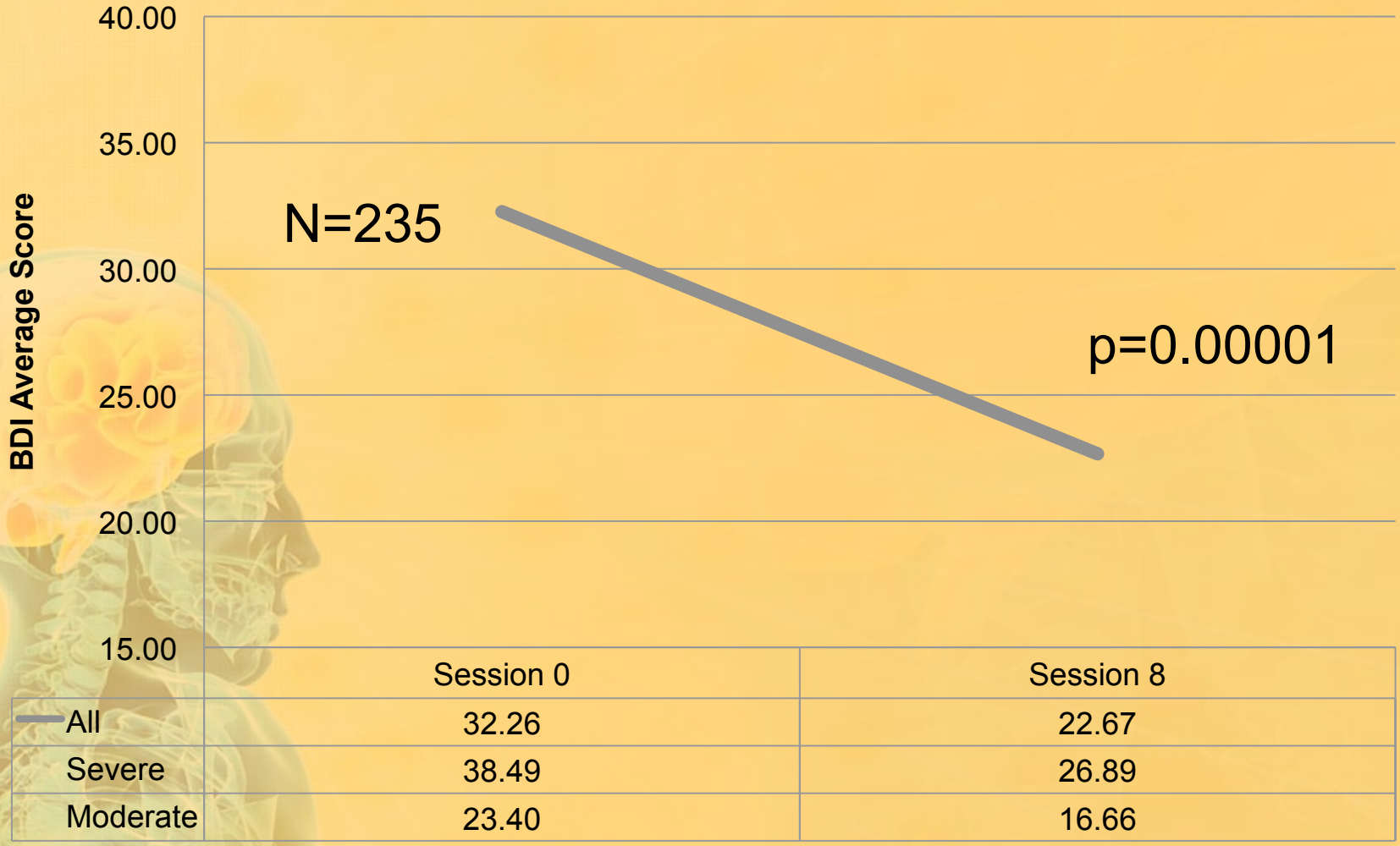
- 21 cognitive and somatic symptoms
- (0) Not at all
  (1) Mildly (It did not bother me much)
  (2) Moderately (It was very unpleasant, but I could stand it)
  (3) Severely (I could barely stand it.)

- Higher scores = more severe anxiety

0-7: minimal level of anxiety
8-15: mild anxiety
16-25: moderate anxiety
26-63: severe anxiety

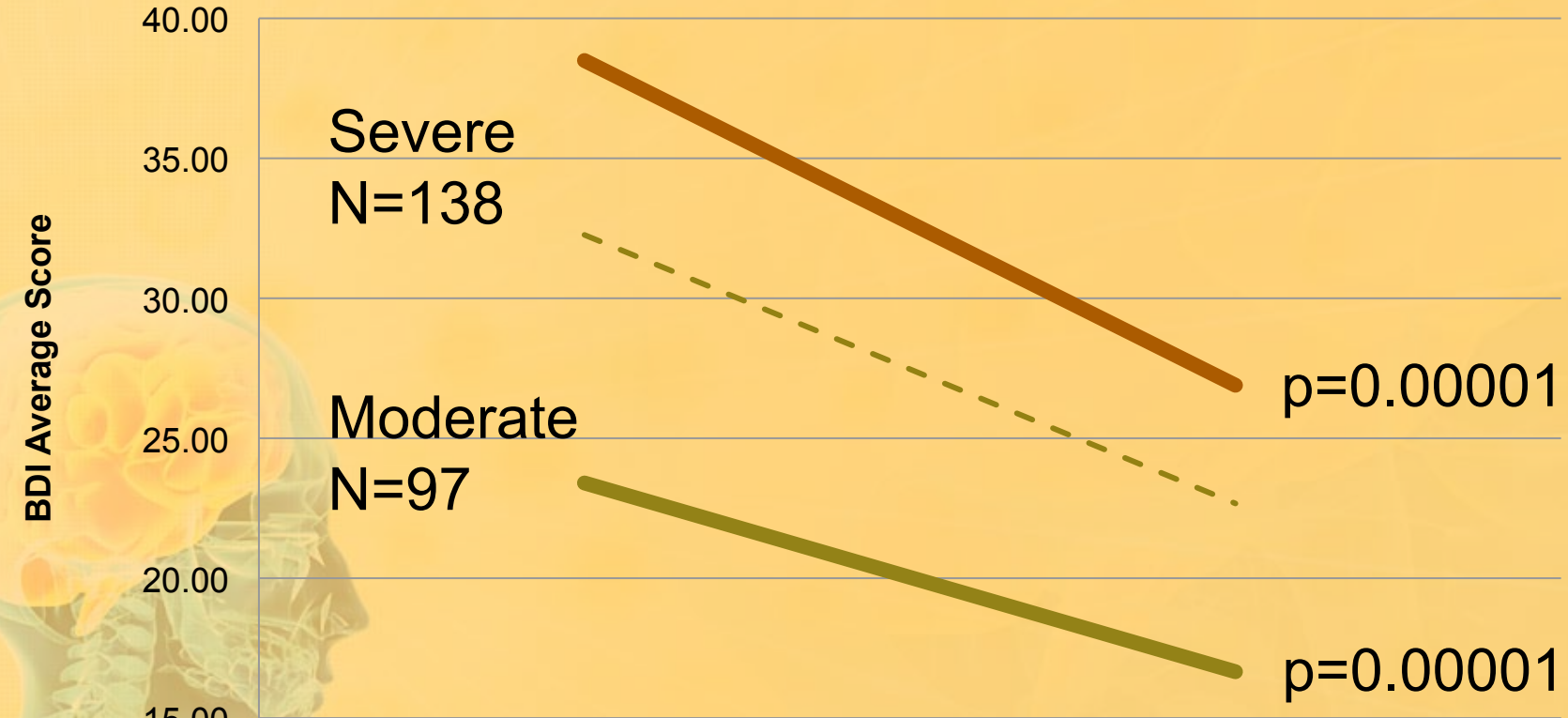


Beck Depression Inventory Results



	Session 0	Session 8
All	32.26	22.67
Severe	38.49	26.89
Moderate	23.40	16.66

Beck Depression Inventory Results



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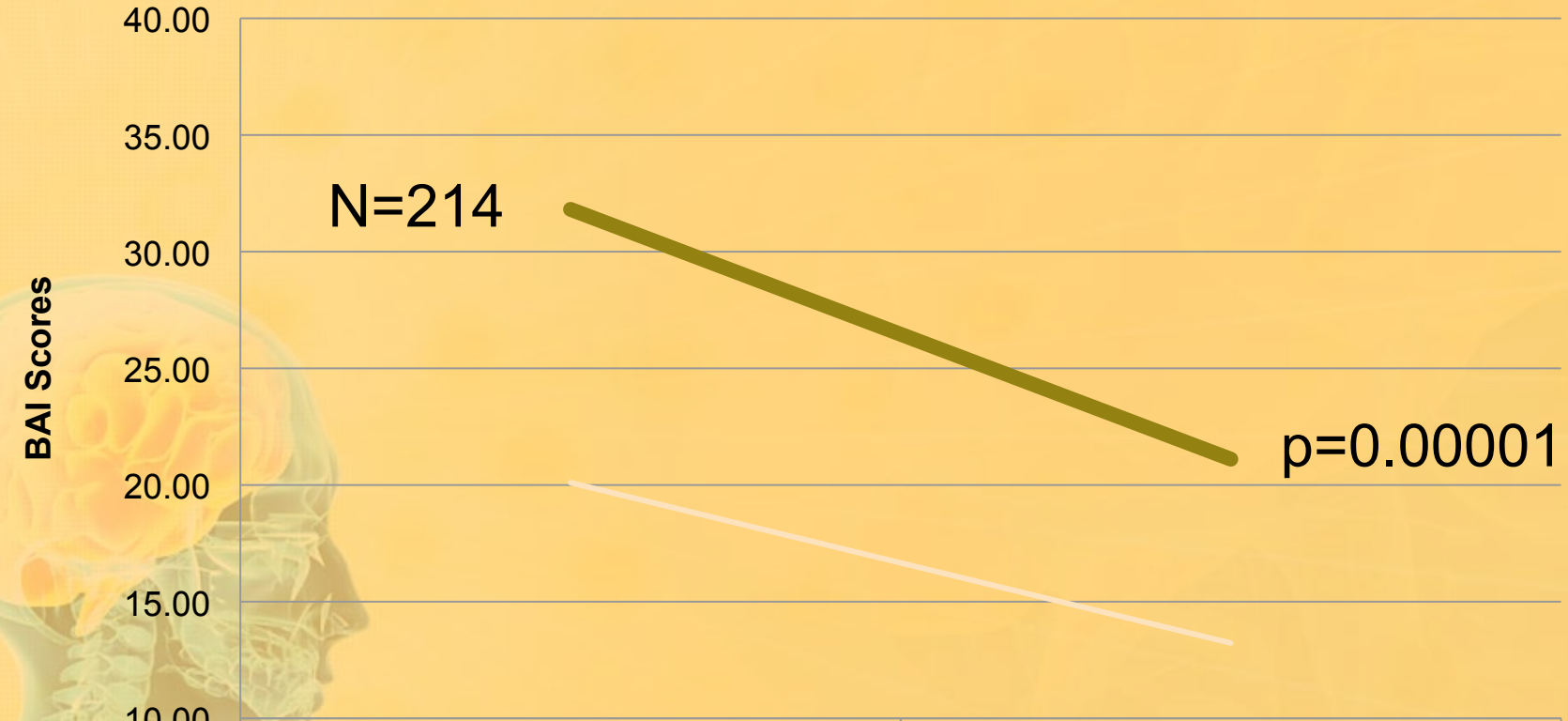
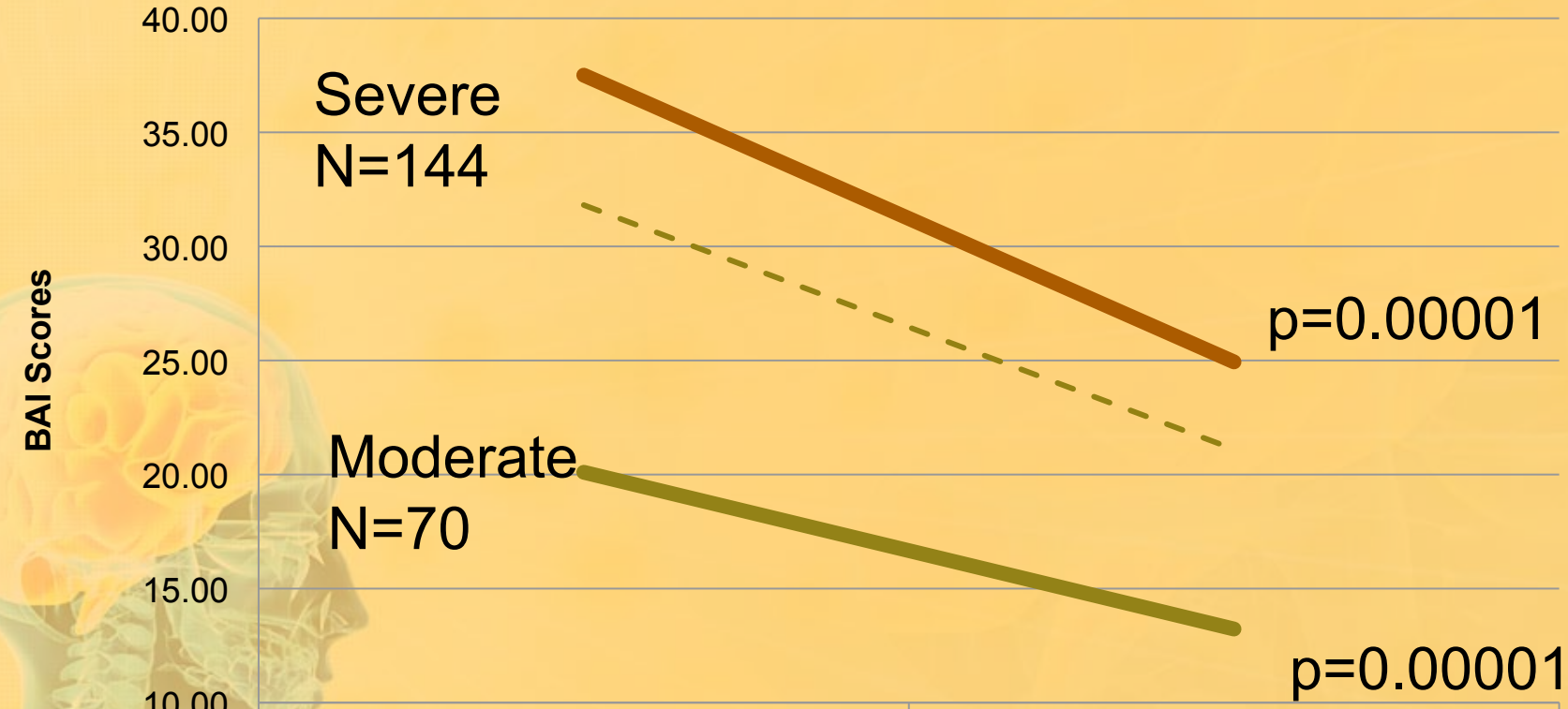
Beck Anxiety Inventory Results



	Session 0	Session 8
All	31.81	21.11
Severe	37.51	24.94
Moderate	20.10	13.23

Beck Anxiety Inventory Results



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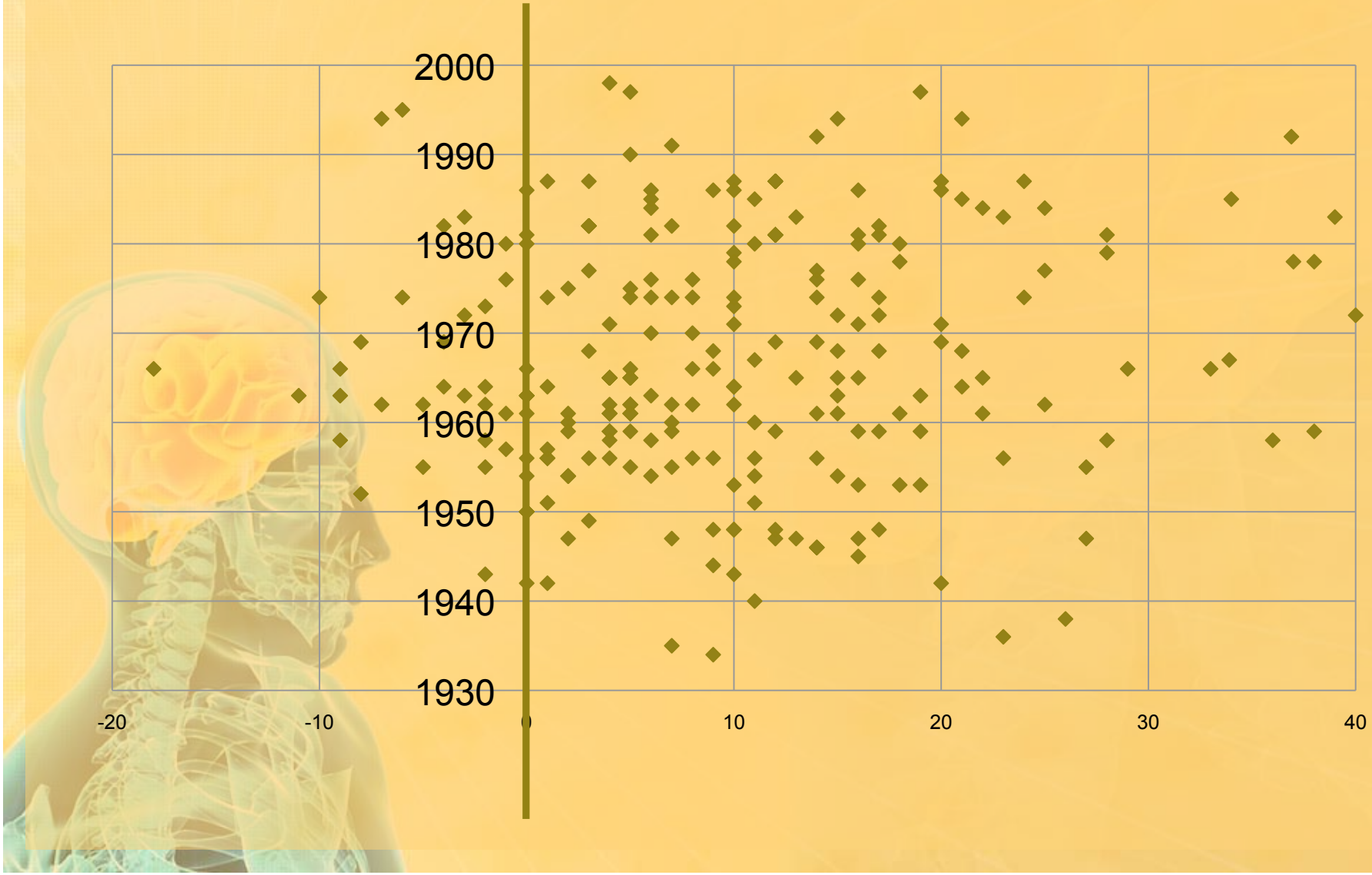
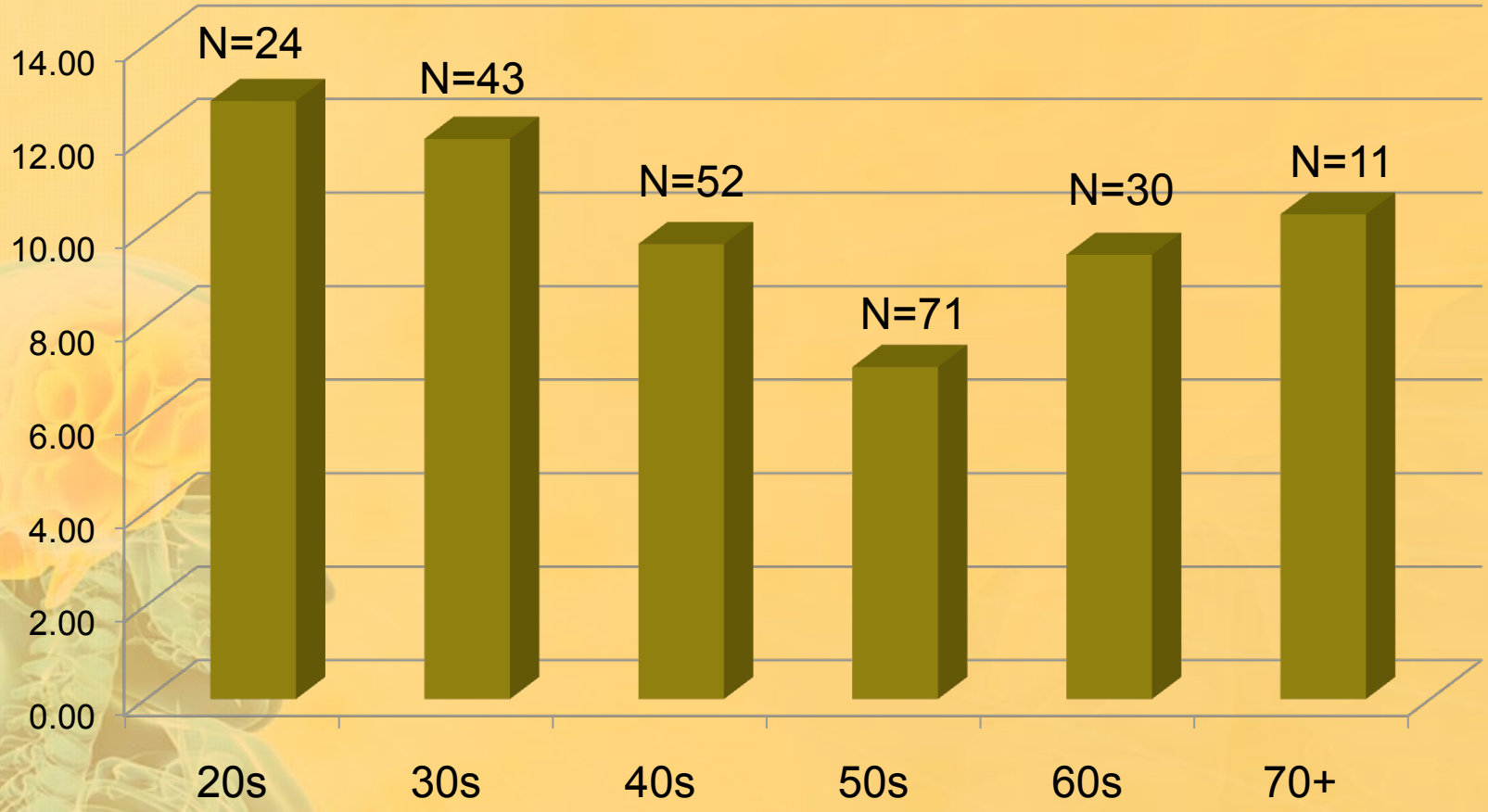
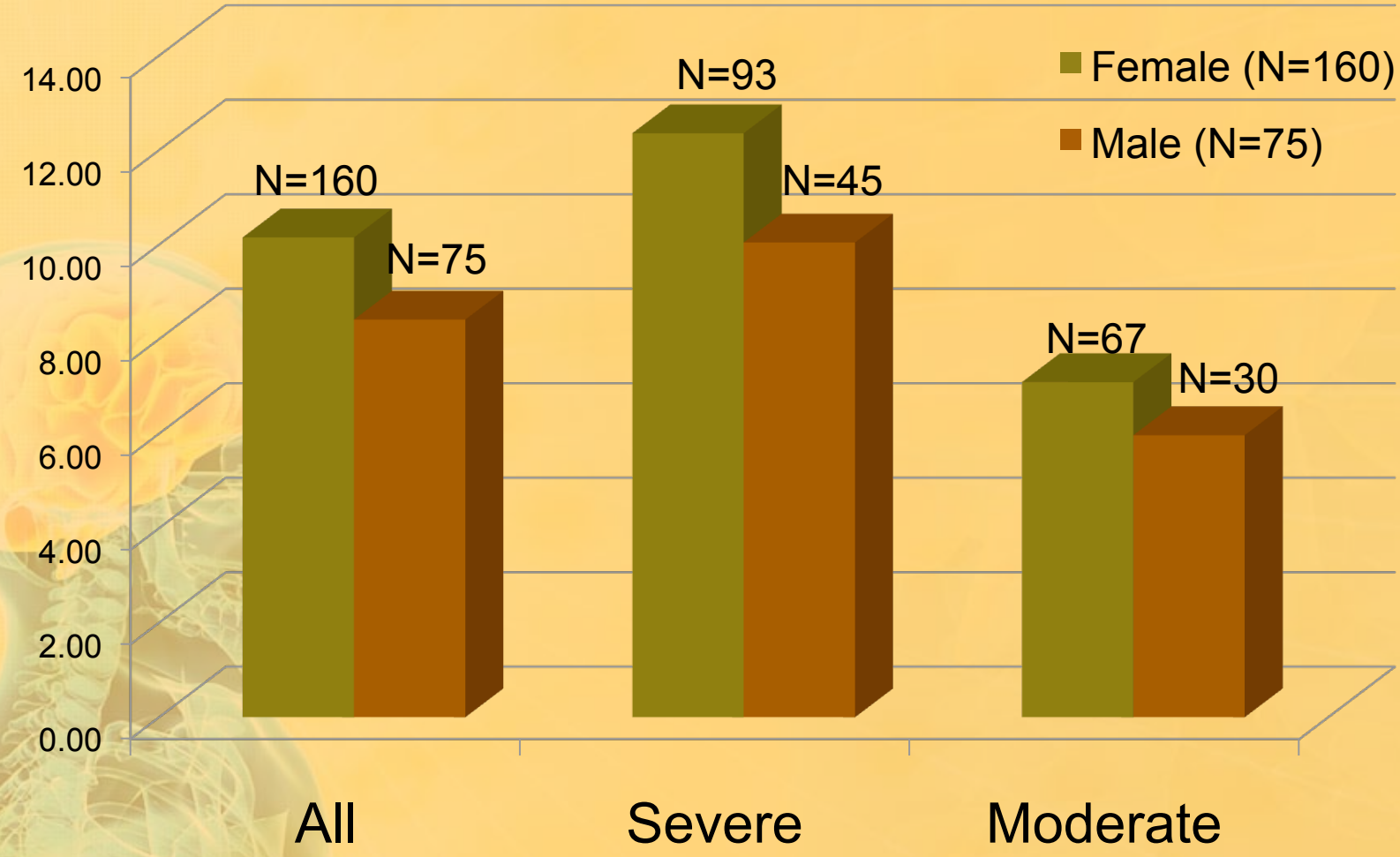
BDI Score Improvement from Session 0 to 8



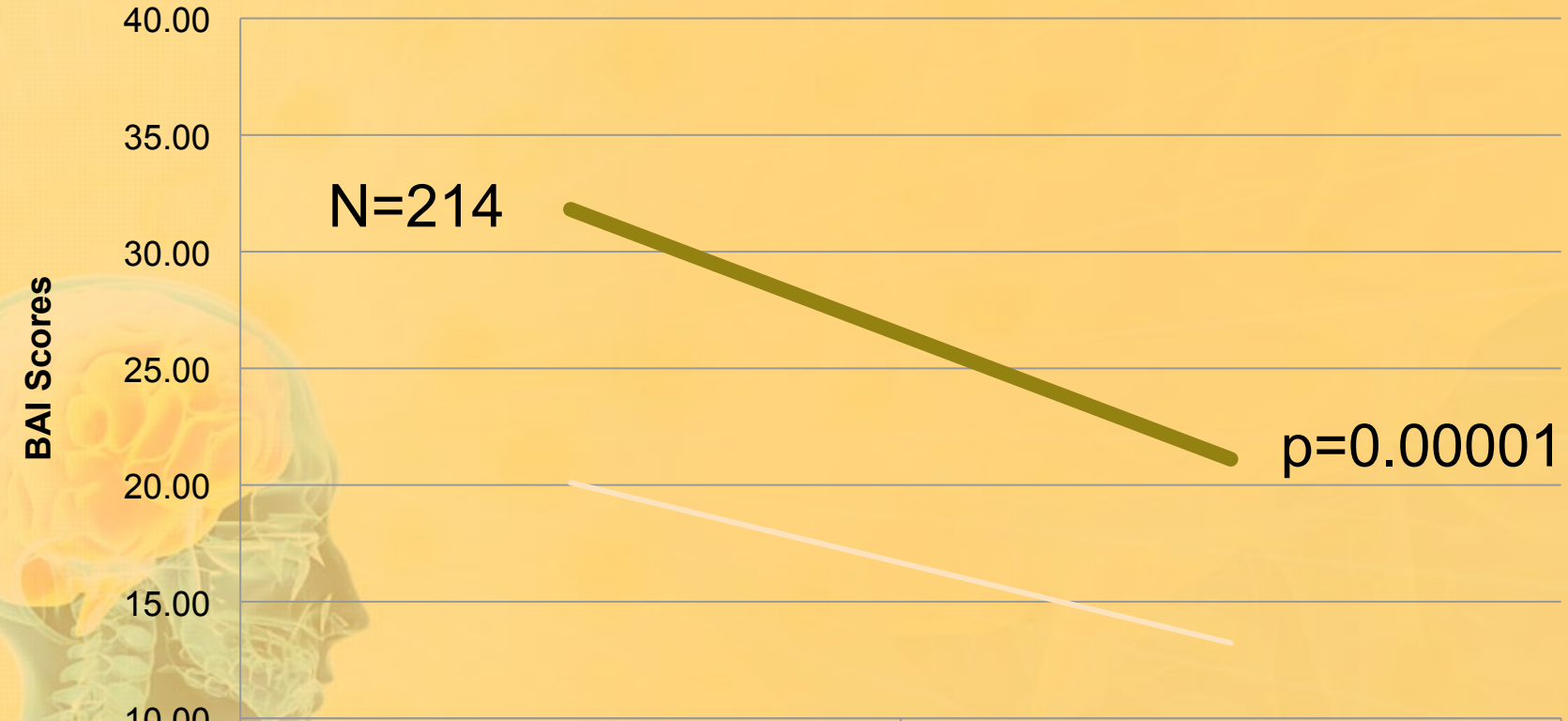
Depression Score Improvement by Age



Depression Score Improvement by Gender



Beck Anxiety Inventory Results



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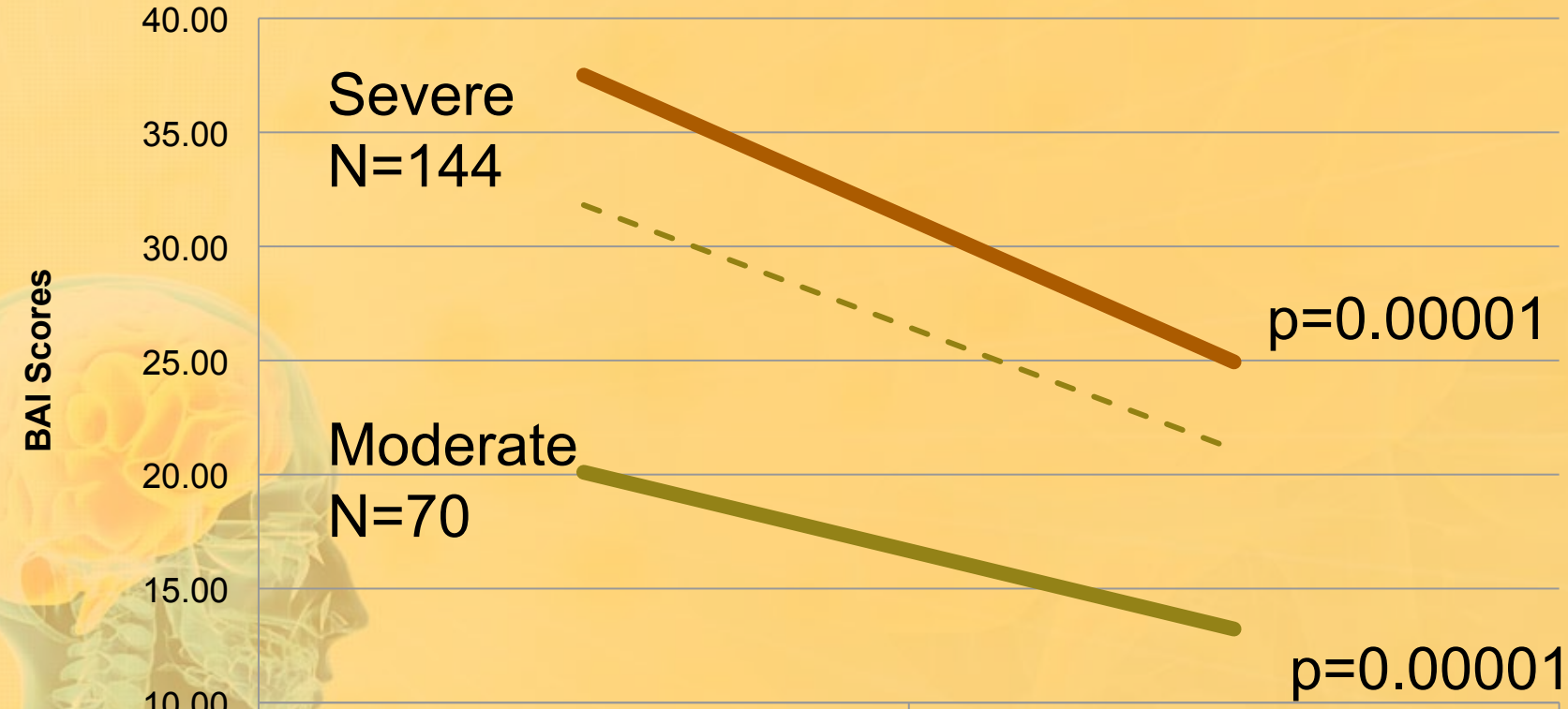

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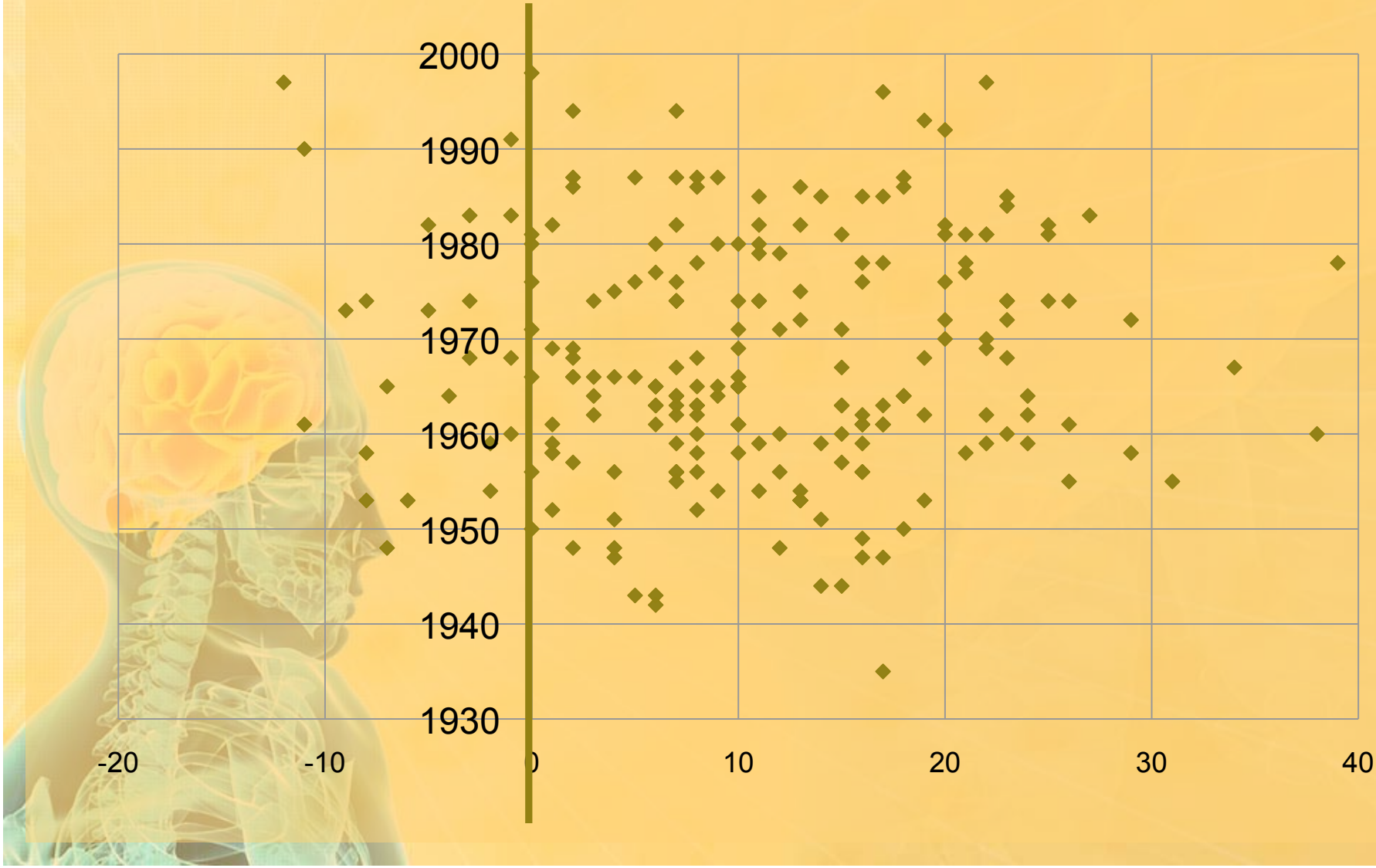
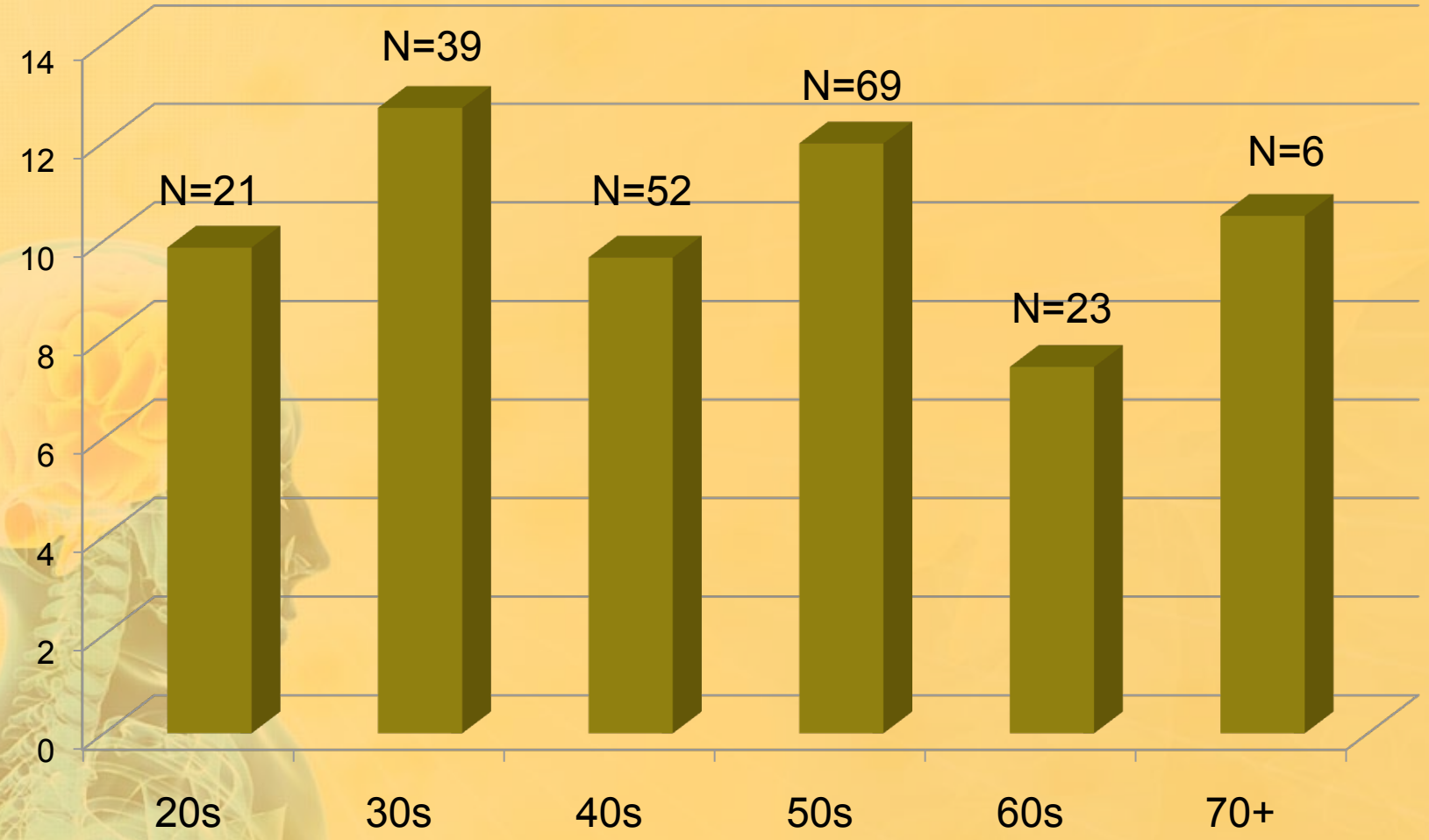
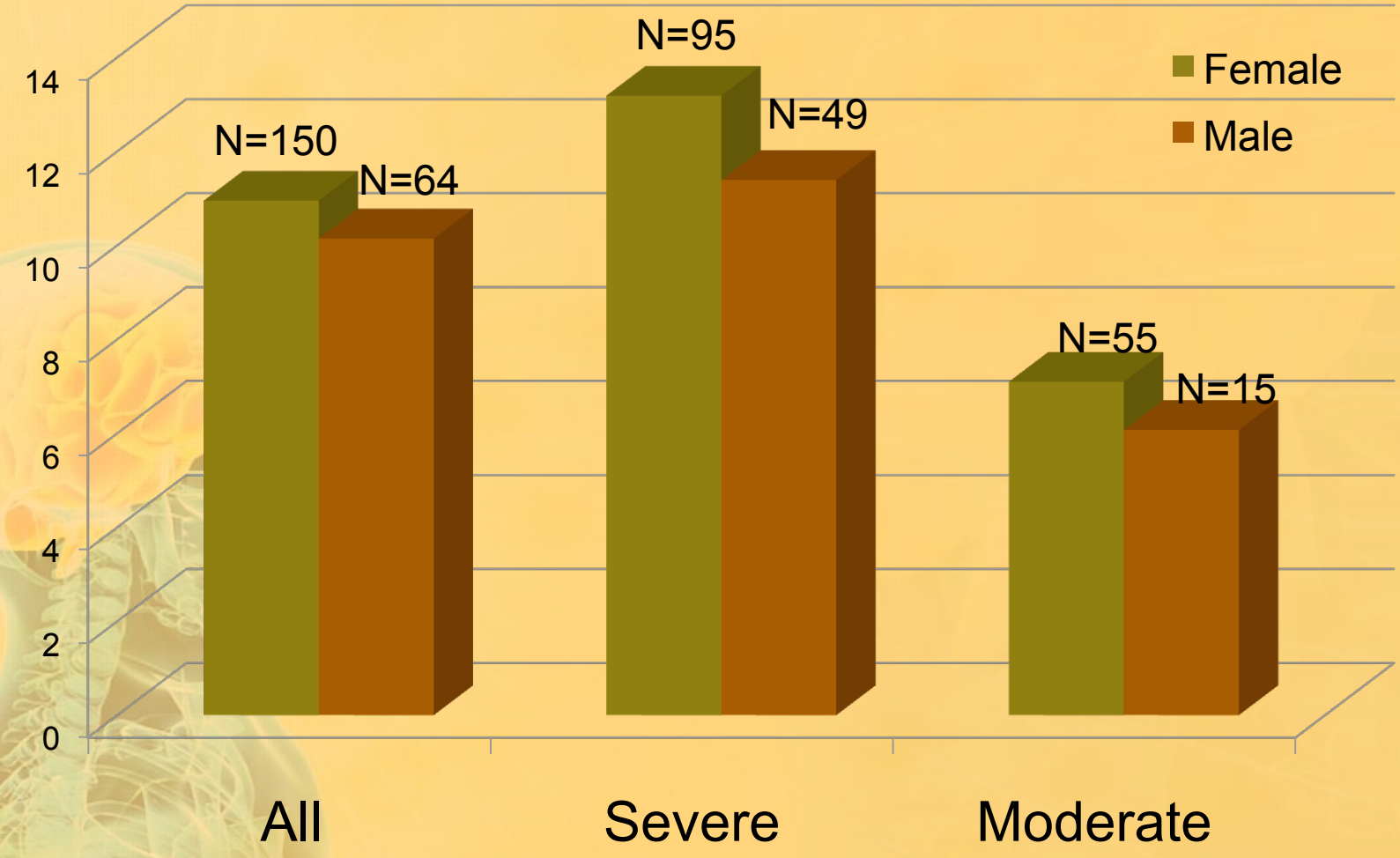
BAI Score Improvement from Session 0 to 8



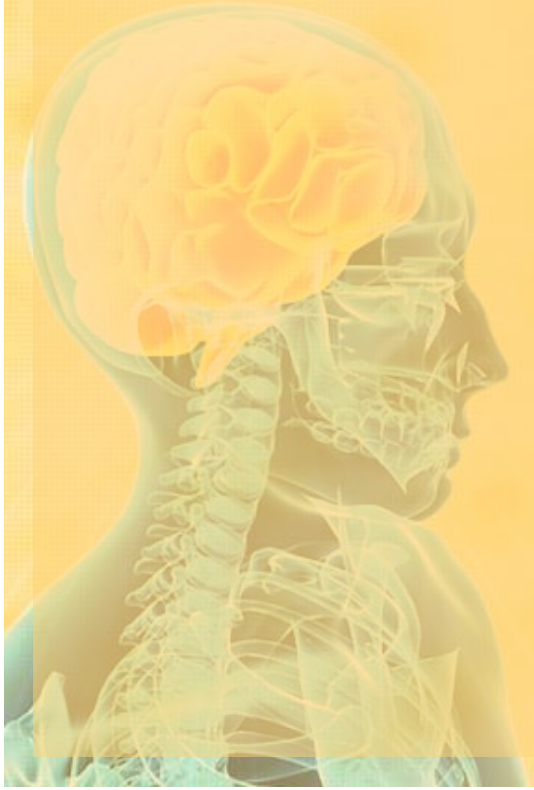
Anxiety Score Improvement by Age



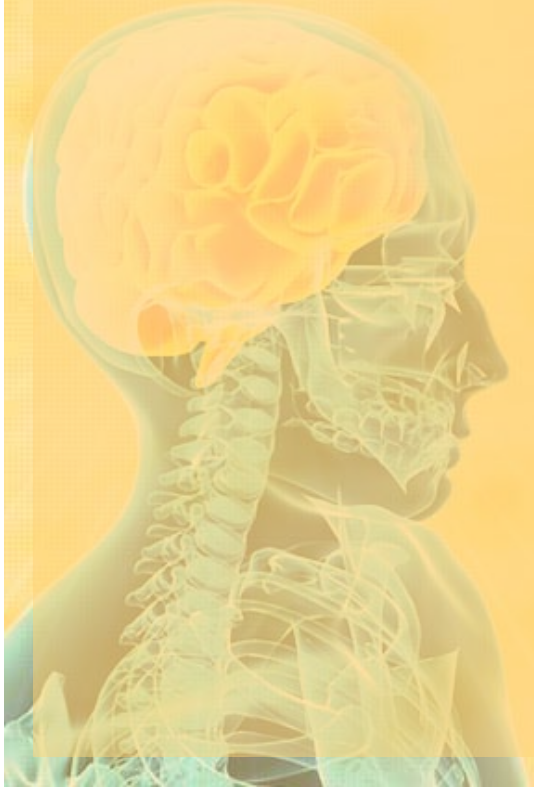
Anxiety Score Improvement by Gender



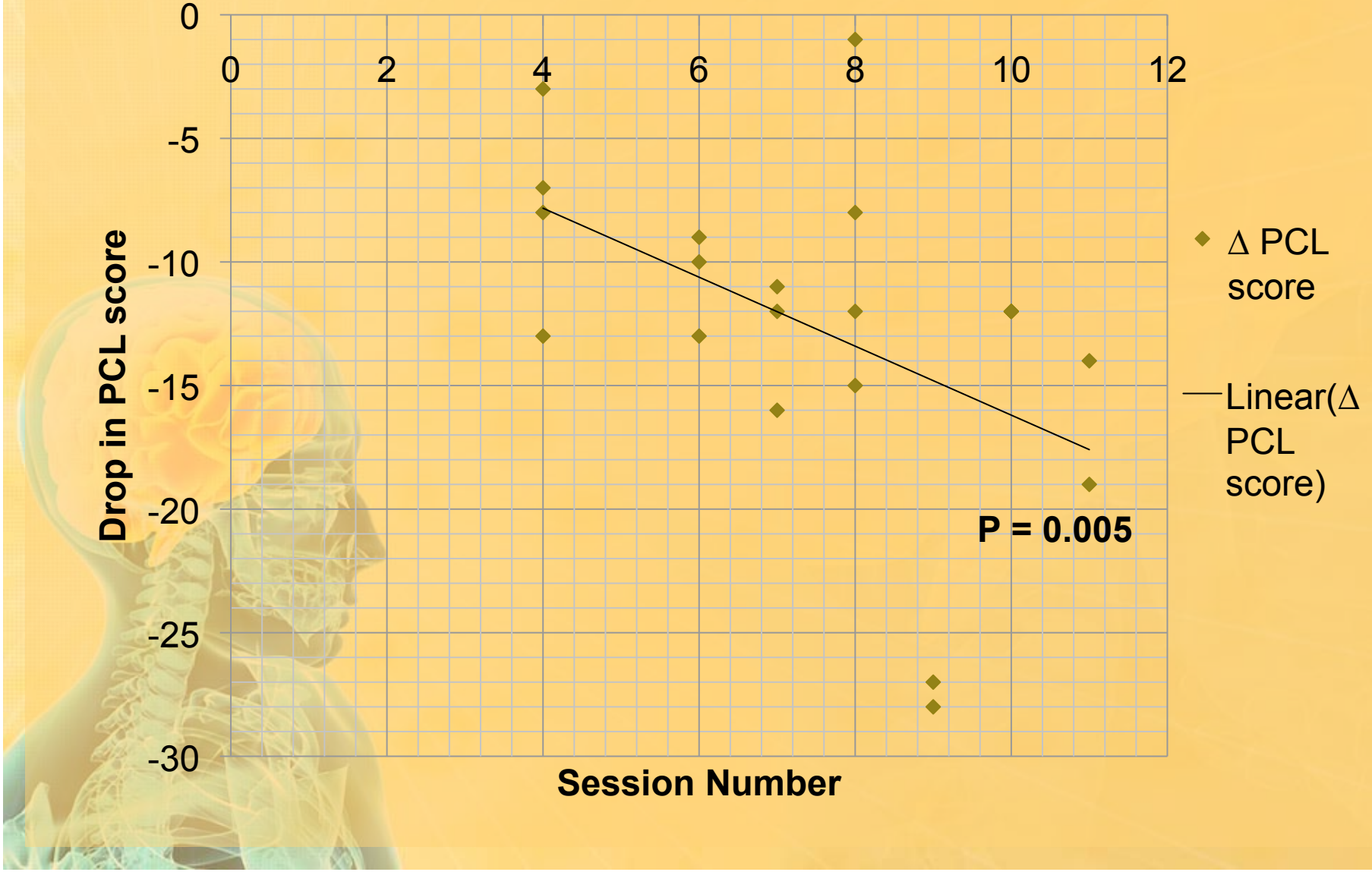
How Does This Compare with Antidepressants?



How Does This Compare with Cognitive Behavioural Therapy (CBT)?



Post Traumatic Checklist



EVALUATING THE EFFICACY OF A BIOFEEDBACK INTERVENTION TO REDUCE CHILDREN'S ANXIETY

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This study explored the reduction of anxiety with children using a combination of electromyograph and thermal biofeedback techniques. One hundred and fifty children (7th and 8th graders) were identified by teachers as anxious and randomly assigned to biofeedback intervention and no-intervention groups. Biofeedback intervention subjects received 6 sessions of thermal training and 6 sessions of electromyographic training over a 6 week period. A post-test anxiety scale demonstrated a significant reduction in both state and trait anxiety. This study suggested biofeedback as a viable intervention which might be coordinated and provided by psychologists to reduce anxiety in children.

For many children, life is a daily confrontation with tension and failure (Sonuga–Barke & Balding, 1993). Family and academic demands combine with changing developmental and social pressures to create an environment in which effective functioning may be difficult. Although anxiety has been shown to have a negative effect on the general health and performance of children, a large number of children face life each day unaware that their symptoms may have solutions (Lail & Schroeder, 1990).

Given these difficulties, numerous authors have called for psychologists to increase intervention services (D'Amato & Rothlisberg, 1992; Kratochwill & Morris, 1991; Sandoval, 1988). Traditionally, psychologists are often called upon to assist children with the broad array of anxiety associated problems which are often concomitant with societal and school difficulties (D'Amato & Dean, 1989). Unfortunately, the array of interventions shown to be effective experimentally is often limited. Therefore, this research was conducted to determine the efficacy of a biofeedback intervention in the reduction of children's anxiety.

Since behavior is biologically related, it is logical to pair biological functions with behavior to illicit change (Hynd & Willis 1988; Pliszka, Hatch, Borcherding, & Rogness, 1993; Whitten, D'Amato, & Chittooran, 1992). One methodology, biofeedback, combines behavior with biological responses and has shown promise. Yet it has often been delegated to a medical setting (Hatch, Prihoda, & Moore, 1992; Orton & Noonberg, 1980). By limiting the use of biofeedback to a medical setting, the benefits are obviously restricted. A logical alternative would be to utilize the procedure in public school settings where children and anxiety are both abundantly available.

Biofeedback may be described as the process of providing information about an ongoing physical response in the body. This knowledge then, is used to change the bodily response and thus increase self control and decrease the anxiety symptoms (Orton & Noonberg, 1980). The procedure provides information to the trainee about bodily reactions (i.e., anxi-

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ety produced responses) such as muscle tension. By learning to control the anxiety reaction in the body, symptoms such as cold hands or test anxiety can be alleviated. This occurs because the cycle of anxiety is broken (Orton & Noonberg, 1980).

Anxiety has been defined as a general discomfort concerning upcoming events in concert with an inability to control the situation (Schalling, 1977; Spielberger, 1977). Clearly, this describes the reality of many children in any group setting. For clarity, Spielberger (1977) has refined the concept of anxiety by identifying *state* and *trait* anxiety. State anxiety is seen as anxiety which is transient while trait anxiety is seen as an intrinsic and lasting reaction to the child's world. Researchers have suggested that children's anxiety responses can be understood as an array of generalized behavioral *and* physiological reactions (Boyle, 1987; Morris & Morris, 1992). Generalized anxiety detrimentally affects learning by impeding the acquisition of new knowledge and impairing the retrieval of previously learned material. Additionally, it may restrict short term memory, depress social and school performance, and impede test performance. Physiologically, anxiety has been associated with symptoms such as tension headaches, elevated blood pressure, asthma and cold hands (Culler & Hollahan, 1980; Gross & Mastenbrook, 1980; King & Ollendick, 1989; Lail & Schroeder, 1990; Smith & Womack, 1987).

Biofeedback has been shown to be a viable intervention which might be used to assist children in any school or group setting. Its successful use has been established with numerous adults and small groups of children in medical settings. For example, biofeedback procedures were able to significantly increase the self concept of elementary age children (Wenck & Worster, 1978). In a research review, Chang and Hiebert (1989) concluded biofeedback to be effective in reducing test anxiety, lowering anxiety in hyperactive children, and in decreasing physiological symptoms.

The reduction of both state and trait anxiety was investigated by Roome and Romney (1985) with a group of 30 children. Three groups of 10 children were assigned to Electromyograph (EMG) biofeedback, Progressive Muscle Relaxation (PMR) biofeedback, or a no-treatment group. Both biofeedback groups were able to significantly reduce state anxiety. In another study designed to reduce the anxiousness of children prior to competition, biofeedback was applied to precompetitive anxiety in 20, high trait-anxious males (ages 10 to 13) involved in bogus competitive athletic events. In this study, electromyograph biofeedback techniques were significantly effective in relaxing the identified target muscle (Blais & Vallerand, 1986). More recently, Potashkin and Beckles (1990) demonstrated the effectiveness of Electromyograph (EMG) biofeedback over Ritilan alone in reducing muscle tension, increasing relaxation, and reducing hyperactivity in a group of 18 males ages 10 to 13 labeled as Attention Deficit Hyperactive Disordered (ADHD).

While these studies have established biofeedback as an efficacious methodology with ADHD children and children involved in athletics, the feasibility and significance of biofeedback when used with a large sample of children within a public school setting has not been established.

Method

Subjects

After providing teachers with a behavioral description of anxiety, approximately 300 7th and 8th grade students were nominated as possessing greater than average levels of anxiety. In an effort to validate anxiety levels, the IPAT Anxiety Scale (Cattell & Scheier, 1976) was administered to all students in this group. This is a 40 question, self-report scale which measures covert and overt anxiety with 20 questions for each area (Auld, 1985). Some 150 subjects were identified as highly anxious on the basis of this measure (i.e., sten score > 6). Next, the 150 students were randomly assigned to an experimental biofeedback intervention group or to a no-intervention group. Thus, extraneous variables were assumed to be equal

between the two groups. With this in mind, a *t*-test was conducted upon available intelligence scores of the two groups with no significant differences found. All students attended one of three middle schools within a midwestern city.

Procedure

The subjects in the biofeedback intervention group ($n = 72$) received 12 sessions of biofeedback training. Specifically, there were 6 sessions of *thermal* training and 6 sessions of *electromyographic* training. Thermal biofeedback training measures the skin temperature of a child. The child is then taught to raise or lower their skin temperature at will in an effort to increase each student's self control. Electromyograph (EMG) biofeedback monitors the muscle tension of the child by measuring the electrical impulses generated by the muscle (Orton & Noonberg, 1980). Students received 12 sessions twice weekly over a 12 week period. Thermal training always preceded electromyographic training, the former being less complex and therefore assumed to be less threatening to the subjects. All subjects were trained on site in their respective middle schools by biofeedback technicians. Following the final training session, the Spielberger State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970) was administered to the 72 biofeedback intervention subjects and to the 75 no-intervention subjects. This inventory is a 40-item, self-report measure that has no time limit for completion. It was utilized because of its differentiation between *state* and *trait* anxiety.

Results and Discussion

To test for significant group differences, group means were subjected to *t*-tests. The biofeedback intervention group attained significantly lower *state* anxiety scores compared to the no-intervention group ($t = -5.4$, $p < .001$). Similarly, the biofeedback intervention subjects also attained significantly lower *trait* anxiety scores as compared with the no-intervention group ($t = -2.25$, $p < .05$).

Studies have shown that anxiety contributes to a host of problems concomitantly with interpersonal difficulties. Logically then, anxiety reduction should be accompanied by more effective functioning such as improved memory, enhanced self esteem, and elevated academic performance including better relationships and higher scores on tests (Grazzi, Leone, Frediani, & Bussone, 1990).

The present study suggests that biofeedback training can effectively and significantly reduce both *state* and *trait* anxiety. It also supports the use of two types of biofeedback training (thermal and electromyographic) in combination. The combination of these two techniques provided children with different methods of anxiety reduction. This concurs with clinical findings indicating that the use of more than one biofeedback method is advantageous (e.g., Lamontagne & Lavalle, 1982).

Prior to the establishment of a biofeedback training intervention program, the psychologist should first determine if the anxiety is environmentally-related. In such situations, anxiety reduction might be best attempted by addressing the system (e.g., family members, teachers) as well as the individual child. If children are anxious, individual biofeedback interventions may need to be considered.

The use of a school-based intervention melds well both with the recent advent of psychological clinics in schools, and with problems within schools themselves. Authors have consistently argued for expanding the psychologist's role to include a focus on interventions (D'Amato & Rothlisberg, 1992). Schools and other group settings have also been criticized for not offering a curriculum that deals appropriately with the emotional and developmental needs of children (D'Amato & Dean, 1989). In fact, biofeedback interventions could be used as a component of outcome or standards based education, in that they are behavioral, measurable, practical and can positively influence academic achievement.

Future research needs to further establish the long-term stability of reduced anxiety levels. Preliminary studies on this stability appear promising (Engel & Rapoff, 1990). Moreover, sophisticated control groups need be developed to evaluate if reduced anxiety is a clear consequence of biofeedback interventions as well as what combination of biofeedback interventions might be best at reducing anxiety.

With anxiety now being such a debilitating condition, first, at home, then in school, and later in society, effective anxiety reduction interventions for children need to be established (Karnes, Oehler–Stinnett & Jones, 1985; Last & Perrin, 1993). For numerous reasons, training in biofeedback may be an appropriate addition to a clinic, hospital, or the school curriculum. Since children are often eager and adept participants, they may well *elect* to participate. If children, early on, are given the opportunity to participate in experiences which address anxiety, anxiety may be prevented before it becomes a serious concern (Hiebert & Jaknavorian, 1989).

In the main, mastery and management of certain physiological functions would appear to offer obvious benefits to individual children. Since children are often unable to control their environments, providing them with a method of control over their behavior may equip them with the personal strategies needed to deal with anxiety. Psychologists are in an ideal position to both develop and supervise biofeedback intervention programs in school settings.

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