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**A COMPARISON OF BLOOD VOLUME PULSE AND FALSE
BIOFEEDBACK IN THE TREATMENT OF MIGRAINE**

by

Paul Greilick

**A Dissertation
Submitted to the
Faculty of The Graduate College
in partial fulfillment of the
requirements for the
Degree of Doctor of Philosophy
Department of Psychology**

**Western Michigan University
Kalamazoo, Michigan
April 1992**

A COMPARISON OF BLOOD VOLUME PULSE AND FALSE BIOFEEDBACK IN THE TREATMENT OF MIGRAINE

Paul Greilick, Ph.D.

Western Michigan University, 1992

The efficacy of temporal artery blood volume pulse (BVP) biofeedback in the treatment of migraine was investigated. After four pre-treatment baseline psychophysiological monitoring sessions, 8 migraineurs were randomly assigned to undergo 12 sessions of either BVP biofeedback or a placebo procedure (false feedback). Both treatments resulted in clinically significant and statistically equivalent reductions in headache activity and medication intake. Subjects exhibited substantial within-session decreases in BVP amplitude during pre-treatment baseline sessions and during false feedback, and the introduction of BVP biofeedback failed to increase the magnitude or the rate of BVP amplitude reductions. All subjects failed to show evidence of learned regulation of temporal artery BVP amplitude or BVP variability. No significant correlations were found between degree of headache reduction and amount of BVP amplitude reduction or amount of BVP variability.

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Paul Greilick

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INTRODUCTION

Surveys have revealed that 20–25% of the general population suffer from migraine headaches, and that 5–10% seek intermittent medical treatment for relief of disabling headache (Bonica, 1990). As defined by the International Headache Society (Olesen, 1988), migraine is a chronic disorder manifesting in attacks of 4 to 72 hours duration and characterized by headache that is unilateral, pulsating, of moderate to severe intensity, is associated with nausea or photo/phonophobia, and is aggravated by physical exertion, is familial, and is related to the menstrual cycle. Migraine with aura (classic migraine) is characterized by neurologic symptoms which precede headache onset, and are unequivocally localized to the cerebral cortex or brain stem. Migraine attacks are associated with a complex sequence of neurological, biochemical, and vascular changes. Experimental evidence has increasingly supported the hypothesis that unstable serotonergic neurotransmission is an important mechanism in migraine pathophysiology, and that vascular dysautoregulation and muscle contraction are probably epiphenomenon (Raskin, 1988).

The vascular features of migraine have been the focus of extensive research since Graham and Wolff (1938) demonstrated the simultaneous reduction of temporal artery blood volume pulse amplitude and head pain following the administration of ergotamine to patients with migraine headache. Vascular changes associated with migraine headache were further detailed by Tunis and Wolff (1953), who reported a

four-stage sequence of change in cranial non-cerebral arteries (vasodilation–vasoconstriction–lability–vasodilation) beginning four days preceding migraine headache, a finding replicated by Feuerstein, Bortolussi, Houle, and Labbe (1983). In addition to these extracranial vasomotor changes associated with migraine onset and termination, some investigators have reported that migraineurs exhibit a number of differences in temporal artery vasomotor activity compared to non-headache controls, including greater beat-to-beat variability under baseline conditions (Morley, 1985), a greater number of abnormal pulse wave forms (Morley & Hunter, 1983), greater dilation and constriction in response to stressors (Arena et al., 1985; Bakal & Kaganov, 1976; Drummond, 1985; Morley, 1985; Rojahn & Gerhards, 1986), and delayed return to baseline during post-stress adaptation (Arena et al., 1985; Gannon, Haynes, Safranek, & Hamilton, 1981).

Citing potential clinical applications in the treatment of migraine headache, successful bidirectional operant conditioning of cephalic vasomotor activity in non-headache subjects was first reported by Christie and Kotses (1973). Subsequently, beginning with Koppman, McDonald, and Kunzel (1974), a number of investigators reported blood volume pulse (BVP) amplitude biofeedback training to be effective in the treatment of migraine (Allen & Mills, 1982; Bild & Adams, 1980; Elmore & Tursky, 1981; Feuerstein & Adams, 1977; Friar & Beatty, 1976; Gauthier, Doyon, Lacroix, & Drolet, 1983; Gauthier, Fournier, & Roberge, 1991; Gauthier, Fradet, & Roberge, 1988; Gauthier, Lacroix, Cote, Doyon, & Drolet, 1985; Knapp, 1982; Lisspers & Ost, 1990; Sturgis, Tollison, & Adams, 1978). Although promising in

terms of positive clinical outcome, the existing research on BVP biofeedback as a treatment for migraine suffer from methodological limitations and from inadequate evaluation of theoretical mechanisms.

The rationale for BVP biofeedback training in the treatment of migraine has typically focused on the vascular changes associated with migraine originally reported by Graham and Wolff (1938). Investigators have reasoned that if migrainuers can learn voluntary regulation of temporal artery blood volume pulse amplitude and/or variability, they can thereby produce and maintain vasomotor response patterns incompatible with headache onset. However, attempts to correlate learned control of BVP amplitude and/or variability with decreased headache in migrainuers have yielded conflicting results. Thus, the mechanism by which BVP training works is unclear. Knapp (1982) suggested that the effectiveness of BVP biofeedback in treating migraine is mediated through lowered sympathetic arousal via learned generalized relaxation, which is consistent with the observation that temporal artery pulse amplitude reduction is one aspect of a general decrease in sympathetic tonic outflow (Dalessio, Kunzel, Sternbach, & Sovak, 1979; Sovak, Kunzel, Sternbach, & Dalessio, 1978).

Two important methodological limitations in the BVP literature hamper interpretation of the data. First, investigators have not adequately controlled for the effects of adaptation of temporal vasomotor activity prior to initiating BVP biofeedback, typically relying on a 5 to 10 minute baseline at the beginning of the first and subsequent biofeedback session. Only one investigator brought subjects into

the clinic for a full pretreatment monitoring session, during which subjects were instructed to attempt to decrease their temporal artery blood flow without the aid of feedback (Gauthier et al., 1985). She found that the majority of subjects (four of seven) exhibited at least a 50% reduction in temporary artery BVP amplitude without having been exposed to feedback, which raises the question of what the subjects learned during subsequent biofeedback sessions. This also raises the question of the role of adaptation in producing BVP amplitude decrements (within and/or across sessions) which investigators have heretofore attributed to BVP biofeedback training.

A second methodological limitation is lack of adequate assessment and/or control of movement-induced BVP signal artifact, to which reflectance photoplethysmography is very susceptible (Flor & Turk, 1989). Investigators typically instruct subjects to lie quietly and to avoid irregular respiration, and then rely on visual rather than EMG monitoring of subjects to detect artifact inducing movement. During pilot investigation for the present study, visual observation of the subject by the experimenter was found to be unreliable in detecting systematic movement artifact. Several subjects inadvertently learned to mediate control of the BVP signal via subtle frontalis movements, which caused displacement of the BVP transducer which, in turn, caused BVP signal attenuation.

The present study sought to replicate prior reports of clinical benefits of BVP biofeedback training using a methodology that addressed some of the limitations of prior research. In the present investigation, multiple physiological responses were monitored over several pre- and post-treatment baseline sessions and, for half of the

subjects, during extended placebo (false feedback) treatment. This procedure permitted both within-subject and group analyses to examine possible mechanisms for changes in BVP activity and in headache activity. Specifically, the present study was designed to answer the following questions:

1. Do migraineurs exhibit significantly greater temporal artery BVP amplitude variability while at rest compared to non-headache controls?
2. Can adaptation account for reductions in BVP amplitude observed during biofeedback training?
3. Is BVP biofeedback superior to an equally credible placebo (false feedback) in reducing headache activity?
4. Is decreased headache activity correlated with learned voluntary control of BVP amplitude or variability?
5. Are decreases in BVP amplitude correlated with lowered autonomic arousal (heart rate)?

METHOD

Subjects

Headache Subjects

Subjects were recruited from the community via newspaper advertisements requesting volunteers to participate in a biofeedback treatment study for migraine. Of the 19 initial respondents, the first 8 individuals who met inclusion criteria and agreed to participate were selected (of the initial respondents, 4 did not meet inclusion criteria and 7 chose not to participate). Inclusion criteria were consistent with that typically required in similar investigations (e.g., Gauthier et al., 1985). Subjects had to report an average of three migraines monthly for a minimum of two years, have received a medical evaluation and diagnosis of migraine by a neurologist within the past year, and meet three of the following criteria: (a) unilateral and throbbing headaches, (b) nausea and vomiting accompanying headaches, (c) headaches preceded by prodromes, (d) photophobia during headaches, and (e) positive family history of migraine. Subjects were excluded from participation if clinical interview revealed significant psychological problems (including depression), medical disorders, a history of head trauma, or if they reported unremitting head pain characterized as diffuse, bandlike, or a dull ache.

Non-Headache Control Subjects

An additional eight individuals who were headache free and of similar age to the headache subjects were recruited as controls. Their participation was limited to a single session of psychophysiologic monitoring.

All subjects signed an informed consent form prior to participation in the study, a copy of which appears in Appendix A. A copy of the Human Subjects Institutional Review Board Human Subjects Approval Form is contained in Appendix B.

Setting and Apparatus

All sessions were conducted in a sound-attenuated and temperature-regulated (approximately 73 degrees Fahrenheit) room, in a private clinic setting. Subjects sat in an overstuffed chair that was reclined at a 45 degree angle, with the experimenter seated outside the subject's field of vision.

A Med Associates (East Fairfield, VT) modular system was used to monitor BVP activity, heart rate, and frontalis EMG. BVP activity was monitored with an ANL-420 reflectance photoplethysmograph. The transducer was attached with a Beckman (Houston, TX) double-adhesive collar over the main ramification of the zygomaticofacial branch of the superficial temporal artery on the side of the head most frequently associated with headache onset. The precision in transducer placement required to obtain a stable waveform with a clear dicrotic notch was aided

by monitoring the BVP signal on an LBO-508 oscilloscope. For data acquisition, the BVP signal was processed through a dual following integrator (Model ANL-610, TC=0.1), an analog to digital converter (Model ANL-944, full wave rectification), and a DIG-600 interface module, to a laboratory computer for online processing and storage. To obtain individual BVP amplitudes, the digitized BVP signal was processed through a count-to-voltage converter (ANL-945), with the logic "1" output of the ANL-420's one-shot used to trigger the reset with each pulse wave. The output of the ANL-945 was routed to a DIG-810 digital display, from which the individual amplitudes were manually recorded. Heart rate was calculated from the rate of the BVP waves by the computer.

Frontalis EMG was monitored with Beckman silver/silver chloride electrodes, with Beckman electrolyte, attached to the skin with adhesive collars 2.5 cm above the eyebrows. Electrode resistance was kept below 5,000 ohms by preparing the skin with Bravisol and alcohol. The EMG signal was processed through an amplifier (ANL-100), EMG coupler and integrator (ANL-140, bandpass = 90-1,000 hertz, TC=0.1), through an analog to digital converter (ANL-944) and DIG-600 interface module, to the computer.

For biofeedback training, the output of the count-to-voltage converter (ANL-945) was fed through a scaling amplifier (ANL-136) to a LED column display visual feedback device (ANL-930), and through a voltage controlled oscillator and audio amplifier (ANL-910) to a shelf speaker. This configuration produced a light column that changed in height and a tone that changed in tone frequency and volume in an

analogue stepwise fashion with each cardiac cycle, according to the height of each BVP amplitude, providing the subject with beat-to-beat feedback of BVP amplitude changes and variability.

The false (placebo) biofeedback signal was created by routing the logic "1" output of the ANL-420 to a count-to-voltage converter (ANL-945), with the output of a time base (DIG-210), through a scaling amplifier (ANL-136), to the feedback devices identified above. The placebo feedback signal was the product of two factors: the subject's pulse rate, with which the signal varied inversely (increased pulse rate/decreased inter-pulse interval produced decreased light column height and decreased tone frequency and volume) and the superimposed signal manipulation of the scaling amplifier by the experimenter. The false feedback signal produced by this configuration appeared identical to the actual BVP biofeedback signal. The fact that the false feedback was subject to movement artifact (i.e., when a subject might cough or "test" the signal via movement) in the same way as the actual BVP biofeedback signal helped make it very credible to the subjects, a problem which exists with using completely random or non-contingent feedback (Burnette & Adams, 1987).

Experimental Design

This study employed both a within-subject repeated measures ABA reversal design, with each subject serving as his/her own control, and a randomized group design, with half of the eight headache subjects randomly assigned to actual BVP biofeedback training, and the other half receiving false biofeedback. Additionally,

eight non-headache subjects served as an additional control group for a measure of BVP variability.

Procedure

All experimental subjects kept a daily headache diary during the five week Pre-Treatment Baseline Phase and the five week Post-Treatment Phase. Subjects were provided with data collection forms (Bakal & Kaganov, 1976) on which they recorded headache intensity 10 times per day, using a 6 point scale (Blanchard, Theobald, Williamson, Silver, & Brown, 1978), ranging from 0 (no headache) to 5 (severe, incapacitating headache), and recorded all medication taken.

All subjects attended an orientation session which served the dual purpose of familiarizing them with the biofeedback equipment and for instituting a procedure designed to minimize BVP measurement artifact in subsequent sessions. As noted above, pilot investigation had revealed that providing subjects with standard instructions to lie quietly was, in some cases, insufficient to prevent learned mediation of the BVP biofeedback signal via frontalis muscle movement. What had initially appeared to be learned BVP amplitude reduction was, in fact, systematic BVP signal error caused by photoplethysmograph transducer displacement associated with within-session EMG trending. Therefore, to minimize EMG-mediated BVP signal error during the study, subjects were attached to the biofeedback monitors and allowed to explore the effects of facial muscle movement on the BVP signal, and briefly instructed in the maintenance of "steady-state" EMG. Additionally, the experimenter

monitored frontalis EMG during all sessions, and verbally prompted subjects to relax if frontalis EMG was elevated during transducer hookup, or if within-session EMG trending occurred of sufficient magnitude to distort the BVP signal.

Subjects attended four pretreatment baseline monitoring sessions, two per week, during the last two weeks of the Pre-Treatment Baseline Phase. They were instructed to arrive at the clinic at least ten minutes early to allow for physiological stabilization. To minimize recording artifact, at the beginning of each session subjects were reminded to lie as quietly as possible and to avoid irregular respiration. Once the transducers were attached to the subject, 10 minutes were allowed for adaptation prior to initiating data collection. At the beginning of data collection, subjects were instructed to simply sit quietly. Physiological recording lasted for 36 minutes, the first 4.8 minutes of which constituted the Session Baseline.

Upon completion of the five week Pre-Treatment Baseline Phase, subjects were randomly assigned to one of two treatment groups (four per group): the BVP biofeedback training group, or the False Feedback group. Subjects in each group underwent 12 sessions of their respective type of biofeedback training (actual BVP feedback versus False Feedback), twice a week for six weeks. As during sessions in the Pre-Treatment Baseline Phase, physiological recording in the Treatment Phase sessions lasted for 36 minutes, the first 4.8 minutes constituting the Session Baseline, the last 31.2 minutes biofeedback training.

Prior to the beginning of the Treatment Phase, all subjects received the same treatment rationale, which emphasized the role of vasomotor and autonomic instability

in migraine onset, and the prophylactic value of cultivating awareness and regulation of their vasomotor activity. The biofeedback signals were explained in terms of BVP activity to both groups of subjects in a standardized fashion, and they were instructed to adopt an experimental approach to discovering techniques to "stabilize and decrease" the light column and tone via imagery, thoughts, or feelings. At every session, subjects were encouraged to be aware of and regulate their physiology throughout their daily activities.

For subjects in the False Feedback condition, the false feedback signal, as noted above, was controlled in part by the experimenter through superimposed manipulation via a scaling amplifier. The signal manipulation was patterned to produce a sense of at least moderate training success for each subject, with the "BVP signal" appearing to gradually decrease 20–50% within each session.

Following completion of the biofeedback training Treatment Phase, subjects returned to the laboratory for three monitoring sessions during the Post-Treatment Phase, once per week for three weeks. Sessions were conducted in the same manner as those in the Pre-Treatment Baseline Phase.

The eight non-headache control subjects participated in one session during which their temporal BVP activity was briefly sampled under conditions designed to be identical to those under which comparable data was sampled from the headache subjects. The BVP activity was sampled following an orientation to the equipment, and following a 10 minute adaptation phase and a 4.8 minute Session Baseline.

Data Reduction

For each session, the computer generated averages for each of 15 2.4 minute epochs ($2.4 \text{ minutes} \times 15 = 36 \text{ minutes}$) for temporal BVP amplitude, heart rate, and frontalis EMG for each headache subject. The first 4.8 minutes of each session constituted the Session Baseline.

For computation of BVP variability for all 19 sessions, the final 16 BVP amplitudes of each Session Baseline and of each session were recorded. Analyses of changes in BVP amplitude are confined to within-session percent changes from Session Baselines. Across-session analyses of temporal BVP amplitudes are unreliable because the BVP signal is a relative measure of blood flow, and is significantly affected by the minute changes in transducer placement across sessions (Stern, Ray, & Davis, 1980).

Dependent Variables

Self-Report Measures

Headache Index

The Headache Index, adapted from Blanchard et al. (1978) represents the average daily headache activity (theoretical range = 0 to 50), and is a product of headache intensity and duration scores recorded on the headache diary forms. It was

calculated for each phase by summing all intensity scores of the phase and dividing by the number of days recorded (35).

Medication Index

The Medication Index is a measure of average daily intake that is sensitive to both the dosage and the relative potency of medications consumed. It was calculated by first assigning each medication a relative potency value of 1 to 7 based on a scale developed by the Menninger group (Coyne, Sargent, Segerson, & Osbourn, 1976) and extended by Blanchard and Andrasik (1985). Each medication's potency value was then multiplied by the number of doses taken, and the product was divided by 35 to yield the within-phase daily average.

At the end of the study, percent improvement scores were calculated for each subject for both headache activity and medication intake using the following formula:

$$\text{Percent Improvement} = \frac{\text{Pre-Treatment Index} - \text{Post-Treatment Index}}{\text{Pre-Treatment Index}} \times 100$$

Treatment Credibility

To assess treatment credibility and expectation for improvement, subjects were administered a questionnaire developed by Borkovec and Nau (1972) and modified by Gauthier, Bois, Allaire, and Drolet (1981) (range = 0–28, higher scores reflecting higher perceived credibility) at the end of sessions 5, 10, and 19.

Attribution of Improvement

Subjects were interviewed upon completion of treatment to assess their perceptions as to why their headaches decreased.

Psychophysiological Measures

BVP Amplitude

The maximum within-session percent decrease in BVP amplitude was calculated for every session using the following formula: $\text{Session Baseline amplitude (4.8 minute epoch)} - \text{lowest session amplitude (2.4 minute epoch)} / \text{Session Baseline amplitude}$.

Rate of BVP Amplitude Change

Acceleration of the rate of the within-session decreases in BVP amplitude would be expected to follow the introduction of BVP biofeedback if subjects indeed learned self-regulation of the BVP signal. Rate of change, as defined by the time interval from Session Baseline to maximum within-session decrease in BVP amplitude, was calculated for all Pre-Treatment Baseline Phase sessions and Treatment Phase sessions.

BVP Variability

Each subject's BVP amplitude beat-to-beat variability was calculated twice for each session, using the final 16 BVP amplitudes of each Session Baseline and each session, and was expressed as the coefficient of variation (CV), which is the ratio of the SD/X. Thus, the CV is calculated by dividing the standard deviation of the BVP amplitudes by their mean value. The CV was used to measure changes in variability because unlike other such measures, the CV is unaffected by differing mean levels associated with changes in the "gain" or sensitivity settings of recording equipment (Burdick, 1972).

Heart Rate

The maximum within-session percent decrease in heart rate was calculated for each session using the following formula: Session Baseline heart rate (4.8 minute epoch) – lowest session heart rate (2.4 minute epoch) / Session Baseline heart rate.

RESULTS

Credibility Data

Treatment credibility was rated high by both groups. Of a possible maximum score of 28, the BVP group means at sessions 5, 10, and 19 were 23.5 (range = 22–25), 24.5 (range = 23–26), and 23 (range = 22–28). Means for the False Feedback group were 24.75 (range = 22–28), 25.35 (range = 23–28), and 24.5 (range = 21–28). A Mann–Whitney U test revealed equivalent treatment credibility across treatment groups on all three occasions. Further analysis by Friedman two–way ANOVA revealed credibility to be statistically unchanged over the course of the study for both treatment groups.

BVP Data

BVP Amplitude

Maximum within–session percent decreases of BVP amplitudes from Session Baselines by session and by subject are displayed in Figure 1 and Figure 2 for BVP and False Feedback treatment groups respectively. Inspection of Figures 1 and 2 reveals within–session decreases ranging from 19% (Subject 8) to 61% (Subject 3) during Pre–Treatment Phase sessions, with significant within– and between–subject variability. Notably, for all subjects, the magnitude of within–session decreases in

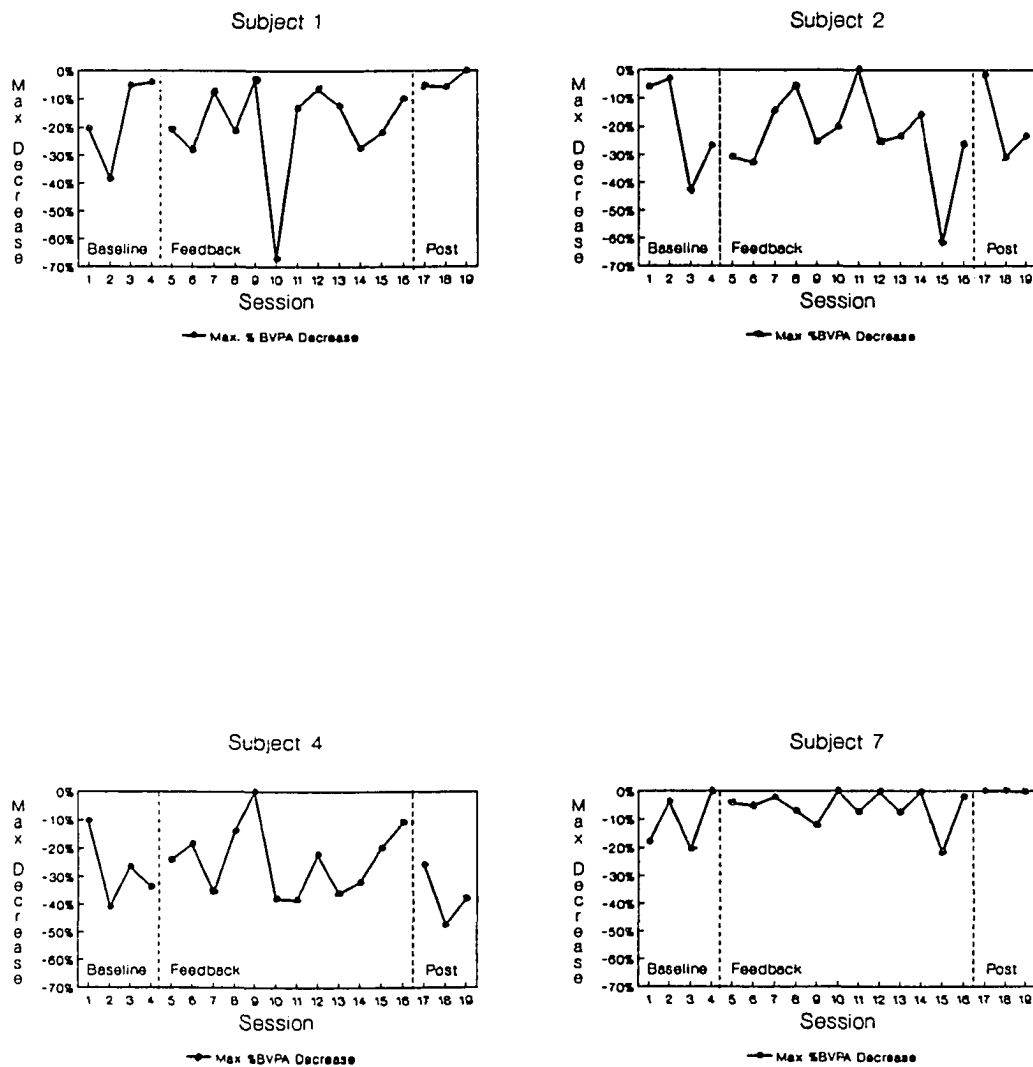


Figure 1. Maximum Within-Session Percent Decrease in Temporal Artery BVP Pulse Amplitude by Session of Individual Subjects in the BVP Biofeedback Group.

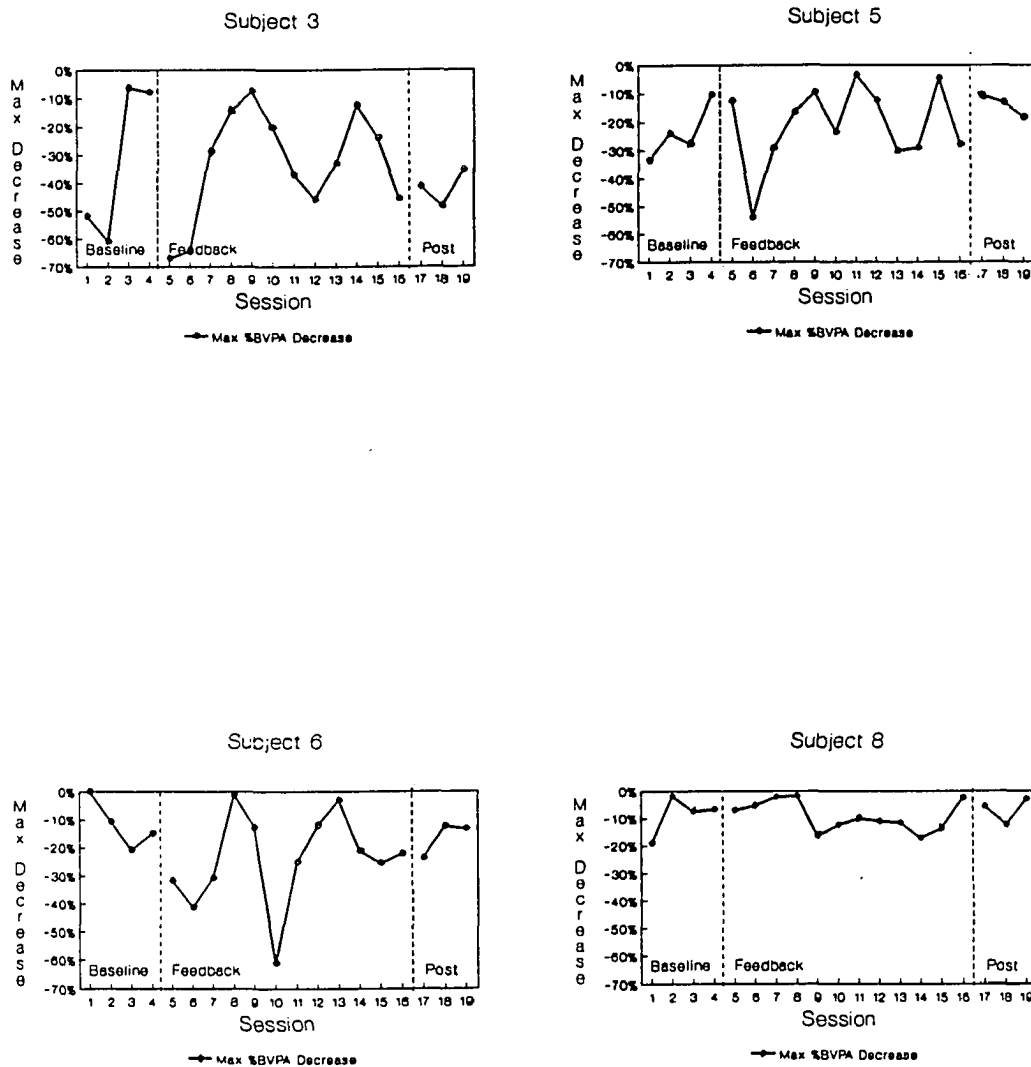


Figure 2. Maximum Within-Session Percent Decrease in Temporal Artery BVP Pulse Amplitude by Session of Individual Subjects in the False Feedback Group.

BVP amplitude observed during Pre–Treatment Phase sessions appears unaffected by the introduction of biofeedback.

Table 1 displays group averages of maximum within–session percent decrease of BVP amplitude by experimental phase. Examination of Table 1 fails to reveal significant differences in average maximum session BVP amplitude decrease across experimental phases or across treatment groups. In sum, analyses of both within–subject data and group comparison data fail to show evidence of learned regulation of BVP amplitude by any subject.

Table 1

Group Averages of Maximum Within–Session Percent Decrease
of BVP Amplitude by Experimental Phase

Group	Pre–Treatment Baseline (4 Sessions)	Biofeedback (12 Sessions)	Post–Treatment Baseline (3 Sessions)
BVP ($n = 4$)	8.8%	18.3%	14.9%
False FB ($n = 4$)	19.0%	22.0%	19.9%

Rate of BVP Amplitude Decrease

During Pre–Treatment Phase sessions, mean time to maximum within–session BVP amplitude decrease was 24 minutes for the BVP group and 25 minutes for the False Feedback group. During Treatment Phase biofeedback sessions, mean time to maximum within–session BVP amplitude decrease was virtually unchanged for both

groups, 23 minutes for the BVP group and 24 minutes for the False Feedback group. Thus, the introduction of BVP biofeedback was not associated with acceleration of the rate of within-session decrease of BVP amplitude.

BVP Variability

Session baseline and end of session BVP amplitude CVs for each subject and session are plotted in Figures 3 and 4 for the BVP group and the False Feedback group respectively. Inspection of Figures 3 and 4 reveals considerable within- and across-subject and within- and across-session variability of the CV. Visual analysis of Figure 3 fails to reveal any evidence that subjects learned to decrease BVP amplitude variability either within or across sessions with the aid of BVP biofeedback.

Clinical Outcome Data

Pre-Treatment Data

Table 2 displays headache and medication index scores and their percent change for each subject. Group comparisons at pre-treatment using *t* tests for independent means revealed that the BVP and False Feedback groups were equivalent in terms of the headache and medication indices.

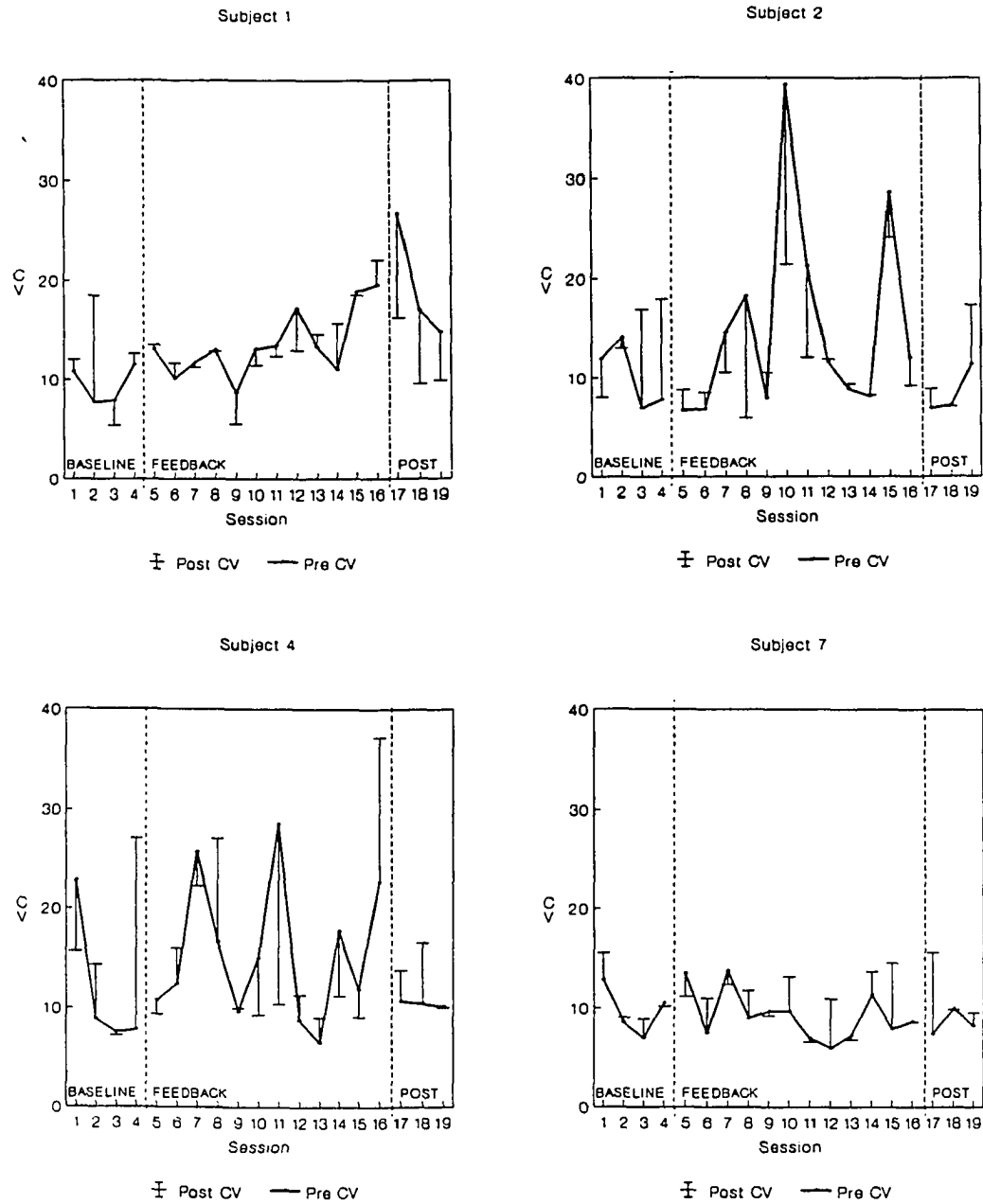


Figure 3. BVP Variability (CV) at the End of Each Session Baseline (Pre-CV) and at the End of Each Session (Post CV), of Individual Subjects in the BVP Biofeedback Group.

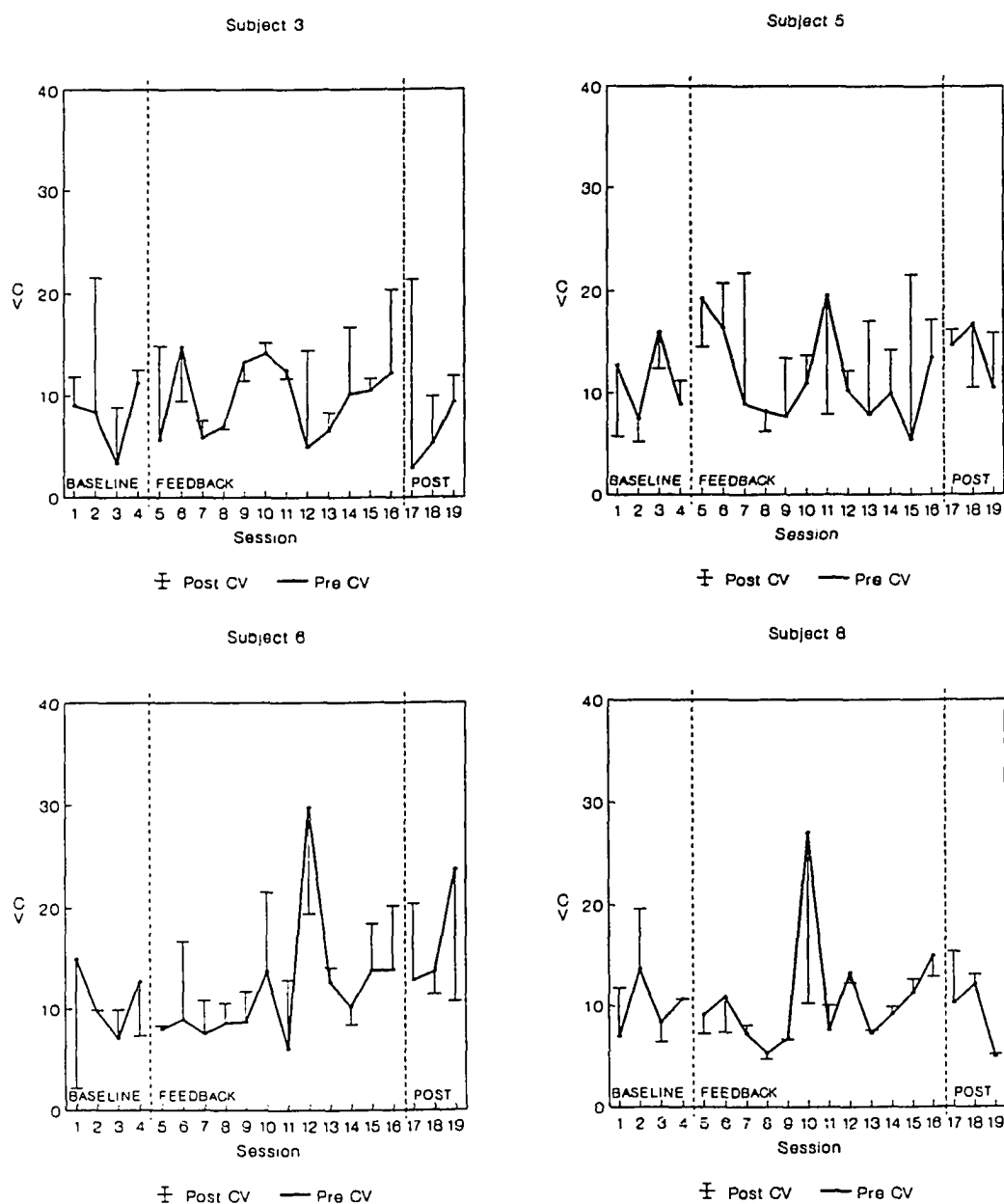


Figure 4. BVP Variability (CV) at the End of Each Session Baseline (Pre-CV) and at the End of Each Session (Post CV), of Individual Subjects in the False Feedback Group.

Table 2

Headache and Medication Index Scores Pre- and Post-Treatment
and Their Percent Change Scores for Individual Subjects

Subjects by Group	Headache Index			Medication Index		
	Pre	Post	% Change	Pre	Post	% Change
BVP						
1	10.2	7.3	-28.8%	3.4	1.8	-46.7%
2	5.7	0.2	-96.0%	2.7	0.2	-93.6%
4	12.8	6.7	-47.3%	9.1	4.5	-50.2%
7	5.8	1.2	-78.8%	2.7	0.3	-88.5%
False Feedback						
3	19.8	1.9	-90.5%	5.7	0.3	-95.0%
5	5.9	3.0	-50.0%	1.4	0.5	-65.3%
6	25.4	14.8	-41.6%	3.1	1.9	-39.8%
8	6.5	2.0	-68.9%	4.9	1.4	-71.5%

Treatment Effects

Mean reductions in headache activity were virtually identical across groups at 60% and 62% for the BVP and False Feedback groups respectively. Paired t tests revealed significant reductions in headache activity for the BVP group ($t(3) = 7.01$, $p < .01$) and the False Feedback group ($t(3) = 2.63$, $p < .05$) using one-tailed probabilities. Mean reduction in medication intake was also similar across groups at 62% and 73% for the BVP and False Feedback groups respectively. The statistical significance of medication reductions was confirmed for both the BVP group ($t(3) = 4.43$, $p < .05$) and the False Feedback group ($t(3) = 2.59$, $p < .05$).

To assess whether the magnitude of improvements were equivalent across groups, *t* tests for independent means were applied to post-treatment headache and medication indices and to their percent change scores. No significant differences were revealed.

To assess the clinical significance of these improvements, subjects were classified as successful if they attained at least a 50% reduction in headache activity. Inspection of Table 2 shows five of the eight subjects to be treatment successes (63% of all subjects), three of the five successes belonging to the False Feedback group. In regard to medication intake, 6 of the 8 subjects (75% of all subjects) reported at least a 50% reduction.

Correlations Between Credibility Ratings and Treatment Outcome

The relationship between perceived treatment credibility and treatment outcome was assessed via Spearman-rank correlation coefficients. Correlations were not significant at sessions one and six. However, a significant association developed by post-treatment ($r_s = .88$, $p < .01$), the subjects who were most improved reporting treatment as most credible.

Correlations Between BVP Amplitude Reductions and Treatment Outcome

To assess whether treatment outcome was related to within-session BVP amplitude reduction, Spearman-rank correlations were calculated between headache

change percentage scores and the number of sessions in which BVP amplitude decreased by various levels. None of the correlations was statistically significant.

Correlations Between BVP Variability and Treatment Outcome

To assess whether treatment outcome was related to BVP beat-to-beat variability, Spearman-rank correlations were calculated between headache percentage change scores and CVs at session baseline and at session end averaged for each treatment phase. None of the correlations was significant.

BVP Variability of Headache Subjects Versus Non-Headache Controls

To determine whether the temporal BVP amplitude variability of headache subjects was greater than that of non-headache controls when sampled under similar conditions (during the Session Baseline of the initial session), the group mean CV of the 8 headache subjects was compared to the mean CV of the 8 non-headache controls (12.8 and 7.8 respectively). A Mann-Whitney U test revealed a significant group mean difference at the .05 level ($U(1,16) = 8, p < .05$).

Within-Subject Correlations Between Decreases in BVP and Heart Rate

The average within-session reduction in heart rate for all subjects was -5.3% during pre-treatment baseline sessions, and -6.4% during feedback sessions. To

assess the extent to which an individual subject's reductions in BVP amplitude were associated with reductions in heart rate, Pearson product-moment correlations were calculated between each session's maximum percent reduction in BVP amplitude and the corresponding percent reduction in heart rate. Correlations were significant for three of the eight subjects: Subject 1 ($r = .69$, $p < .02$), Subject 2 ($r = .72$, $p < .01$), and Subject 6 ($r = .84$, $p < .01$), and approached significance for Subject 5 ($r = .51$, $p = .07$).

Attribution of Improvement

Following treatment, subjects were interviewed to assess what they attributed their improvement to. Several themes were identified in the responses of all eight subjects:

1. The learning and practicing of relaxation skills (subjects generally perceived biofeedback training as an aid towards cultivating relaxation).
2. An increased awareness and regulation of pre-headache states (in terms of physiological sensations and emotions).
3. Dietary changes (i.e., avoiding caffeine, eating a healthier diet, not skipping meals).
4. Cognitive changes (i.e., "seeing" self as more competent, feeling more confident, "perceiving stressful situations differently").

DISCUSSION

The present data confirm that migraineurs exhibit greater beat-to-beat variability of temporal artery BVP amplitudes under baseline conditions compared to non-headache controls, a finding consistent with previous reports (Graham & Wolff, 1938; Morley, 1985; Tunis & Wolff, 1953). However, migraineurs were not able to learn to decrease beat-to-beat BVP amplitude variability with the aid of biofeedback training. Furthermore, there was no correlation between the degree of headache relief and the amount of BVP amplitude variability. Thus, the data suggest that learned self-regulation of BVP amplitude variability is neither a necessary or even an attainable therapeutic goal in the treatment of migraine.

While within-session reductions of temporal artery BVP amplitude were observed during biofeedback training, these reductions appear to be a result of adaptation to the experimental situation rather than biofeedback training. Specifically, all subjects showed significant within-session reductions in BVP amplitude during the four pre-treatment baseline monitoring sessions, and the introduction of BVP biofeedback training failed to increase the magnitude or the rate of these reductions. Furthermore, subjects undergoing False Feedback showed reductions in BVP amplitude comparable to those shown by subjects undergoing BVP biofeedback. Thus, there was no evidence that the reductions in BVP amplitude which occurred

during biofeedback training were the result of learned control of the BVP feedback signal or self-regulation of temporal artery BVP amplitude.

The data confirm that undergoing BVP biofeedback training is followed by significant reductions in headache activity and medication intake. However, equally significant reductions were reported by subjects who underwent the equally credible False Feedback placebo procedure. Further, the degree of headache relief was not correlated with the magnitude of BVP amplitude reduction or with the amount of pre- or post-treatment BVP variability. Thus, the data do not support a specific treatment effect of BVP biofeedback.

Regarding the mechanisms by which the BVP biofeedback and the False Feedback procedures resulted in significant clinical improvement, the present data provide some support for lowered physiological arousal, or what has been termed the "conditioned adaptation-relaxation reflex" (Dalessio et al., 1979; Sovak et al., 1978) as a mediator of treatment success. Relaxation is associated with a general decrease in sympathetic tonic outflow, and characterized by reduced blood flow to the head, dilation of peripheral arteries, and decreased heart rate. The results of the majority of well-controlled investigations indicate that relaxation training, with or without the aid of biofeedback, is effective in the treatment of migraine, and that for most migrainuers, treatment efficacy is unchanged by the addition of biofeedback (Chapman, 1986). In the present study, all subjects showed evidence of general relaxation through decreased heart rate and temporal artery BVP amplitude. Additionally, a strong correlation between the magnitude of reductions in heart rate

and BVP amplitude were shown by half of the subjects. However, no significant correlation was found between the magnitude of reductions in BVP and headache, which could theoretically be present but undetected due to a small subject pool and considerable variability of the responses involved.

An alternative explanation for headache reduction following biofeedback training is that the regulation or lowering of physiological arousal, as measured by psychophysiological parameters, is but one of several mechanisms which operate in the context of biofeedback training. As noted by Chapman (1986), the ritual of biofeedback training is exceedingly complex, and whatever association that may exist between headache parameters and physiological parameters, it is not simple. Biofeedback is increasingly being conceptualized as a process which alters cognitive, emotional, biochemical, and behavioral responses. In reviewing the medical literature, Raskin (1988) noted a growing consensus that migraine is a manifestation of a lowered biological threshold to a wide variety of internal and external stimuli, that instability of serotonergic neurotransmission (centrally and peripherally) is an important pathogenic mechanism, and that associated vascular and muscular changes are probably epiphenomena. This model of migraine suggests that a mechanism or "final common pathway" of various behavioral (and medical) interventions in treating migraine is via the prevention of the dysregulation of serotonergic neurotransmission, to which migrainuers appear to be inherently susceptible (Raskin, 1988). Thus, regulation of physiological arousal through the practice of learned relaxation skills, the alleviation of emotional distress, avoiding caffeine, alcohol, and other dietary

triggers, and regularity of eating and sleeping may all potentially serve to prevent the dysregulation of serotonergic neurotransmission in the migrainuer. Indeed, in the present study, all subjects reported increased awareness and regulation of emotional and physiological arousal, avoidance of caffeine and sugar, increased regularity of eating, and an altered perception of self and others in which they felt more control, even though the experimenter provided no instruction in these matters. It appeared that during the course of biofeedback training (false or otherwise), subjects were stimulated by their biofeedback experience (including perhaps their perceived success) to act on previously acquired information regarding headache prevention and management. This is consistent with the model of therapeutic change in biofeedback training which emphasizes mediating cognitive factors which include enhanced self-efficacy and internal locus of control, leading to altered coping behaviors (Bandura, 1977; Frank, 1976; Holroyd et al., 1984). Holroyd et al. (1984) reported that changes in self-efficacy and locus of control correlated with tension headache reduction in subjects undergoing EMG biofeedback, while EMG activity was uncorrelated with treatment outcome. Future investigations designed to assess the mechanism of biofeedback's effectiveness in treating migraine should assess changes in diet, sleep, exercise, emotional arousal, interpersonal relationships, cognitive factors, and perceived success at biofeedback training, all of which may be affected by the biofeedback training ritual, whether or not they are explicit intervention targets. For example, in a recent study the majority of migrainuers reported a reduction of at least

50% in headache frequency after receiving brief instruction on the identification and avoidance of headache triggers (Blau & Thavapalan, 1988).

The present data underscore the importance of assessing for the effects of adaptation before attributing physiological changes to BVP biofeedback training, and raises questions about prior research in which adaptation was not assessed. Further, based on clinical outcomes, the present data fail to justify the additional expense and time required to utilize BVP biofeedback training in migraine treatment.

APPENDICES

Appendix A
Informed Consent

INFORMED CONSENT

I, _____, willingly agree to participate in this research study which has been explained to me to my satisfaction. This study is being conducted at Health Psychology, P.C.

This study has been designed to investigate the effects of biofeedback training on migraine headache symptomology and a variety of body functions including muscle tension, heart rate, skin temperature, and vasomotor activity (constriction and dilation of blood vessels).

By electing to participate in this study I will be consenting to 1) daily record headache symptomology and medication intake, 2) complete a questionnaire on medical and headache history, 3) complete a questionnaire on depression symptomology, 4) refrain from taking vaso-active medications during the study, and 5) undergo approximately 20 biofeedback training/monitoring sessions of 40-60 minutes duration scheduled over a 5-6 month period. I understand that surface sensors will be attached to my hand, forehead, and temple.

I understand that by the process of random (chance) selection, I will be assigned to receive one of two types of biofeedback, one of which may not be as effective as the other type. I understand there is no guarantee that the biofeedback training I receive will result in a reduction in my headache pain.

I understand that there are virtually no risks involved in biofeedback training. It has been explained to me that the biofeedback used in this study is constructed with built-in electronic/optical isolation to protect me from receiving electrical shock in the event of equipment failure, local power problems, etc.

It has been explained to me that I may benefit from participation in this study by gaining self-awareness of headache triggers, and possible decreased medication intake and/or headache symptomology. I understand that my participation may be of value in advancing scientific knowledge with respect to the effects of biofeedback training on migraine headache symptomology and physiology.

I understand that the information obtained from my participation in this study will be held confidential. I understand that in the event that data resulting from my participation are used in a publication, my name or any other identifying information will not be released so as to protect my anonymity.

I understand that my participation is entirely voluntary. I also understand that I may withdraw from the study at any time. I understand that I may ask questions concerning my participation during the course of the study.

I understand that it is unlikely that physical or psychological injury will result from these research procedures. In the event of untoward side effects however, care will be arranged but not paid for by the Principal Investigator or Western Michigan University.

I have read the Informed Consent, have had my questions answered to my satisfaction by the Principal Investigator, and freely agree to participate.

(signature of participant) (date)

(witness) (date)

I understand that if I have any complaints after giving consent, I may contact the Principal Investigator and/or the Human Subjects Review Board of Western Michigan University. I understand that I may contact the Human Subjects Review Board without going through the Principal Investigator.

Principal Investigator: Paul Greilick, M.A.
Limited Licensed Psychologist
Health Psychology, P.C.
375-0624

Human Subjects Review
Board Chair: Mike Pritchard, Ph.D.
Department of Philosophy
Western Michigan University

Appendix B

Human Subjects Institutional Review Board Human Subjects Approval Form

Reviewer: Expeditious MS? Overlook *Hartman Western Michigan University
 Subjects Institutional Review Board
 Human Subjects Approval Form
 85-02-04
 Received: 1/31/85

DIRECTIONS: Please type or print each response - except signatures. Refer to the Western Michigan University Policy for the Protection of Human Subjects to determine the appropriate level of review.

PRINCIPAL INVESTIGATOR Paul Greilick DEPARTMENT Psychology
 Home Phone 375-9557 Office Phone 375-0624
 Home Address 3822 Greenleaf Circle Office Address 3503 Greenleaf Blvd
Kalamazoo, MI 49008 Kalamazoo, MI

PROJECT TITLE: Blood Volume Pulse Biofeedback in the Treatment of
Migraine Headache

SUBMISSION DATE: _____ PROPOSED PROJECT DATES 1-85 TO 5-85

Note: The principal investigator should not initiate the research project until the protocol has been reviewed and approved by the Human Subjects Institutional Review Board.

APPLICATION IS: ☒ New ☐ Renewal ☐ Continuation ☐ Supplement

(SOURCE OF FUNDING: Graduate College WMU
 (if applicable))

Paul Greilick
 Signature of Investigator

STUDENT RESEARCH (Fill out if applicable.)

Name of Student Paul Greilick Phone 375-9557 Address 5933 S. 9th
 The research is: ☐ Undergraduate Level ☒ Graduate Level
 Faculty Advisor R. Wayne Fuqua, PhD Department Psychology
 Signature of Faculty Advisor R. Wayne Fuqua Phone 383-6052

VULNERABLE SUBJECT INVOLVEMENT (Fill out if applicable.)

Research involves subjects who are: (check as many as apply)

- | | |
|--|--|
| 1. <input type="checkbox"/> children
<u>approximate age</u> _____ | 6. <input type="checkbox"/> Other subjects whose life
<u>circumstances may interfere</u>
<u>with their ability to make</u>
<u>free choices in consenting</u>
<u>to take part in research</u> |
| 2. <input type="checkbox"/> mentally retarded persons
<u>check if institutionalized</u> _____ | _____ |
| 3. <input type="checkbox"/> mental health patients
<u>check if institutionalized</u> _____ | _____ |
| 4. <input type="checkbox"/> prisoners | _____ |
| 5. <input type="checkbox"/> pregnant women | (Describe Please) |

Received: _____

LEVEL OF REVIEW: Please indicate here if you think that the research project is exempt from review, subject to expedited review, or subject to full review.

____ Exempt (Forward 1 application to IRB Chair)

Which category of exemption applies? # _____

X Expedited (Forward 3 applications to IRB Chair)

____ Subject to full IRB review (Forward 8 applications to IRB Chair)

Comments:

Your application was reviewed and the Human Subject Institutional Review Board (HSIRB) has determined that:

X 1. The proposed activities, subject to any conditions and/or restrictions indicated in Remarks below, have (a) provided adequate safeguards to protect the rights and welfare of human subjects involved, (b) established appropriate procedures and/or documents to obtain informed consent, and (c) demonstrated that the potential benefits of the research substantially out-weigh the risks.

____ 2. The proposed activities, for reasons indicated in Remarks below do not provide adequate protection for the rights and welfare of the human subjects.

its meeting on 2/6/85, the HSIRB (approved) (provisionally approved... the remarks) this application with regard to the treatment of human subjects. The HSIRB categorized this application as:

X 1. Involving subjects at no more than minimal risk.

____ 2. Involving subjects at more than minimal risk.

REMARKS:

04 Approval: minimal risk

Recommendation: What will you do if symptoms develop during the course of the experiment which suggest that the subject might benefit from taking some medication? If a physician suggests that the subject take medication, would you encourage the subject to withdraw from the experiment, or could you have the subject continue while keeping a careful record of the use of medication and the apparent results? Some statement to the patient about this contingency might be helpful.

M. J. [Signature]
Signature HSIRB Chair

2/6/85
Date

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